

**Government of the Republic of Union of Myanmar
Ministry of Education**



**ENVIRONMENTAL TOXICOLOGY
AND
HUMAN HEALTH**

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Member of the Executive Committee
The Myanmar Academy of Arts and Science**

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Author's Preface

Environmental toxins are chemicals both human-made and naturally occurring. Over the last 50 years, from 70,000 to 100,000 different chemicals have been introduced into the world's markets, with about 1,500 new ones added each year. Over 3,000 chemicals are added to our food supply. More than 10,000 chemical solvents, emulsifiers and preservatives are used in food processing. We are surrounded by environmental toxins and they can be found in everything that we eat, in everything that we drink and even in the air we breathe.

Worldwide pollution is responsible for 8.9 million deaths – or 1 in 7 deaths globally. Of the 8.4 million pollution-caused deaths in developing countries, air pollution was the leading offender. Forty-four percent of pollution-caused deaths resulted from household air pollution, such as cooking stoves that contaminate the air, and 38 percent were caused by ambient air pollution, including particulates from power plants, cars and trucks. The contamination of soil and food from heavy metals released by industry and mining accounted for 10 percent of pollution-caused deaths, while local water systems, polluted by sewage and industrial waste, made up 8 percent. Cancers, strokes, and heart and respiratory diseases are just some of the fatal health conditions that can result from exposure to environmental toxins.

Evidence of the negative impact of environmental toxicants on human health is on the rise. A growing body of research suggests that maternal exposure to environmental toxicants poses a risk to women's health as well as fetal and child health and development. We don't realize that we're being affected until we come down with a chronic disease after years of subtle and often consistent exposure to a combination of these toxins. It is almost impossible to pinpoint a specific environmental toxin as the source of illness. The increasing numbers of cancers, immune system disorders, neurological problems, chronic fatigue syndrome, multiple chemical sensitivities, allergies and hormonal disturbances are facing the nation.

Chemicals are used in every step of the process that puts food on our table: production, harvesting, processing, packing, transport, marketing and consumption and can be dangerous to our health. Some of these chemicals remain in our food and many persist in the environment and our bodies for decades to come. Preservatives are added to many processed foods including breads, cereals, and meat. Studies have found that additives are a source of headaches, nausea, weakness and difficulty in breathing. Children are at greater risk from chemicals found in food, water, dirt and air for several reasons. They're more at risk because for their size, they eat, drink and breathe more than grown-ups.

It's impossible in this day and age to avoid all environmental toxins. What you can do, however, is limit your exposure to them as much as possible. The first objective of bringing out this publication is to inform the general public of how environmental toxins can be harmful to human health. The second is to help the general public come to know the precautionary and preventive measures. The third is to provide information to the University students in Myanmar who are studying “Environmental Science”.

The author is grateful to former president of the Myanmar Academy of Arts and Science, Professor Dr. Thein Myint, for his wholehearted encouragement to write this book to be published by Myanmar Academy of Arts and Science, Ministry of Education.

The author is deeply indebted to Professor Thi-Ha, ex-Head of the Department of English, Yangon Institute of Education, for his valuable advice and unstinted efforts in editing and proof-reading of this book.

Professor Kyaw Myint Oo

4th May, 2018

A Profile of Professor Kyaw Myint Oo

Professor Kyaw Myint Oo, son of U Hla Oo and Daw Kyin Sein, was born in Pyay, Bago Division. He matriculated from the No (1) State High School, Pyay, in 1963. He obtained his BSc degree in Zoology in 1967 and his MSc degree in 1976 from the University of Yangon. In 1999, he was sent to the International Center for Environmental and Industrial Toxicology (ICEIT), to study Environmental Toxicology, Pollution Control and Management under the United Nations Development Program. He contributed his services to the state in the following capacities:

1968 – 1972	Demonstrator, Taunggyi College
1972 – 1978	Demonstrator, Institute of Medicine (1)
1978 – 1984	Head/Assistant Lecturer, Lashio College
1984 – 1986	Assistant Lecturer and Head, Taunggyi College
1986 – 1989	Head/Lecturer, Taunggyi Degree College
1989 – 1993	Lecturer, University of Yangon
1993 – 1995	Associate Professor/Head, University of Mawlamyine
1995 – 2001	Professor and Head, University of Mawlamyine
June, 2001 to November 2001	Principal, Bago Degree College
2001 – 2005	Rector, University of Taungoo
2005 – 2007	Rector, University of Dagon

He has supervised several research projects in the area of toxicity of pesticides on fish with respect to water pollution. He has published 45 health education articles dealing with environmental toxicology in annual magazines of various Universities, Newspapers and News Letters issued by the National Commission for Environmental Affairs and the International Centre for Environmental and Industrial Toxicology. Since 1976, he has published 27 research articles on Environmental Toxicology in various domestic and international research journals. He served as a member of editorial board, The Journal of Myanmar Zoological Society, University of Yangon from 1990 to 2003.

He has published three books: Environmental Science for M.Sc Students (2000), Instructions for Research Paper Presentation (2008) and Environmental Toxicology: Effect of Environmental Pollutants on Living Systems (2010). His major research areas include toxicity of pesticides on fish, environmental toxicology and pollution control. He had attended several domestic and international research conferences, workshops and seminars on Environmental Toxicology. He conducted seminars on "Environmental Toxicology" at various universities in Myanmar after his retirement. He is also conducting refresher courses on "Environmental Toxicology" to Zoology subject teaching staff of various universities under the Ministry of Education. He was awarded the Eminent Educator Prize from the Ministry of Education on World Teachers' Day 2012.

In 2008 he was appointed member of the Executive Committee, Myanmar Academy of Arts and Science, Ministry of Education. Currently he is a member, Steering Committee for Ph.D Programme, Zoology Department, University of Yangon; member, Yangon University Revitalization Committee, Ministry of Education; member, Pesticide Technical Committee, Pesticide Registration Board, Ministry of Agriculture, Livestock and Irrigation, Government of the Republic of the Union of Myanmar. He is also a member of the Network of Tropical Aquaculture and Fisheries Professionals (NTAFP), International Center for Living Aquatic Resources Management (ICLARM), Penang, Malaysia.

Table of Contents

1. Introduction.....	1
2. Environmental Toxicology and Environmental Health.....	5
2.1. Environmental Toxicology.....	5
2.1.1. Historical development.....	6
2.1.2. Acute Toxicity and Chronic	
2.1.3. The Median Lethal Dose.....	8
2.2. Environmental Health.....	8
2.2.1. Environmental health objectives.....	9
2.2.2. Why is Environmental Health important?	11
2.2.3. Emerging issues in Environmental Health.....	11
2.2.4. Environmental Health hazards.....	12
3. Environmental Toxins and Exposure to Humans.....	16
3.1. Introduction.....	16
3.2. Environmental Toxins in our Environment.....	17
3.3. Four Routes of Environmental Toxins' Exposure to Humans.....	19
3.4. Common Environmental Toxins.....	21
3.4.1. Aluminum.....	21
3.4.2. Air particulate matter.....	21
3.4.3. Arsenic.....	22
3.4.4. Asbestos.....	22
3.4.5. Benzene.....	23
3.4.6. BPA.....	24
3.4.7. Cadmium.....	24
3.4.8. Carbon Monoxide.....	25
3.4.9. Chlorine.....	26
3.4.10. Chloroform.....	27
3.4.11. Chromium.....	27
3.4.12. Diesel Exhaust.....	28
3.4.13. Dioxins.....	28
3.4.14. Excitotoxins (Food Additives).....	29
3.4.15. Formaldehyde.....	29

3.4.16. Genetically Modified Foods (GMO's).....	30
3.4.17. Lead	30
3.4.18. Mercury.....	32
3.4.19. Nitrogen Oxides	33
3.4.20. Ozone	33
3.4.21. PCBs (Polychlorinated biphenyls)	34
3.4.22. Pesticides	35
3.4.23. Phthalates	36
3.4.24. Radon	36
3.4.25. Secondhand Smoke	37
3.4.26. Sulfur dioxide	37
3.4.27. Volatile Organic Compounds (VOC's)	38
3.4.28. Microwaves and Electromagnetic Fields	38
3.4.29. Ultraviolet Radiation	41
3.4.30. Heavy Metals	41
3.5. Health Effects from Exposure to Toxic Substances.....	42
3.5.1. Respiratory Tract	43
3.5.2. Skin.....	45
3.5.3. Eyes.....	47
3.5.4. Central Nervous System.....	48
3.5.5. Liver	50
3.5.6. Kidneys.....	51
3.5.7. Blood.....	51
3.5.8. Spleen.....	53
3.5.9. Reproductive System.....	53
3.6. Toxic chemicals in our Food System.....	54
3.7. Ten Dangerous Everyday Things in your Home.....	57
3.8. More Toxic Sources You may not have Considered.....	60
3.9. Reducing Environmental Exposures for Children.....	62
3.10. Almost a Quarter of all Disease caused by Environmental Exposure.....	66
3.11. Conclusion.....	68
4. Environmental Pollution and Human health.....	70
4.1. Introduction	70
4.2. Causes of Environmental Pollution.....	71

4.2.1. Industries.....	72
4.2.2. Transportation	72
4.2.3. Agricultural activities.....	73
4.2.4. Trading activities	73
4.2.5. Residences	73
4.2.6. Pollution from cars, trucks, and other vehicles.....	74
4.2.7. Fossil fuel emissions from power plants	74
4.2.8. Water pollution is a major issue	74
4.2.9. Radiation	74
4.3. Effects of Environmental Pollution	75
4.3.1. Effects on human health	75
4.3.2. Effects on animals.....	76
4.3.3. Effects on plants.....	77
4.3.4. Effects on the ecosystem.....	77
4.3.5. Effects on environment.....	77
4.4. Toxic Pollution and Human Health	78
4.5. Various types of Environmental Pollution	83
4.5.1. Air Pollution	83
4.5.2. Water Pollution	88
4.5.3. Land Pollution	92
4.5.4. Soil Pollution	93
4.5.5. Noise Pollution	95
4.5.6. Thermal Pollution	97
4.5.7. Industrial Pollution	99
4.5.8. Light Pollution	101
4.5.9. Radioactive Pollution	103
4.5.10. Plastic Pollution	104
4.6. World's top 10 toxic pollution problems	107
4.6.1. Lead-Acid Battery Recycling	107
4.6.2. Mercury and Lead Pollution from Mining	107
4.6.3. Coal Mining (Sulphur Dioxide and Mercury Pollution)...	108
4.6.4. Artisanal Gold Mining (Mercury Pollution)	108
4.6.5. Lead Smelting	108
4.6.6. Pesticides Pollution from Agriculture and Storage	109
4.6.7. Arsenic in Ground Water	109

4.6.8. Industrial Waste Water	109
4.6.9. Chromium Pollution (Dye Industry)	109
4.6.10. Chromium Pollution (Tanneries)	110
4.7. Industrial Pollution in Developing Countries	110
4.8. Common Indoor Air Pollutants	124
4.9. Top 10 Countries killing the Planet	127
4.10. Pollution is the Leading Cause of Death in the Developing world	131
4.11. Conclusion.....	132
5. Environmental Disasters.....	135
5.1. Introduction.....	135
5.2. Different Type of Environmental Disasters.....	137
5.2.1. Agricultural Disasters.....	138
5.2.2. Industrial Environmental Disasters.....	138
5.2.3. Mining Disasters.....	139
5.2.4. Oil Industry.....	139
5.2.5. Nuclear.....	140
5.2.6. Air.....	141
5.2.7. Land.....	141
5.2.8. Water.....	142
5.2.9. Marine.....	142
5.3. Twenty five biggest Man made Environmental Disasters.....	142
5.3.1. The Great Smog of London, 1952.....	143
5.3.2. The Chernobyl Nuclear Explosion, 1986.....	144
5.3.3. The Bhopal Disaster, 1984.....	145
5.3.4. The Shrinking of the Aral Sea, 1989 – 2009.....	146
5.3.5. Baia Mare Cyanide Spill, 2000.....	147
5.3.6. E-waste in Guiyu, China, 2005.....	149
5.3.7. The Seveso Disaster, 1976.....	151
5.3.8. Methylmercury Poisoning (Minamata Disease), 1956.....	152
5.3.9. Gulf of Mexico Dead Zone disaster.....	155
5.3.10. The Love Canal Disaster, 1978.....	156
5.3.11. The Al-Mishraq Fire, 2003.....	158

5.3.12.	Ecocide in Vietnam during Vietnam War.....	159
5.3.13.	Amoco Cadiz Oil Spill, 1978.....	160
5.3.14.	Deep water horizon (BP) oil spill, 2010.....	162
5.3.15.	Libby, Montana Asbestos Contamination,1979–2011.....	164
5.3.16.	The Sidoarjo Mudflow and the Muddiness of an Environmental Disaster, 2006.....	165
5.3.17.	The Palomares Incident, 1966.....	167
5.3.18.	Darvasa gas crater “Door to Hell”,1971	168
5.3.19.	The Kuwait Oil Fire, 1991.....	169
5.3.20.	The Three Mile Island Nuclear Explosion, 1979.....	170
5.3.21.	Castle Bravo, 1954.....	171
5.3.22.	Jilin Chemical Plant Explosions in China, 2005.....	173
5.3.23.	Pacific Gyre Garbage Patch.....	173
5.3.24.	The Exxon Valdez Oil Spill, 1989.....	174
5.3.25.	TVA Kingston Fossil Plant Coal Fly Ash Slurry Spill,2008.....	176
5.4.	Disaster Risk Reduction and Disaster Risk Management.....	178
6.	Case Studies of some Man made Environmental Disasters.....	183
6.1.	Case Study of Bhopal Plant Disaster, 1984.....	183
6.1.1.	Introduction.....	183
6.1.2.	The initial effects of exposure.....	185
6.1.3.	Long-term health effects.....	186
6.1.4.	Health Care.....	188
6.1.5.	Environmental Rehabilitation.....	189
6.1.6.	Ongoing contamination.....	190
6.1.7.	Conclusion	191
6.2.	Case Study of Chernobyl disaster, 1986.....	192
6.2.1.	Introduction.....	192
6.2.2.	Environmental effects.....	194
6.2.3.	Residual radioactivity in the environment.....	196
6.2.4.	Groundwater contamination.....	197
6.2.5.	Flora and fauna.....	197
6.2.6.	Human impact	197
6.2.7.	Thyroid cancer	198
6.2.8.	Other health disorders.....	199

6.2.9. Chernobyl human mutations.....	200
6.2.10. Deaths due to radiation exposure	202
6.2.11. Abortion requests.....	203
6.2.12. Radioactive materials and waste management.....	204
6.2.13. Conclusion.....	205
6.3. Case Study of the Seveso Disaster, 1976.....	206
6.3.1. Introduction.....	206
6.3.2. Immediate health effects.....	207
6.3.3. Long-term health effects.....	211
6.3.4. Conclusion.....	211
6.4. Case Study of Love Canal Toxic Waste Disaster, 1978.....	213
6.4.1. Introduction.....	213
6.4.2. Contaminants.....	215
6.4.3. Health effects.....	215
6.4.4. Evacuations.....	222
6.4.5. Relocation of residents.....	222
6.4.6. Conclusion.....	223
6.5. Case Study of Methylmercury Poisoning in Japan, 1956	224
(Minamata disease)	
6.5.1. Introduction.....	224
6.5.2. Causes of Minamata Disease.....	225
6.5.3. Symptoms and Signs of Minamata Disease.....	228
6.5.4. Diagnosis of Minamata Disease.....	229
6.5.5. Treatment for Minamata Disease.....	229
6.5.6. Congenital Minamata Disease.....	230
6.5.7. Outbreak of Niigata Minamata Disease.....	231
6.5.8. Epidemiology.....	232
6.5.9. Conclusion.....	232
6.6. Case Study of Iraq Poison Grain Disaster, 1971.....	233
6.6.1. Introduction.....	233
6.6.2. Context.....	234
6.6.3. Causes.....	235
6.6.4. Symptoms, outbreak and treatment.....	236
6.6.5. Effects.....	237
6.6.6. Conclusion.....	239

6.7.	Case Study of Air Pollution in London, 1952.....	239
6.7.1.	Introduction.....	239
6.7.2.	Sources of pollution.....	241
6.7.3.	Weather.....	242
6.7.4.	Effect on London.....	242
6.7.5.	Health effects.....	243
6.7.6.	Environmental impact.....	244
6.7.7.	Conclusion.....	244
7.	Environmental Toxins and Human Cancers.....	246
7.1.	Introduction.....	246
7.2.	Sources of Cancer-inducing and Cancer-promoting Toxins.....	246
7.3.	How Toxins produce Cancer and Cancer Prevention.....	249
7.3.1.	DNA Damage.....	250
7.3.2.	Liver Detoxification Systems.....	251
7.3.3.	Immune Surveillance.....	253
7.3.4.	Endocrine Disruptors.....	254
7.3.5.	Loss of Apoptosis.....	255
7.4.	Conclusion.....	257
8.	Treating Cancer with Foods.....	259
8.1.	Introduction.....	259
8.2.	Worldwide Cancer Mortality.....	260
8.2.1.	Mortality in Males.....	261
8.2.2.	Mortality in Females.....	262
8.2.3.	World Health Rankings with respect to Cancer Mortality.....	264
8.3.	The Top Cancer-causing Foods.....	264
8.3.1.	Red and processed meat.....	264
8.3.2.	Cured, pickled or salty foods.....	265
8.3.3.	Burnt or barbecued foods.....	265
8.3.4.	Fat.....	265
8.3.5.	Peanuts.....	266
8.3.6.	French fries.....	266
8.3.7.	Alcohol.....	266
8.3.8.	Genetically-modified organisms (GMOs).....	267

8.3.9. Microwave popcorn.....	267
8.3.10. Soda pop.....	267
8.3.11. Conventional apples, grapes, and other 'dirty' fruits.....	267
8.4. The Anti-Cancer Diets.....	268
8.4.1. Cancer prevention diet-1.....	268
8.4.2. Cancer prevention diet -2.....	268
8.4.3. Cancer prevention diet -3.....	270
8.4.4. Cancer prevention diet -4.....	271
8.4.5. Cancer prevention diet -5.....	272
8.4.6. Cancer prevention diet -6.....	273
8.5. Best foods for cancer prevention	274
8.5.1 Grapefruit.....	274
8.5.2. Peanuts and peanut butter.....	274
8.5.3. Berries.....	275
8.5.4. Sweet potatoes.....	275
8.5.5. Wild salmon.....	275
8.5.6. Ground flaxseed.....	275
8.5.7. Turmeric	275
8.5.8. Tea.....	275
8.5.9. Cruciferous vegetables.....	276
8.5.10. Pomegranates.....	276
8.6. Conclusion.....	276
9. Thyroid Diseases and Environmental Toxins	278
9.1. Introduction	278
9.2. Thyroid Gland	279
9.2.1. Anatomy of Thyroid gland.....	279
9.2.2. Functions of the Thyroid Gland.....	279
9.3. Thyroid Disease.....	280
9.3.1. Goiters	280
9.3.2. Thyroiditis.....	283
9.3.3. Thyroid cancers.....	283

9.4. Causes of Thyroid Diseases	284
9.4.1. Toxins that affect the thyroid hormones synthesis, metabolism and action.....	284
9.4.2. Thyroid diseases sometimes caused by problems in the thyroid gland itself.....	285
9.4.3. Radiation release from nuclear bomb and accidents.....	286
9.5. Symptoms and Complications	287
9.6. Making the Diagnosis	289
9.7. Treatment	289
9.8. Prevention of Thyroid diseases	291
9.8.1. Try your best to avoid environmental toxins.....	291
9.8.2. Protect yourself against X-rays.....	292
9.8.3. Stop smoking.....	292
9.8.4. Test for and treat thyroid antibodies.....	292
9.8.5. Detox to save your thyroid.....	293
9.8.6. Too much soy is not healthy.....	293
9.8.7. Try Selenium: A Thyroid Super-Nutrient.....	293
9.8.8. Keep Potassium Iodide on hand for a Nuclear Emergency.....	293
9.8.9. Watch out for Fluoride.....	294
9.9. Conclusion	294
10. Effects of Pesticides on Human Health	297
10.1. Introduction	297
10.2. Pesticide Residues	298
10.2.1. Organochlorine residues in fish from LakeVictoria, Kenya.....	298
10.2.2. Analysis of environmental chemical residues in products of emerging aquaculture industry in Uganda....	299
10.2.3. Organochlorine pesticide residues in paddy fish in Malaysia and the associated health risk to farmers.....	299
10.2.4. Residual Toxicity of different insecticides to <i>Channa punctata</i>	300
10.2.5. Residues in fruits and vegetables.....	301
10.2.6. Residues of Cypermethrin and Methamidophos on cauliflower at various intervals after treatment.....	302
10.2.7. Pesticide residues in organisms of Malaysian waters.....	302

10.2.8. Levels of organochlorine pesticide residues in meat.....	303
10.2.9. Organochlorine Pesticides BHC and DDE in human blood in and around Madurai, India.....	304
10.2.10. Organochlorine pesticide residues in human milk of a Hmong hill tribe living in Northern Thailand.....	305
10.2.11. Evaluation of organochlorine pesticide residues in Human serum from an urban and two rural Population in Portugal.....	305
10.3. Effects of Pesticides on Human Health	306
10.3.1. Asthma.....	307
10.3.2. Birth Defects.....	307
10.3.3. Neurological Effects.....	307
10.3.4. Cancers.....	308
10.3.5. Hormone Disruption.....	309
10.4. Home Food Preparation to Reduce Exposure to Pesticide Residues.....	310
10.5. The Health Benefits of eating Organic Food	312
10.6. Conclusion	313
11. Pesticide Residue Monitoring and Food Safety.....	315
11.1. Introduction.....	315
11.2. Maximum Residue Limits (MRLs).....	316
11.3. United Nations Codex Alimentarius Commission.....	317
11.4. Safe Level of a Pesticide Residue.....	318
11.5. Monitoring the Residue Levels of Pesticides in Food.....	318
11.6. Maximum Residue Limits (MRLs) of some Agricultural and Environmental Chemical Contaminants on Fish, Mollusks, Crustaceans and Food.....	319
11.7. Analysis of Environmental Chemical Residues in Products of emerging Aquaculture Industry in Uganda.....	322
11.8. Trade Problems arising from differing Maximum Residue Levels.....	322
11.9. Conclusion.....	323

12.	Effects of Mosquito Coil Smoke Inhalation on Human Health	326
12.1.	Introduction	326
12.2.	Mosquito Coil Ingredients	327
12.3.	Side Effects of using Mosquito Coil	329
12.4.	Exposure to Mosquito Coil Smoke may be a Risk Factor for Lung Cancer in Taiwan	329
12.5.	Mosquito coil exposure associated with small cell lung cancer	329
12.6.	Toxicological Effects of Inhaled Mosquito Coil Smoke. on Rat Spleen	334
12.7.	Toxic Effects of Mosquito Coil Smoke on Rats	335
12.8.	Mosquito Coil Smoke Inhalation Effects on Kidney of Albino Rat	335
12.9.	Conclusion	338
13.	Harmful Effects of Genetically Modified Organisms(GMOs)	341
13.1.	Introduction	341
13.2.	Effects of Genetically Modified Foods on Human Health	342
13.2.1.	Cancer	342
13.2.2.	Super viruses	342
13.2.3.	Antibiotic resistance.....	343
13.2.4.	Birth Defects and Shorter Life Spans.....	343
13.2.5.	Interior Toxins.....	344
13.2.6.	Decreased Nutritional Value.....	345
13.2.7.	Food Allergy.....	345
13.3.	Effects on Environment	346
13.3.1.	Soil Sterility and Pollution.....	346
13.3.2.	Super weeds.....	346
13.3.3.	Destruction of Forest Life.....	347
13.3.4.	Terminator Trees.....	347
13.3.5.	Super pests.....	348
13.3.6.	Animal Bio-invasions.....	348
13.3.7.	Killing Beneficial Insects.....	348
13.3.8.	Poisonous to Mammals.....	349

13.3.9. Animal Abuse.....	349
13.3.10. Genetic Pollution.....	349
13.3.11. General Economic Harm to Small Family Farms.....	350
13.3.12. Losing Natural Pesticides.....	351
13.3.13. Monopolization of Food Production.....	352
13.3.14. Impact on Long -Term Food Supply.....	352
13.4. Conclusion	353
14. Effects of Food Additives on Health	354
14.1. Introduction	354
14.2. Health Effects of Food Additives	354
14.3. Some Food Additives and their Side-Effects	355
14.4. Who are affected?	359
14.5. Nutritional and Toxic Chemical Influences on Behavior	359
14.6. Food Additives and Malnutrition	360
14.7. Subclinical Malnutrition in Reproduction	361
14.8. Conclusion	362
15. Effects of Formaldehyde on Health	363
15.1. Introduction	363
15.2. Industrial Applications	363
15.3. Contaminants in Foods	364
15.4. Effects on Human Health	365
15.5. How is the General Population Exposed to Formaldehyde?	366
15.6. Can Formaldehyde Cause Cancer?.....	366
15.7. What has been done to protect workers from formaldehyde?.....	367
15.8. How can People Limit Formaldehyde Exposure in their Home?	367
15.9. Conclusion	368
16. Arsenic Toxicity and Human Health	370
16.1. Introduction	370
16.2. Sources of Exposure	370
16.2.1. Background Exposure.....	371
16.2.2. Food Exposure.....	371

16.2.3. Water Exposure.....	372
16.2.4. Industrial processes.....	372
16.2.5. Tobacco.....	372
16.3. Health Effects	373
16.3.1. Acute effects.....	373
16.3.2. Long-term effects.....	373
16.4. Magnitude of the problem	375
16.5. Reducing Exposure	376
16.6. Prevention and Control	376
16.7. Conclusion	377
17. Lead Poisoning and Health	379
17.1. Introduction	379
17.2. Sources and Routes of Exposure	379
17.3. Health Effects of Lead Poisoning	380
17.3.1. Complications.....	381
17.3.2. Kidneys.....	381
17.3.3. Cardiovascular system.....	382
17.3.4. Reproductive system.....	382
17.3.5. Nervous system.....	382
17.4. Symptoms	384
17.5. Causes of Lead Poisoning	385
17.6. How is Lead Poisoning Diagnosed?.....	386
17.7. Treatment	386
17.8. Prevention	387
17.9. Conclusion	387
18. Health Effects of Exposures to Mercury	389
18.1. Introduction	389
18.2. Exposure to Mercury	390
18.3. Health Effects of Mercury Exposure	392
18.4. How to Reduce Human Exposure from Mercury Sources.....	394
18.5. Conclusion	395
References	397

List of Figures

Figure 4.1. Health effects of pollution.....	76
Figure 4.2. Sources of Air Pollutants.....	84
Figure 4.3. Sources of emissions of Air Pollutants.....	85
Figure 4.4. Girl walks through smog in Beijing, where small-particle pollution is 40 times over International Safety Standard.	85
Figure 4.5. Air pollution in India.....	86
Figure 4.6. Health effects of Air Pollution.....	86
Figure 4.7. The 20 worst cities worldwide for air pollution.....	87
Figure 4.8. Contamination of water bodies.....	89
Figure 4.9. Water pollution killing fish species.....	89
Figure 4.10. Pollutants entering the oceans.	91
Figure 4.11. In 2050, more people will be at high risk of water pollution due to increasing BOD, Nitrogen and Phosphorous.	91
Figure 4.12. Land Pollution.....	92
Figure 4.13. Soil Pollution.....	93
Figure 4.14. Noise pollution in India.....	96
Figure 4.15. When Manufacturing plants release back heated water to the river or ocean, the water temperature rises sharply.....	98
Figure 4.16. The releasing of smoke, material wastes and toxic byproducts that eventually end up in the environment thereby causing pollution.....	100
Figure 4.17. The releasing of wastes and pollutants generated by industrial activities into the natural environment.....	100
Figure 4.18. Excessive lighting.....	102
Figure 4.19. Light pollution in Europe.....	102
Figure 4.20. Large amount of radioactive waste is generated from nuclear reactors used in nuclear power plant.....	103
Figure 4.21. Plastic Pollution.....	105
Figure 4.22. Albatross killed by excessive plastic ingestion in Midway Islands (NorthPacific).....	105
Figure 4.23. Plastic pollution by numbers.....	106

Figure 4.24.	Ozone levels in two zones of Mexico City. One-hour daily maximum by month, 1994	117
Figure 4.25.	Particulates (PM ₁₀) in two zones of Mexico City, 1988-1993	118
Figure 4.26.	Air lead levels in two zones of Mexico City, 1988-1994.....	118
Figure 5.1.	The Great Smog of London, 1952.....	143
Figure 5.2.	A picture of a nuclear blast. Chernobyl witnesses reported seeing 'blue twinkles' in the wreckage of Reactor No.4, 1986.....	144
Figure 5.3.	The Chernobyl nuclear reactor after the disaster. Reactor No.4 (centre).....	145
Figure 5.4.	Survivors suffering blindness after Bhopal Disaster, 1984.....	146
Figure 5.5.	Comparisons of the shrinking of Aral Sea (1989, 2003 and 2009).	147
Figure 5.6.	Cyanide spill in Baiba Mare, Romania, 2000.....	148
Figure 5.7.	Fishermen hauling dead fish ashore following the Baia Mare cyanide spill in 2000.....	148
Figure 5.8.	Chinese town of Guiyu built its economy on recycling waste from overseas. Largest electronic waste (e-waste) site in the world.....	150
Figure 5.9.	Primitive recycling operations in Guiyu are toxic and dangerous to workers' health with 80% of children suffering from lead poisoning.....	151
Figure 5.10.	Hazardous effect of the Seveso Disaster, 1976.....	152
Figure 5.11.	A severe case of Minamata Disease.....	153
Figure 5.12.	Shinobu Sakamoto a sufferer of fetal Minamata disease speaks at a news conference.....	154
Figure 5.13.	Minamata disease suffering patients got loss of motor functions and uncontrollable limb movements.....	154
Figure 5.14.	The Gulf of Mexico “Dead Zone”	155
Figure 5.15.	Gulf of Mexico dead zone in July 2017.....	156
Figure 5.16.	Love Canal dump site located near Niagara Fall, 1978.....	157
Figure 5.17.	Al Mishraq Fire 2003, Man-Made Disaster.....	158
Figure 5.18.	Destruction of the farmland and rice paddies by spraying a variety of herbicides during the Vietnam War.....	159

Figure 5.19. Herbicide disaster in Southeast Asia during the Vietnam War	160
Figure 5.20. Sinking of the Amoco Cadiz in Portsall, France in March, 1978.....	161
Figure 5.21. Amoco Cadiz spill was the largest recorded spill in History, 1978.....	161
Figure 5.22. BP oil spill explosion, 2010.....	163
Figure 5.23. Total extent of oil spill, 68,000 square miles in the Gulf of Mexico, 2010.....	163
Figure 5.24. Large numbers of dead dolphins began washing ashore in Alabama and Mississippi in 2010.....	164
Figure 5.25. Libby asbestos contamination, 1979–2011.....	165
Figure 5.26. Mud Flow buried entire villages, East Java, Indonesia, 2006.....	166
Figure 5.27. Mud and steam are spewing from the earth on the island of Java Indonesia, leaving thousands of people homeless, 2006.....	166
Figure 5.28. Statues honouring the mudflow victims, Java, Indonesia, 2006.....	167
Figure 5.29. The B52G was carrying four Mk28 hydrogen bombs. Two of the non-nuclear explosives on the bombs went off when they hit the ground, 1966.....	168
Figure 5.30. Darvasa gas crater, nicknamed the ‘door to hell’, in Turkmenistan has been burning for over 40 years.....	169
Figure 5.31. The Kuwait Oil Fires, 1991.....	170
Figure 5.32. An accident at Three Mile Island, a nuclear power plant located near Harrisburg, Pennsylvania, led to the meltdown of the core reactor, 1979.....	171
Figure 5.33. Cloud top rose and peaked at 130,000 feet after only six minutes after explosion of hydrogen bomb, 1954.....	172
Figure 5.34. Smoke rised into the sky after explosions at a chemical plant in Jilin City in northeast China, 2005.....	173
Figure 5.35. The Great Pacific Garbage Patch.....	174
Figure 5.36. An oil skimming operation works in a heavy oil slick near Latouche Island on April 1, 1989.	175

Figure 5.37.	The Exxon Valdez Oil Spill caused deaths as many as 250,000 seabirds, at least 2,800 sea otters 300 harbor seals, 247 Bald Eagles, and 22 Orcas, and an unknown number of salmon and herring, 2008.	175
Figure 5.38.	Aerial photograph of site taken the day after the event of Coal Fly Ash Slurry Spill, 2008	177
Figure 5.39.	The wet fly ash engulfed this house, one of 12 damaged in the spill, 2008.....	177
Figure 6.1.	Bhopal gas tragedy, 1984.	183
Figure 6.2.	The accidental release of 40 metric tons of methyl isocyanate from a Union Carbide pesticide plant in the heart of Bhopal.....	184
Figure 6.3.	Poison gas leaking from a Union Carbide pesticide factory in Bhopal spread throughout the city, killing thousands of people outright.....	185
Figure 6.4.	Children were born with mental and physical disabilities in Bhopal.....	188
Figure 6.5.	The meltdown of Chernobyl created the largest uncontrolled release of radioactive materials into the environment of any civilian operation in history, 1986.....	193
Figure 6.6.	Piglet with dipygus on exhibit at the Ukrainian National Chernobyl Museum.....	195
Figure 6.7.	Birth defects and Chernobyl mutations from radiation, Chernobyl Power Plant Disaster.....	201
Figure 6.8.	Deformed child nicknamed 'the Chernobyl Child' in a special school for abandoned children Belarus.(1988)....	202
Figure 6.9.	Industrial accident occurred at Pesticide plant in the town of Seveso, 1976.....	206
Figure 6.10.	21,000 tons of hazardous chemical waste put into Love Canal. Important examples of groundwater pollution in the U.S. was the Love Canal tragedy in Niagara Falls, New York.....	214
Figure 6.11.	The release of methylmercury in the industrial wastewater from the Chisso Corporation's chemical factory, which continued from 1932 to 1968.....	224

Figure 6.12.	Minamata disease is caused through the absorption of methyl mercury into the human body through various environmental sources, primarily through seafood.....	226
Figure 6.13.	A sack of "pink grain". Grain treated with a methylmercury fungicide, 1971.....	234
Figure 6.14.	Incidence of cases and fatalities, by age group, Iraq Poison Grain Disaster, 1971.....	238
Figure 6.15.	Great Smog of London, 1952.	240
Figure 6.16.	The smoke belching from millions of London's chimney pots, 1952.....	241
Figure 6.17.	Patient suffering from respiratory tract infections. Air pollution in London, 1952.....	243
Figure 8.1.	Estimated worldwide cancer mortality 2008 (with comparison to UK 2008).....	261
Figure 8.2.	The ten most common cancer deaths in males worldwide 2008 (with comparison to UK 2008).....	262
Figure 8.3.	The ten most common cancer deaths in females worldwide 2008 (with comparison to UK 2008)	263
Figure 9.1.	Anatomy of Thyroid and Parathyroid glands.....	279
Figure 9.2.	Goiter (Enlarged Thyroid Gland).....	281
Figure 9.3.	Hashimoto's disease.	281
Figure 9.4.	Multinodular goiter.....	282
Figure 9.5.	Solitary thyroid nodule.	283
Figure 9.6.	Cancer (Tumor) cells in Thyroid gland.....	284
Figure 12.1.	Representative images from radiography and chest CT revealing the presence of a mass in the upper lobe of the left lung.....	331
Figure 12.2.	Representative images from radiography and chest CT revealing the presence of a mass in the upper lobe of the left lung and enlarged lymph nodes.....	332
Figure 12.3.	Representative images from radiography and chest CT revealing the presence of a mass in the middle lobe of the right lung, pleural effusion and enlarged lymph nodes.....	333
Figure 12.4.	Histological analysis of endobronchial biopsy specimens from cases one, two and three	334

Figure 12.5. Photomicrograph of the kidney from the control group (A) showing renal corpusles lined by parietal squamous epithelium with central normal glomerulus surrounded by bowman’s space	336
Figure 12.6. Photomicrograph of cortex of kidney from group (B) showing degenerated glomerulus.....	337
Figure 12.7. Photomicrograph of the kidney from the group C showing hemorrhage and cellular infiltrate in interstitium of kidney..	337
Figure 12.8. Photomicrograph of kidney from group (C) showing renal fibrosis, congested blood vessel and tubular necrosis.....	338

List of Tables

Table 2.1. Selected major environmental disease outbreaks.....	14
Table 3.1. Examples of Industrial Toxicants that Produce Disease of the Respiratory Tract.....	44
Table 3.2. Human Exposure to Environmental Chemicals	55
Table 4.1. Major sources of outdoor air pollutants	112
Table 4.2. Summary of short-term exposure-response relationship of PM ₁₀ with different health effects indicators ...	114
Table 4.3. Health outcomes associated with changes in peak daily ambient ozone concentration in epidemiological studies.	115
Table 6.1. Contaminants dumped in the landfill at the Love Canal Site (1979)	216
Table 6.2. List of some chemicals identified at the Love Canal site and the effects on human.....	218
Table 6.3. Congenital Malformations among children from the Love Canal.....	219
Table 6.4. Ten compounds found in air samples taken from basements of 88 houses peripheral to those built adjacent to the Love Canal landfill site.....	221
Table 7.1. Toxins that induce or promote cancers.....	248
Table 7.2. Nutrients that reduce DNA damage.....	251
Table 7.3. Nutrients that prevent cancer by regulating the liver's detoxification enzymes.....	252
Table 7.4. Nutrients that boost immune surveillance.....	253
Table 7.5. Nutrients capable of inhibiting endocrine-disrupting pollutants.....	255
Table 7.6. Nutrients that promote or restore apoptosis	256
Table 8.1. Myanmar Cancer Ranks by Type Per 100,000 Population. WHO data (2011).....	264
Table 10.1. Residual toxicity of 10 different insecticides to <i>Channa punctata</i>	300
Table 10.2. Residue in fruits and vegetables.....	301

Table 10.3. Residues of Cypermethrin and Methamidophos on cauliflower at various intervals after treatment.....	302
Table 10.4. Pesticide residues in organisms of Malaysian waters.....	303
Table 10.5. Levels of pesticide residues in beef fat from Buoho, Ghana (ug/kg).....	304
Table 10.6. Differences between conventional and organic farming.....	312
Table 11.1. Maximum Residue Limits (MRLs) (ppm) of some Agricultural Chemicals on fish in Japan.....	319
Table 11.2. Environmental chemical contaminants. Maximum Residue Limits (MRLs) in fish, mollusk and crustaceans. ...	320
Table 11.3. Some Maximum Residue Limits (MRLs) in mg/kg on onions registered for use in Australia	320
Table 11.4. Analysis of wild caught fish for trace elements and persistent organic pollutants.....	321
Table 11.5. Banned pesticides in Myanmar, 2010.....	324

1. Introduction

There is an ever-increasing use of chemicals and exposure to these chemicals and the resultant toxicity has long been a concern in humans. From a historical perspective, toxicology has always been associated with poisons. The word toxicology is derived from two Greek terms: *toxicon*—a poisonous substance into which arrowheads were dipped, and *toxicos*—a bow. A poison can be defined as any substance which has a harmful or deleterious effect on a living system. The earliest poisons that were used for hunting, waging war, and official execution consisted of plant extracts, animal venoms, and minerals, including arsenic, lead, opium, and cyanogenic glycosides. It is inevitable today that poisons include both harmful naturally occurring substances, either from animals, plants, or microorganisms, and synthetic chemicals. The former are generally termed toxins and the latter are better known as toxicants. The toxicants include synthetic chemicals/pharmaceuticals, which have economic benefits such as pesticides or drugs, and also those resulting from anthropogenic activities such as by-products of incomplete combustion.

Environmental toxicology is concerned primarily with the movement and impact of toxicants and their metabolites in the environment, in food chains, and upon the structure and function of biological systems. The biological systems include any living systems such as human and other mammals, plants, other organisms, and their habitats. One of the major problems arising from the environmental threat is the loss of biodiversity. Biodiversity, coined from biological diversity, is a term relating to species, genes and habitats. The diversity occurs in species from plant and animal kingdoms, fungi, bacteria, protozoa, and viruses, as well as the ecological complex such as the rain forest, tundra, and coral reefs. For the purpose of this theme, emphasis will be on the harmful effects of environmental toxicants on human health, rather than ecological changes in the environment itself. In this respect, another relatively new and related field called environmental health plays a major role in contributing knowledge on the adverse health effects of environmental stressors.

In a meeting of WHO European member states in 1993, a definition for environmental health was proposed:

Environmental health comprises of those aspects of human health, including quality of life, that are determined by physical, biological, social and psychosocial factors in the environment. It also refers to the theory and practice of assessing, correcting and preventing those factors in the environment that can potentially affect adversely the health of present and future generations.

Environmental toxicology is a relatively new field and perhaps its rapid expansion and progress is attributed to Rachel Carson (1962), who wrote *Silent Spring*. Carson's emphasis then was to stop the wide and indiscriminate use of pesticides and other chemicals. This had the impact of awakening the public to the dangers of polluting the environment with chemicals. In a nutshell, she delivered her high impact message: For as long as man has dwelt on this planet, spring has been the season of rebirth, and the singing of birds. Now in some parts of America spring is strangely silent, for many of the birds are dead—incidental victims of our reckless attempt to control our environment by the use of chemicals that poison not only the insects against which they are directed but the birds in the air, the fish in the rivers, the earth which supplies our food, and, inevitably (to what degree is still unknown), man himself.

This led to the establishment, from 1970 onwards, of many regulatory laws to protect human health and the environment by the US Environmental Protection Agency. The awakening of environmental awareness in industry has led to the development of yet another important field called occupational medicine. This field involves understanding of the diseases which occur as a result of human exposure, and which are occupational or industrial in origin, as well as ways to regulate and prevent the diseases. Indeed, in the last few centuries, toxicologic plagues and disaster have become increasingly common events either accidental or incidental in nature.

Many chemicals are encountered by humans, either incidentally because they are in the atmosphere, or by contact during occupational or recreational activities, or by ingestion of food additives. When atmospheric

pollution is the source of chemicals, it is obvious that all exposures are incidental, whereas exposure to chemicals used in industry is limited by industrial hygiene and occupational safety and the health regulations and practices of that particular industry. It is conceivable that some chemicals may be inadvertently released into the environment and therefore be hazardous to human health. With the increasing rate of production and the growing use of industrial chemicals, it seems that no occupation is entirely free of exposure to a variety of chemicals capable of producing undesirable effects on biologic tissues.

At the present time, more than 10,000 chemical entities contribute to some 500,000 products that are used in industries to produce many durable and non-durable products. Some toxicity data are available on many of these chemicals and a majority of chemical manufacturers find it necessary to obtain at least a minimal amount of toxicity information on each and every compound they use.

Chemicals added to the feed of animals may be drugs intended for therapeutic purposes, or pesticides and insecticides. Limitations have been placed on the amounts that may be used, and on the time in which such additives must be withdrawn, if the animals are to be used for human food. The use of pesticides and insecticides in agriculture has enabled the farmer to produce more and better products, but present the possibility of residues of these chemicals being present when the food is consumed. The addition of chemical preservatives to processed foods has made possible the existence and prolonged life of certain food preparations. The study of the limitations that must be observed with regards to food additives, and the evaluation of safety from the harmful effects of such chemicals is the concern of environmental toxicologists.

A continuing trend toward urbanization of human populations has accompanied the industrialization of the world. In the United States, more than half the population lives on less than 5 percent of the total land space. Pollution of water by products of industry and improper sewage disposal, as well as pollution of the atmosphere by industry and by automobiles in the areas of concentrated populations, has created a distinct public health hazard. Although humanity has depended upon the enormous volume of

atmosphere to dilute the pollutant concentrations to below effective levels, it is now apparent that atmospheric dilution is not unlimited, but rather is dependent upon weather conditions and the amount and extent of pollution that is created.

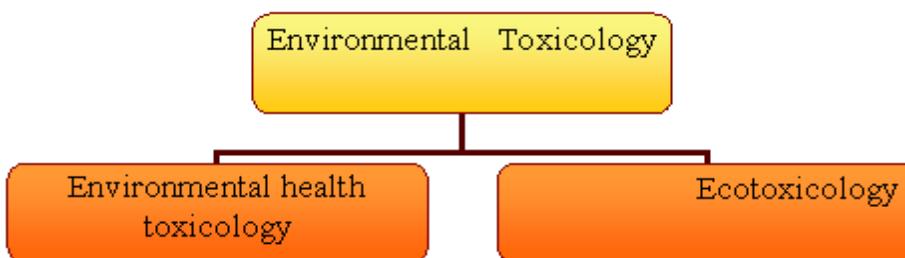
Pollution varies from gases to aerosols to dusts, and from carbon monoxide to alkalis and acids. Such pollution of the air and water is not only aesthetically undesirable, but exposes large populations to an unlimited variety of potentially harmful chemicals capable of producing known acute harmful effects and possible chronic debilitating illness in humans.

Environmental toxicology is therefore that branch of toxicology dealing with incidental exposure of biologic tissue, and more specifically human tissue, to chemicals that are basically contaminants to environment, food, or water. It is the study of the causes, conditions, effects, and limits of safety of such exposure to chemicals.

2. Environmental Toxicology and Environmental Health

2.1. Environmental Toxicology

Environmental toxicology is a multidisciplinary field of science concerned with the study of the harmful effects of various chemical, biological and physical agents on living organisms. Among its primary interests are the assessment of toxic substances in the environment, the monitoring of environments for the presence of toxic substances, the effects of toxins on biotic and abiotic components of ecosystems, and the metabolism and biological and environmental fate of toxins. Environmental toxicology can be divided into two subcategories:



Environmental health toxicology is the study of the adverse effects of environmental chemicals on human health. **Ecotoxicology** is a sub discipline of environmental toxicology concerned with studying the harmful effects of toxicants at the population and ecosystem levels.

To live in the 21st Century means to live in a toxic world, where **we are exposed daily to numerous environmental toxins and pollutants**. **Environmental toxins** are on the increase and pose a problem in the form of very serious health risks, as thousands of toxic substances find their way into our air, water and the soil in which we grow our food.

We spend our days inhaling **pollutants such as car fumes and cigarette smoke**. We drink water that has been thoroughly treated with chemicals, and eat food that is grown in toxic soil, pumped with hormones and packaged with preservatives.

While our livers, kidneys, skin and lymphatic systems work round the clock to **eliminate these dangerous toxins from our body**, they very often just cannot keep up, and the result is a build up of poisons in the system which destroy body tissue, damage organs, depress the immune system and leave the door open to a number of serious illnesses.

2.1.1. Historical development

Environmental toxicology is a relatively young field, with its origins in the mid-20th century. The modern science of toxicology, on the other hand, was born in the early 19th century, and by the later decades of that century, some scientists had begun to consider the effects of toxic substances that had been released into the environment. But awareness of environmental pollutants did not increase markedly until the publication of American biologist Rachel Carson's *Silent Spring* in 1962. Despite strong opposition from the chemical industry, which felt that Carson's work unfairly attacked their products, Carson highlighted the environmental side effects from the use of pesticides such as DDT. The book suggested that pollutants used in one area could quickly affect neighbouring areas and that the destruction of a particular part of the food chain upsets the balance of nature, leading to the destruction of an ecosystem. In 1969 scientist René Truhart coined the term *ecotoxicology* to describe the study of the toxic effects of pollutants on the biological components of ecosystems. Although narrower in scope, ecotoxicology played an important role in the development of environmental toxicology.

In the 1970s scientists concerned with toxins in the environment increasingly focused their research on the impacts of agents of biological warfare (e.g., Agent Orange), on industrial pollution, and on discharge from mines. In the next decade the relevance of environmental toxicology to modern society was realized with the Bhopal disaster, in which 45 tons of poisonous methyl isocyanate gas escaped from an insecticide plant, and the Chernobyl accident, in which massive amounts of radioactive material were released into the atmosphere following the explosion of a nuclear reactor. The Bhopal disaster killed between 15,000 and 20,000 people, with many thousands more suffering from acute and chronic conditions. The Chernobyl accident ultimately was responsible for an estimated several thousand deaths from radiation sickness and cancer and the contamination of millions of acres

of land across Belarus, Russia, and Ukraine. In the late 20th and early 21st centuries, the field of environmental toxicology expanded. Among its major concerns were oil spills, the dumping of medical and nuclear waste, air and water pollution, and the impact of substances such as synthetic hormones that were regularly released into environmental reservoirs.

2.1.2. Acute Toxicity and Chronic Toxicity

There are many sources of environmental toxicity that can lead to the presence of toxicants in our food, water and air. These sources include organic and inorganic pollutants, pesticides and biological agents, all of which can have harmful effects on living organisms. There can be so called point sources of pollution, for instance the drains from a specific factory but also non-point sources (diffuse sources) like the rubber from car tires that contain numerous chemicals and heavy metals that are spread in the environment. Environmental chemicals are largely taken up by organisms by passive diffusion. Primary sites of uptake include membranes of the lungs, gills, and gastrointestinal tract.

Acute Toxicity: Acute toxicity is defined as toxicity elicited as a result of short-term exposure to a toxicant. Incidences of acute toxicity in the environment are commonly associated with accident or imprudent use of the chemical. Acute toxicity occurs over a shorter period of time to higher concentrations. Acute toxicity describes the adverse effects of a substance that result either from a single exposure or from multiple exposures in a short period of time (usually less than 24 hours). To be described as acute toxicity, the adverse effects should occur within 14 days of the administration of the substance. **Acute health effects** are quickly seen, usually after exposures to fairly high levels or concentrations of hazardous substances.

Chronic Toxicity: Chronic toxicity is defined as toxicity elicited as a result of long-term exposure to a toxicant. Sublethal end points are generally associated with chronic toxicity. These include reproductive, immune, endocrine, and developmental dysfunction. Chronic toxicity describes the adverse health effects from repeated exposures, often at lower levels, to a substance over a longer time period (months or years). **Chronic effects** usually develop slowly. For example, if you breathe small amounts of asbestos fibers, you won't even notice them. There are no acute effects. But if

you inhale asbestos month after month, year after year, you greatly increase your chances of getting asbestos disease, such as lung cancer. This is a chronic effect. Other examples of chronic health effects include hearing loss and cumulative trauma disorders; these are examples of physical health hazards.

2.1.3. The Median Lethal Dose, LD₅₀

In toxicology, the **lethal dose (LD)** is an indication of the lethal toxicity of a given substance or type of radiation. Because resistance varies from one individual to another, the "lethal dose" represents a dose (usually recorded as dose per kilogram of subject body weight) at which a given percentage of subjects will die.

The median lethal dose, LD₅₀ (abbreviation for "lethal dose, 50%"), LC₅₀ (lethal concentration, 50%) or LCt₅₀ (lethal concentration and time) of a toxin, radiation, or pathogen is the dose required to kill half the members of a tested population after a specified test duration. LD₅₀ figures are frequently used as a general indicator of a substance's acute toxicity.

Toxicologists can use many kinds of animals but most often testing is done with rats and mice. It is usually expressed as the amount of chemical administered (e.g., milligrams) per 100 grams (for smaller animals) or per kilogram (for bigger test subjects) of the body weight of the test animal. The LD₅₀ can be found for any route of entry or administration but dermal (applied to the skin) and oral (given by mouth) administration methods are the most common.

2.2. Environmental Health

Toxic substances include both toxicants and toxins. A toxicant is any chemical, of natural or synthetic origin, which is poisonous and can cause harm to a living organism, whereas a toxin is a type of toxicant that is produced by a living organism. Environmental toxins and toxicants exposures can affect human health, fertility, reproduction and development. Evidence of the negative impact of environmental toxicants on human health is on the rise. A growing body of research suggests that maternal exposure to environmental toxicants poses a risk to women's health as well as fetal and child health and development

The prenatal and infancy periods are especially vulnerable to these potential effects during critical stages of development of the fetus and infant. In addition, certain behaviours exhibited by older infants (e.g., crawling on the floor, playing outside) put them at greater risk of exposure. Widespread awareness of environmental toxicants and their effects on reproductive and perinatal outcomes is essential in order to decrease preconception and prenatal exposure.

As environmental toxicants are identified, the associated risks of exposure should be communicated to women so they can reduce their exposure or eliminate it completely. Reproductive health providers play a significant role in communicating environmental health risks, as they are a key, trusted source of health information for women during the preconception and prenatal periods. Women of child-bearing age and their families are encouraged to ask their primary healthcare providers for information and guidance in order to address and manage environmental exposures effectively.

Humans interact with the environment constantly. These interactions affect quality of life, years of healthy life lived, and health disparities. The World Health Organization (WHO) defines environment, as it relates to health, as “all the physical, chemical, and biological factors external to a person, and all the related behaviors.” Environmental health consists of preventing or controlling disease, injury, and disability related to the interactions between people and their environment.

2.2.1. Environmental health objectives

The **Healthy People 2020 Environmental Health objectives** focus on 6 themes, each of which highlights an element of environmental health:

1. Outdoor air quality
2. Surface and ground water quality
3. Toxic substances and hazardous wastes
4. Homes and communities
5. Infrastructure and surveillance
6. Global environmental health

The 6 themes of the Environmental Health topic area draw attention to elements of the environment and their linkages to health.

2.2.1.1. Outdoor air quality. Poor air quality is linked to premature death, cancer, and long-term damage to respiratory and cardiovascular systems. Progress has been made to reduce unhealthy air emissions, but, in 2008, approximately 127 million people lived in U.S. counties that exceeded national air quality standards. Decreasing air pollution is an important step in creating a healthy environment.

2.2.1.2. Surface and ground Water. Surface and ground water quality applies to both drinking water and recreational waters. Contamination by infectious agents or chemicals can cause mild to severe illness. Protecting water sources and minimizing exposure to contaminated water sources are important parts of environmental health.

2.2.1.3. Toxic substances and hazardous wastes. The health effects of toxic substances and hazardous wastes are not yet fully understood. Research to better understand how these exposures may impact health is ongoing. Meanwhile, efforts to reduce exposures continue. Reducing exposure to toxic substances and hazardous wastes is fundamental to environmental health.

2.2.1.4. Homes and communities. People spend most of their time at home, work, or school. Some of these environments may expose people to:

- Indoor air pollution
- Inadequate heating and sanitation
- Structural problems
- Electrical and fire hazards
- Lead-based paint hazards

2.2.1.5. Infrastructure and surveillance. Prevention of exposure to environmental hazards relies on many partners, including State and local health departments. Personnel, surveillance systems, and education are important resources for investigating and responding to disease, monitoring for hazards, and educating the public. Additional methods and greater capacity to measure and respond to environmental hazards are needed.

2.2.1.6. Global Environmental Health. Water quality is an important global challenge. Diseases can be reduced by improving water quality and sanitation and increasing access to adequate water and sanitation facilities.

Creating health-promoting environments is complex and relies on continuing research to understand more fully the effects of exposure to environmental hazards on people's health.

2.2.2. Why is Environmental Health important?

Maintaining a healthy environment is central to increasing quality of life and years of healthy life. Globally, nearly 25 percent of all deaths and the total disease burden can be attributed to environmental factors. Environmental factors are diverse and far reaching. They include:

- Exposure to hazardous substances in the air, water, soil, and food
- Natural and technological disasters
- Physical hazards
- Nutritional deficiencies
- The built environment

Poor environmental quality has its greatest impact on people whose health status is already at risk. Therefore, environmental health must address the societal and environmental factors that increase the likelihood of exposure and disease.

2.2.3. Emerging issues in Environmental Health

Environmental health is a dynamic and evolving field. While not all complex environmental issues can be predicted, some known emerging issues in the field include:

Climate change. Climate change is projected to impact sea level, patterns of infectious disease, air quality, and the severity of natural disasters such as floods, droughts, and storms.

Disaster preparedness. Preparedness for the environmental impact of natural disasters as well as disasters of human origin includes planning for human health needs and the impact on public infrastructure, such as water and roadways.

Nanotechnology. The potential impact of nanotechnology is significant and offers possible improvements to:

- Disease prevention, detection, and treatment
- Electronics
- Clean energy
- Manufacturing
- Environmental risk assessment

However, nanotechnology may also present unintended health risks or changes to the environment.

The built environment. Features of the built environment appear to impact human health-influencing behaviors, physical activity patterns, social networks, and access to resources.

Exposure to unknown hazards. Finally, every year, hundreds of new chemicals are introduced to the world market. It is presumed that some of these chemicals may present new, unexpected challenges to human health, and, therefore, their safety should be evaluated prior to release. These cross-cutting issues are not yet understood well enough to inform the development of systems for measuring and tracking their impact. Further exploration is warranted. The environmental health landscape will continue to evolve and may present opportunities for additional research, analysis, and monitoring.

Blood Lead Levels. Eliminating elevated blood lead levels in children remains a goal of utmost importance to public health. Efforts must and will continue to reduce blood lead levels and to monitor the prevalence of children with elevated blood lead levels.

2.2.4. Environmental Health hazards

Environmental health hazards may be biological, chemical, physical, biomechanical or psychosocial in nature. Environmental health hazards include traditional hazards of poor sanitation and shelter, as well as agricultural and industrial contamination of air, water, food and land. These hazards have resulted in a host of health impacts, ranging from catastrophic direct effects (e.g., the recent cholera epidemic in Latin America and the chemical poisoning outbreak in Bhopal, India), to chronic effects (e.g., in Minamata, Japan), to subtle, indirect, and even disputed effects (e.g., in Love Canal, USA).

Table 2.1 summarizes some of the major notorious disasters in the last half century that have caused “environmental disease” outbreaks. There are undeniably countless other examples of environmental disease outbreaks, some of which are not easily detectable on the macrostatistical level. Meanwhile, over a billion people in the world lack access to safe drinking water (WHO 1992b) and over 600 million are exposed to ambient levels of sulphur dioxide that well exceed recommended levels. Moreover the pressure on agriculture and food production as both population and per capita demand increase, will likely lead to a greater burden on the environment. Environmental health impacts thus include the indirect effects of industrial disruption of adequate food and housing, as well as the degradation of the global systems on which the health of the planet depends.

In many countries large-scale agriculture and the concomitant active use of toxic pesticides is a major health hazard both for workers and for their households. Pollution by fertilizers or biological waste from the food industry, paper industry and so on can also have harmful effects on waterways, reducing fishing and food supplies. The fishermen and gatherers of other seafood may have to travel much further to get their daily catch, with increased risks of drowning accidents and other mishaps. The spread of tropical disease by the environmental changes associated with developments such as the building of dams, roads and so on constitutes another type of environmental health risk. The new dam may create breeding grounds for schistosomiasis, a debilitating disease affecting rice farmers who have to walk in water. The new road may create fast communication between an area with endemic malaria and another area hitherto spared from this disease.

It should be pointed out that the major basis for a harmful environment in the workplace or in the general environment is poverty. The traditional health threats in developing countries or in poor sections of any country include poor sanitation, water and food which spread communicable diseases, poor housing with high exposures to cooking smoke and high fire

Table 2.1. Selected major environmental disease outbreaks (WHO, 1992b)

Location and year	Environmental hazard	Type of disease	affected
London, UK 1952	Severe air-pollution with sulphur dioxide and suspended particulate matter	Increase in heart and lung disease manifestations	3,000 deaths, many others ill
South-east Turkey 1955-61	Hexachlorobenzene in seed grains	Porphyria; neurological disease	3,000
Minamata, Japan 1956	Methylmercury in fish	Neurological disease ("Minimata disease")	200 with severe disease, 2,000 suspected
USA cities 1960s-70s	Lead in paint	Anaemia, behavioural and mental effects	Many thousands
Fukuoka, Japan 1968	Polychlorinated biphenyls (PCBs) in food oil	Skin disease, general weakness	Several thousands
Iraq 1972	Methylmercury in seed grains	Neurological disease	500 deaths, 6,500 hospitalized
Madrid, Spain 1981	Aniline or other toxin in food oil	Various symptoms	340 deaths, 20,000 cases
Bhopal, India 1985	Methylisocyanate	Acute lung disease	2,000 deaths, 200,000 poisoned
California, USA 1985	Carbamate pesticide in watermelons	Gastrointestinal, skeletal, muscle, autonomic and central nervous system effects (Carbamate illness)	1,376 reported cases of illness resulting from consumption, 17 severely ill
Chernobyl, USSR 1986	Iodine-134, Caesium-134 and -137 from a reactor explosion	Radiation illness (including increases in cancer and thyroid diseases in children)	300 injured, 28 died within 3 months, more than 600 cases of thyroid cancer
Peru 1991	Cholera epidemic	Cholera	139 deaths, many thousand ill

risks, as well as high injury risks in small-scale agriculture or cottage industries. Reduction of poverty and improved living and working conditions is a fundamental priority for improved occupational and environmental health for billions of people. Despite efforts for energy conservation and sustainable development, failure to address the underlying inequities in wealth distribution threatens the global ecosystem.

Forests, for example, which represent the culmination of ecological successional processes, are being destroyed at an alarming rate, due to commercial logging and clearance by impoverished peoples for agriculture and firewood. The effects of forest depletion include soil erosion, which, if extreme, can lead to desertification. Loss of biodiversity is an important consequence. It is estimated that one-third of all carbon dioxide emissions are from the burning of tropical forests. Thus, addressing poverty is imperative with respect to global environmental health as well as individual, community and regional well-being.

The WHO has estimated that environmental exposures contribute to 19% of cancer incidence worldwide. Additionally, a WHO Global Health Risks report looked at five environmental exposures, (unsafe water, sanitation and hygiene, urban outdoor air pollution, indoor smoke from solid fuels, lead exposure and climate change), and estimated they account for nearly 10% of deaths and disease burden globally and around one quarter of deaths and disease burden in children under the age of five. The connection between pollution, notably toxic substance pollution, and human health has long been made in the developed world. Incidents such as Love Canal, a hazardous waste site in New York causing illness in the 1970s, brought industry pollutants and their effect on human health to prominence in public health studies. However, these connections between toxic pollution and human health have largely not been made as clearly in the developing world.

The lack of investigation and quantification of the human health impacts of contaminated sites have left an often-marginalized population with few resources to address this growing problem. Sadly, health impacts from environmental pollution often affect the most vulnerable, especially children, within these already neglected populations.

3. Environmental Toxins and Exposure to Humans

3.1. Introduction

Environmental toxins are chemicals both human-made and naturally occurring that can harm our health by disrupting sensitive biological systems. A toxicant is man-made (synthetic) substance that presents a risk of death, disease, injury, or birth defects in living organisms through absorption, ingestion, inhalation, or by altering the organism's environment. In comparison, a toxin is produced in nature by living animal or plant. We are surrounded by synthetic chemicals and encounter them countless times on a daily basis. Plastics, household cleaners, solvents, detergents, cosmetics and perfumes are all toxicants. So are antibiotics, prescription drugs, steroids, food additives, preservatives and other things we ingest. Pesticides, herbicides and fertilizers are also toxicants.

To live in the 21st Century means to live in a toxic world, where we are exposed daily to numerous environmental toxins and pollutants. Environmental toxins are on the increase and pose a problem in the form of very serious health risks, as thousands of toxic substances find their way into our air, water and the soil in which we grow our food. We spend our days inhaling pollutants such as car fumes and cigarette smoke. We drink water that has been thoroughly treated with chemicals, and eat food that is grown in toxic soil, pumped with hormones and packaged with preservatives. While our livers, kidneys, skin and lymphatic systems work round the clock to eliminate these dangerous toxins from our body, they very often just cannot keep up, and the result is a build-up of poisons in the system which destroy body tissue, damage organs, depress the immune system and leave the door open to a number of serious illnesses.

We are surrounded by environmental toxins. Substances that may cause distress or disease to our bodies can be found in everything that we eat, in everything that we drink and even in the air we breathe. Some of these compounds are a by-product of an industrialized world. Heavy metals like lead and cadmium are released from factories or are produced as waste substances in the industry. We are also exposed to many naturally-occurring toxic substances. For example, volcanic eruptions release much of the free mercury that can be found in the environment. Our bodies have a variety of

mechanisms for dealing with this toxicity, but the current total load exceeds the body's ability to adapt. When our bodies fail to break down or remove these toxins, the only other way to deal with them is through sequestration.

The body will try to deposit these compounds into tissue to minimize their potential damage. For example, lead may be sequestered into bone, displacing calcium and increasing the risk of osteoporosis. The overall load of these toxins is sometimes called our "Body Burden." A high body burden has been implicated in: Immunotoxicity - leading to asthma, allergies, cancers and chronic disease; Neurotoxicity - leading to cognition impairment, memory loss as well as sensory and motor dysfunction; and Endocrine toxicity - leading to reproductive issues, loss of libido and metabolic impairment.

In research published in 2005, New York University School of Medicine researchers provided some of the most compelling evidence yet that long-term exposure to air pollution - even at levels within federal standards - causes heart disease. Previous studies have linked air pollution to cardiovascular disease but until now it was poorly understood how pollution damaged the body's blood vessels.

3.2. Environmental Toxins in our Environment

Many of these toxins are things that you can't see, smell or feel, at least not right away. One of the major problems with them is just that. We don't realize that we're being affected until we come down with a chronic disease after years of subtle and often consistent exposure to a combination of these toxins. This makes it almost impossible to pinpoint a specific environmental toxin as the source of illness, yet when you look at the facts -- the increasing numbers of cancers, immune system disorders, neurological problems, chronic fatigue syndrome, multiple chemical sensitivities, allergies and hormonal disturbances that are facing the nation.

Environmental toxicity is a global concern. These pollutants don't recognize national or political boundaries. As an example, Japan has experienced a phenomenon known as "yellow sands" over the past several years. This is caused by pollution blowing in from Chinese factories across the Sea of Japan.

3.2.1. In the Air

There are now 1460 metric tons of airborne toxicants that travel on the jet stream around the world. Because of this there is no place on the planet that can be considered a pristine environment. Facilities in the United States released 4.7 billion pounds of toxins into the air in 2005- 72 million pounds are known carcinogens. In 2005 the city of Chicago experienced 68 days when the air quality was too unhealthy for children, elderly and the ill. Coal-fired power plants spew sulfates, nitrates and mercury into the air. These compounds have been linked to more than 20,000 premature deaths each year.

3.2.2. In the Water

There are an estimated 7 million illnesses and 1,000 deaths each year in the United States from waterborne microbes. Chlorinated chemicals in drinking water from pesticides, herbicides and refrigerants have been linked to increased risk of breast cancer. Cyanobacterial toxins in municipal water have been linked to illness and disease worldwide. Sewage treatment plant workers are at much higher risk of respiratory illness, skin rashes, headaches and body aches.

3.2.3. In our Food

Environmental toxins work their way into the food chain. As of late 2005, 47 states have advisories to limit intake of freshwater fish due to mercury contamination. In 2005, the FDA reported finding chlorinated pesticides, like DDE (dichlorodiphenyldichloroethylene - a breakdown product of DDT), in 63% of foods surveyed. Pesticides and herbicides in food have been linked to many cancers.

The most surprising thing about our body burden is that we are at risk even before we are born. A study conducted in 2005 by the Environmental Working Group in cooperation with the American Red Cross examined the umbilical cord blood of newborns. They found that the average newborn has 200 different industrial chemicals, pollutants and pesticides in their blood. These included over 70 known carcinogens (toxins that may cause cancer). Other studies have found high levels of the metals cadmium and mercury in the breast milk of nursing mothers.

As we grow up we are exposed to seemingly benign compounds that are even meant to benefit us but have been shown to have long-term negative consequences. For example, Fluoride in our drinking water has been linked to Osteosarcoma and Hypothyroidism. Additionally, vaccinations which undoubtedly prevent disease may contain the mercury compound Thimerosal which has been linked to the rise of autism in children.

3.3. Four Routes of Environmental Toxins' Exposure to Humans

Chemicals, including pesticides, are widely distributed in the environment. Therefore there are many possible sources of exposure to these chemicals for humans. Substances which are in ambient and indoor air may be inhaled into the lungs while those in water or food may be ingested or inhaled through mist or steam (such as in the shower). Direct contact with the chemical is the most prevalent way environmental chemicals can penetrate the skin, but exposure through the skin may also occur as a result of contact with chemical contaminants in air and water (for example bathing or swimming).

A single chemical can enter the body through all three routes of exposure -- inhalation, ingestion and skin penetration (dermal exposure). A compound, such as chloroform, which evaporates readily and which may be found in drinking water illustrates this point. When this water is used for drinking, ingestion is the route of exposure. When it is used for showering, exposure may occur due to inhalation of the steam or mist and from direct contact through the skin. Similarly, pesticide use can involve more than one route of exposure if precautions are not taken. A pesticide which is sprayed can be inhaled during use; penetrate through the skin during mixing and application; and be ingested through food if not washed off hands or food before eating.

There are four routes by which a substance can enter the body: inhalation, skin (or eye) absorption, ingestion, and injection.

3.3.1. Inhalation

For most chemicals in the form of vapors, gases, mists, fumes or particulates, inhalation is the major route of entry. Once inhaled, chemicals are either exhaled or deposited in the respiratory tract. If deposited, damage

can occur through direct contact with tissue or the chemical may diffuse into the blood through the lung-blood interface.

Upon contact with tissue in the upper respiratory tract or lungs, chemicals may cause health effects ranging from simple irritation to severe tissue destruction. Substances absorbed into the blood are circulated and distributed to organs that have an affinity for that particular chemical. Health effects can then occur in the organs, which are sensitive to the toxicant.

3.3.2. Skin (or eye) absorption

Skin (dermal) contact can cause effects that are relatively innocuous such as redness or mild dermatitis; more severe effects include destruction of skin tissue or other debilitating conditions. Many chemicals can also cross the skin barrier and be absorbed into the blood system. Once absorbed, they may produce systemic damage to internal organs. The eyes are particularly sensitive to chemicals. Even a short exposure can cause severe effects to the eyes or the substance can be absorbed through the eyes and be transported to other parts of the body causing harmful effects.

3.3.3. Ingestion

Chemicals that inadvertently get into the mouth and are swallowed do not generally harm the gastrointestinal tract itself unless they are irritating or corrosive. Chemicals that are insoluble in the fluids of the gastrointestinal tract (stomach, small, and large intestines) are generally excreted. Others that are soluble are absorbed through the lining of the gastrointestinal tract. They are then transported by the blood to internal organs where they can cause damage.

3.3.4. Injection

Substances may enter the body if the skin is penetrated or punctured by contaminated objects. Effects can then occur as the substance is circulated in the blood and deposited in the target organs. Once the chemical is absorbed into the body, three other processes are possible: metabolism, storage, and excretion. Many chemicals are metabolized or transformed via chemical reactions in the body. In some cases, chemicals are distributed and stored in specific organs. Storage may reduce metabolism and therefore, increase the persistence of the chemicals in the body. The various excretory mechanisms

(exhaled breath, perspiration, urine, feces, or detoxification) rid the body, over a period of time, of the chemical. For some chemicals elimination may be a matter of days or months; for others, the elimination rate is so low that they may persist in the body for a lifetime and cause deleterious effects.

3.4. Common Environmental Toxins

Over the last 50 years, from 70,000 to 100,000 different chemicals have been introduced into the world's markets, with about 1,500 new ones added each year. Over 3,000 chemicals are added to our food supply. More than 10,000 chemical solvents, emulsifiers and preservatives are used in food processing.

The following environmental toxins are among the most prevalent in our air, water and/or food supply. This list is by no means all-inclusive, as thousands of other toxins are also circulating in our environment.

3.4.1. Aluminum

Aluminum is the most widely distributed metal on the planet and it's used in the production of many every-day products. Cookware is made from aluminum, soda cans are aluminum, and aluminum foil is found in most kitchens. Aluminum is also in antacids, aspirin, vaccines, and even flour. This overwhelming infestation of aluminum means that your risk of exposure is through the roof, which is also made of aluminum. Unlike vitamins, minerals, and trace elements, the body does not need aluminum. And aluminum is no innocent or benign participant. Aluminum accumulates in the kidneys, brain, lungs, liver and thyroid where it competes with calcium for absorption and can affect skeletal mineralization. In infants, this can slow growth. The position of the Department of Neurology and Psychiatry at Saint Louis University is that aluminum may cause liver toxicity and lead to degenerative symptoms, including Alzheimer's.

3.4.2. Air particulate matter

Some particulates occur naturally, originating from volcanoes, dust storms, forest and grassland fires, living vegetation, and sea spray. Human activities, such as the burning of fossil fuels in vehicles, power plants and various industrial processes, also generate significant amounts of particulates.

Coal combustion in developing countries is the primary method for heating homes and supplying energy.

The size of the particle is a main determinant of where in the respiratory tract the particle will come to rest when inhaled. Larger particles are generally filtered in the nose and throat via cilia and mucus, but particulate matter smaller than about 10 micrometers, can settle in the bronchi and lungs and cause health problems.

Particulate matter studies in Bangkok Thailand from 2008 indicated a 1.9% increased risk of dying from cardiovascular disease, and 1.0% risk of all disease for every 10 micrograms per cubic meter. The Mongolian government agency recorded a 45% increase in the rate of respiratory illness in the past five years (reported in September 2014). Bronchial asthma, chronic obstructive pulmonary disease and interstitial pneumonia were the most common ailments treated by area hospitals. Levels of premature death, chronic bronchitis, and cardiovascular disease are increasing at a rapid rate.

3.4.3. Arsenic

Arsenic is a natural component of the earth's crust and is widely distributed throughout the environment in the air, water and land. It is highly toxic in its inorganic form. People are exposed to elevated levels of inorganic arsenic through drinking contaminated water, using contaminated water in food preparation and irrigation of food crops, industrial processes, eating contaminated food and smoking tobacco. Long-term exposure to inorganic arsenic, mainly through drinking of contaminated water, eating of food prepared with this water and eating food irrigated with arsenic-rich water, can lead to chronic arsenic poisoning. Skin lesions and skin cancer are the most characteristic effects. Long-term exposure to arsenic in drinking water can cause cancer in the skin, lungs, bladder and kidney. It can also cause other skin changes such as thickening and pigmentation.

3.4.4. Asbestos

Asbestos refers to a group of silicate fibers that are naturally occurring in the earth. These fibers are used for their strength and flexibility, they can be bonded together to create products like insulation, roofing, shingles, tiles, paper products packaging, and car parts. People may be exposed to asbestos in their workplace, their communities, or their homes. If products containing

asbestos are disturbed, tiny asbestos fibers are released into the air. When asbestos fibers are breathed in, they may get trapped in the lungs and remain there. Over time, these fibers can accumulate and cause scarring and inflammation, which can affect breathing and lead to serious health problems.

Asbestos affects the whole respiratory system. There are three serious health impacts, asbestosis, lung cancer and mesothelioma. Asbestosis is a serious, non-cancer form of lung disease. There is no treatment or cure for it and it causes shortness of breath. Lung cancer is the leading cause of death from asbestos exposure. Mesothelioma, another type of cancer, affects the lining of the lungs, abdomen and heart; largely all cases of mesothelioma can be directly linked back to asbestos exposure.

3.4.5. Benzene

Benzene is a colourless liquid that evaporates quickly. It is naturally found in crude oil and is a basic petrochemical. Unfortunately, it is also a known human carcinogen. Benzene is found in tobacco smoke, gasoline (and therefore car exhaust), pesticides, synthetic fibres, plastics, inks, oils, and detergents. Benzene has also been found in dryer emissions from scented laundry detergent and dryer sheets, and in soft drinks, although these have since been reformulated to exclude it.

About 50% of the benzene exposure in the US results from smoking tobacco or from second-hand smoke. Substantial amounts of data link benzene to aplastic anemia, bone marrow abnormalities, and leukemia — particularly acute myeloid leukemia (AML) and acute non-lymphocytic leukemia (ANLL).

To reduce benzene exposure:

- Don't smoke and try to avoid second hand smoke.
- Ensure adequate ventilation in your home.
- Use non-scented laundry detergents.
- Keep plants in the home.

3.4.6. BPA

Bisphenol A is commonly found in plastic bottles, plastic food containers, dental materials, and the linings of metal food and infant formula cans. Another exposure comes from receipt paper commonly used at grocery stores and restaurants, because today the paper is commonly coated with a BPA containing clay for printing purposes.

BPA is a known endocrine disruptor, and numerous studies have found that laboratory animals exposed to low levels of it have elevated rates of diabetes, mammary and prostate cancers, decreased sperm count, reproductive problems, early puberty, obesity, and neurological problems

3.4.7. Cadmium

Buildup of cadmium levels in the water, air, and soil has been occurring particularly in industrial areas. Environmental exposure to cadmium has been particularly problematic in Japan where many people have consumed rice that was grown in cadmium-contaminated irrigation water. This phenomenon is known under the name itai-itai disease.

Food is another source of cadmium. Plants may only contain small or moderate amounts in non-industrial areas, but high levels may be found in the liver and kidneys of adult animals. The daily intake of cadmium through food varies by geographic region. Intake is reported to be approximately 8 to 30 μ g in Europe and the United States versus 59 to 113 μ g in various areas of Japan.

Cigarettes are also a significant source of cadmium exposure. Although there is generally less cadmium in tobacco than in food, the lungs absorb cadmium more efficiently than the stomach.

Cadmium (Cd) is an extremely toxic industrial and environmental pollutant classified as a human carcinogen [Group 1 – according to International Agency for Research on Cancer;¹ Group 2a – according to Environmental Protection Agency (EPA); and 1B carcinogen classified by European Chemical Agency

Acute exposure to cadmium fumes may cause flu-like symptoms including chills, fever, and muscle ache sometimes referred to as "the cadmium blues." Symptoms may resolve after a week if there is no respiratory damage. More severe exposures can cause tracheo-bronchitis, pneumonitis, and pulmonary edema. Symptoms of inflammation may start hours after the

exposure and include cough, dryness and irritation of the nose and throat, headache, dizziness, weakness, fever, chills, and chest pain.

Inhaling cadmium-laden dust quickly leads to respiratory tract and kidney problems which can be fatal (often from renal failure). Ingestion of any significant amount of cadmium causes immediate poisoning and damage to the liver and the kidneys. Compounds containing cadmium are also carcinogenic.^[14]

The bones become soft (osteomalacia), lose bone mineral density (osteoporosis) and become weaker. This causes the pain in the joints and the back, and also increases the risk of fractures. In extreme cases of cadmium poisoning, mere body weight causes a fracture

3.4.8. Carbon Monoxide

Carbon monoxide, or CO, is a toxic gas that you cannot see or smell. CO is given off whenever fuel or other carbon-based materials are burned. CO usually comes from sources in or near your home that are not properly maintained or vented.

You may be exposed to unsafe levels of CO by:

- using poorly maintained or unvented heating equipment;
- improperly vented natural gas appliances like stoves or water heaters;
- running vehicles in garages or other enclosed spaces;
- using a gas stove, grill, or oven to heat the home;
- house or building fires;
- clogged chimneys or blocked heating exhaust vents;
- running generators or gas-powered tools indoors or outside near windows, doors, or vents;
- cooking with a charcoal or gas grill inside the home or other enclosure;
- using a propane camp stove, heater, or light inside a tent; and
- being near boat engine exhaust outlets.

Breathing CO can cause headache, dizziness, vomiting, and nausea. If CO levels are high enough, you may become unconscious or die. Exposure to moderate and high levels of CO over long periods of time has also been linked

with increased risk of heart disease. People who survive severe CO poisoning may suffer long-term health problems.

3.4.9. Chlorine

Chlorine is an important chemical in water purification, in disinfectants, in bleach and in mustard gas. Sources are household cleaners, drinking water (in small amounts), air when living near an industry (such as a paper plant) that uses chlorine in industrial processes. The health effects resulting from most chlorine exposures begin within seconds to minutes. The severity of the signs and symptoms caused by chlorine will vary according to amount, route and duration of exposure.

Inhalation: Most chlorine exposures occur via inhalation. Low level exposures to chlorine in air will cause eye/skin/airway irritation, sore throat and cough. Chlorine's odor provides adequate early warning of its presence, but also causes olfactory fatigue or adaptation, reducing awareness of one's prolonged exposure at low concentrations. At higher levels of exposure, signs and symptoms may progress to chest tightness, wheezing, dyspnea, and bronchospasm. Severe exposures may result in noncardiogenic pulmonary edema, which may be delayed for several hours.

Ingestion: Since chlorine is a gas at room temperature, it is unlikely that a severe exposure will result from ingestion. However, ingestion of chlorine dissolved in water (e.g., sodium hypochlorite or household bleach) will cause corrosive tissue damage of the gastrointestinal tract.

Eye/Dermal Contact: Low level exposures to chlorine gas will cause eye and skin irritation. Higher exposures may result in severe chemical burns or ulcerations. Exposure to compressed liquid chlorine may cause frostbite of the skin and eyes.

Risks: Sore throat, coughing, eye and skin irritation, rapid breathing, narrowing of the bronchi, wheezing, blue coloring of the skin, accumulation of fluid in the lungs, pain in the lung region, severe eye and skin burns, lung collapse, reactive airways dysfunction syndrome (RADS) (a type of asthma)

3.4. 10. Chloroform

Chloroform, colorless liquid has a pleasant, nonirritating odor and a slightly sweet taste, and is used to make other chemicals. Major Sources of chloroform are air, drinking water and food can contain chloroform. When it's inhaled, it can cause heartbeat irregularities that may be deadly. It can also cause liver and kidney damage. High concentrations of the chemical may produce headaches, dizziness and gastrointestinal problems such as nausea and vomiting. In addition, chloroform has been classified as a probable carcinogen – a chemical that can cause cancer. Newer anesthetics have replaced chloroform in the operating room.

3.4.11. Chromium

As chromium compounds are used in dyes and paints and the tanning of leather, these compounds are often found in soil and groundwater at abandoned industrial sites. Primar paint containing hexavalent chromium is still widely used for aerospace and automobile refinishing applications. Water-insoluble chromium compounds and chromium metal are not considered a health hazard, while the toxicity and carcinogenic properties of chromium have been known for a long time.

Chromium is recognized as a human carcinogen when it is inhaled. Chronic inhalation of chromium has been shown to increase risk of lung cancer and may also damage the small capillaries in kidneys and intestines.

Other adverse health effects associated with chromium-6 exposure, according to the National Institute for Occupational Safety and Health (NIOSH), include skin irritation or ulceration, allergic contact dermatitis, occupational asthma, nasal irritation and ulceration, perforated nasal septa, rhinitis, nosebleed, respiratory irritation, nasal cancer, sinus cancer, eye irritation and damage, perforated eardrums, kidney damage, liver damage, pulmonary congestion and edema, epigastric pain, and erosion and discoloration of one's teeth.

3.4.12. Diesel Exhaust

Diesel exhaust is the gaseous exhaust produced by a diesel type of internal combustion engine, plus any contained particulates. Its composition may vary with the fuel type or rate of consumption, or speed of engine operation (e.g., idling or at speed or under load), and whether the engine is in an on-road vehicle, farm vehicle, locomotive, marine vessel, or stationary generator or other application. Diesel exhaust is a Group 1 carcinogen, which causes lung cancer and has a positive association with bladder cancer. It contains several substances that are also listed individually as human carcinogens by the IARC.

Emissions from diesel vehicles have been reported to be significantly more harmful than those from petrol vehicles. Diesel combustion exhaust is a source of atmospheric soot and fine particles, which is a component of the air pollution implicated in human cancer, heart and lung damage, and mental functioning. Moreover, diesel exhaust contains contaminants listed as carcinogenic for humans by the IARC (part of the World Health Organization of the United Nations), as present in their List of IARC Group 1 carcinogens. Diesel exhaust pollution is thought to account for around one quarter of the pollution in the air in previous decades, and a high share of sickness caused by automotive pollution.

3.4.13. Dioxins

Dioxins are a group of chemically-related compounds that are persistent environmental pollutants (POPs). Dioxins are found throughout the world in the environment and they accumulate in the food chain, mainly in the fatty tissue of animals. More than 90% of human exposure is through food, mainly meat and dairy products, fish and shellfish. Many national authorities have programmes in place to monitor the food supply. Dioxins are highly toxic and can cause reproductive and developmental problems, damage the immune system, interfere with hormones and also cause cancer. Due to the omnipresence of dioxins, all people have background exposure, which is not expected to affect human health. However, due to the highly toxic potential, efforts need to be undertaken to reduce current background exposure. Prevention or reduction of human exposure is best done via source-directed measures, i.e. strict control of industrial processes to reduce formation of dioxins.

3.4.14. Excitotoxins (Food Additives)

Excitotoxins are substances, largely amino acids like glutamate and aspartate, that stimulate taste receptors on the tongue. While they enhance the flavor of foods, they perform no other function. Excitotoxins (there are more than 70 known today) are found in most packaged and processed foods, particularly soups, sauces, gravy mixes, frozen dinners, diet foods and beverages, chips, as well as fast foods. Excitotoxins over-excite brain cells, which can lead to their death.

3.4.15. Formaldehyde

Formaldehyde is found in:

- Resins used in the manufacture of composite wood products (i.e., hardwood plywood, particleboard and medium-density fiberboard)
- Building materials and insulation
- Household products such as glues, permanent press fabrics, paints and coatings, lacquers and finishes, and paper products
- Preservatives used in some medicines, cosmetics and other consumer products such as dishwashing liquids and fabric softeners
- Fertilizers and pesticides

It is a byproduct of combustion and certain other natural processes, and so is also found in:

- Emissions from un-vented, fuel burning appliances, like gas stoves or kerosene space heaters.
- Cigarette smoke.

The primary way you can be exposed to formaldehyde is by breathing air containing off-gassed formaldehyde. Everyone is exposed to small amounts of formaldehyde in the air that has off-gassed from products, including composite wood products. Formaldehyde can cause irritation of the skin, eyes, nose, and throat. High levels of exposure may cause some types of cancers.

To reduce formaldehyde exposure:

- Use “exterior-grade” pressed-wood products to limit formaldehyde exposure in the home.
- Ensure adequate ventilation and moderate temperatures.
- Reduce humidity levels with air conditioners and dehumidifiers.
- Go natural and grow plants in your home.

3.4.16. Genetically Modified Foods (GMO’s)

Genetically modified organisms (GMO’s) are a broad group of plants, animals, and bacteria that are engineered for a wide variety of applications ranging from agricultural production to scientific research. Because GMO’s that could directly effect human health are primarily products that can enter the human food supply. To date, the only types of products that have been approved for human consumption in the U.S. are genetically modified plants (FDA website).

Food allergy affects approximately 5% of children and 2% of adults in the U.S. and is a significant public health threat (Bakshi, 2003).). Allergic reactions in humans occur when a normally harmless protein enters the body and stimulates an immune response (Bernstein et al., 2003). If the novel protein in a GM food comes from a source that is known to cause allergies in humans or a source that has never been consumed as human food, the concern that the protein could elicit an immune response in humans increases. Although no allergic reactions to GM food by consumers have been confirmed, in vitro evidence suggesting that some GM products could cause an allergic reaction has motivated biotechnology companies to discontinue their development (Bakshi, 2003).

3.4.17. Lead

Lead is a naturally occurring toxic metal found in the earth’s crust. Its widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world. Important sources of environmental contamination include mining, smelting, manufacturing and recycling activities, and, in some countries, the continued use of leaded paint, leaded gasoline, and leaded aviation fuel. More than three

quarters of global lead consumption is for the manufacture of lead-acid batteries for motor vehicles. Lead is, however, also used in many other products, for example pigments, paints, solder, stained glass, lead crystal glassware, ammunition, ceramic glazes, jewellery, toys and in some cosmetics and traditional medicines. Drinking water delivered through lead pipes or pipes joined with lead solder may contain lead. Much of the lead in global commerce is now obtained from recycling.

People can become exposed to lead through occupational and environmental sources. This mainly results from:

- inhalation of lead particles generated by burning materials containing lead, for example, during smelting, recycling, stripping leaded paint, and using leaded gasoline or leaded aviation fuel; and
- ingestion of lead-contaminated dust, water (from leaded pipes), and food (from lead-glazed or lead-soldered containers).

The use of some traditional cosmetics and medicines can also result in lead exposure.

When humans inhale or ingest lead it is distributed to the brain, liver, kidney and bones and can be stored in the blood, teeth or bones. Because lead is an element, it cannot be broken down or destroyed; it accumulates in the body as long as a person continues to be exposed to it. Lead accumulation leads to neurological, gastrointestinal, and cardiovascular problems. Lead exposure during pregnancy can lead to miscarriage, stillbirth, low birth weights, premature births and birth defects. The International Agency for Research on Cancer declares it to be a possible human carcinogen. Children are exceptionally vulnerable because their bodies absorb 4-5 times as much lead as adults; even at the lowest levels of exposure lead is toxic to children. The brain damage resulting from lead exposure in children is untreatable and includes mild mental retardation, decreased IQ, shortened attention spans, loss of executive function, increased risk of dyslexia, and diminished productivity. It is estimated that the effects of mild mental retardation and cardiovascular problems alone, caused by lead exposure, amount to almost 1% of the total global burden of disease, with developing countries carrying the largest burden.

3.4.18. Mercury

Mercury occurs naturally in the earth's crust. It is released into the environment from volcanic activity, weathering of rocks and as a result of human activity. Human activity is the main cause of mercury releases, particularly coal-fired power stations, residential coal burning for heating and cooking, industrial processes, waste incinerators and as a result of mining for mercury, gold and other metals.

Once in the environment, mercury can be transformed by bacteria into methylmercury. Methylmercury then bioaccumulates (bioaccumulation occurs when an organism contains higher concentrations of the substance than do the surroundings) in fish and shellfish. Methylmercury also biomagnifies. For example, large predatory fish are more likely to have high levels of mercury as a result of eating many smaller fish that have acquired mercury through ingestion of plankton.

People may be exposed to mercury in any of its forms under different circumstances. However, exposure mainly occurs through consumption of fish and shellfish contaminated with methylmercury and through worker inhalation of elemental mercury vapours during industrial processes. Cooking does not eliminate mercury.

Health effects of mercury exposure: Elemental and methylmercury are toxic to the central and peripheral nervous systems. The inhalation of mercury vapour can produce harmful effects on the nervous, digestive and immune systems, lungs and kidneys, and may be fatal. The inorganic salts of mercury are corrosive to the skin, eyes and gastrointestinal tract, and may induce kidney toxicity if ingested.

Neurological and behavioural disorders may be observed after inhalation, ingestion or dermal exposure of different mercury compounds. Symptoms include tremors, insomnia, memory loss, neuromuscular effects, headaches and cognitive and motor dysfunction. Mild, subclinical signs of central nervous system toxicity can be seen in workers exposed to an elemental mercury level in the air of 20 µg/m³ or more for several years. Kidney effects have been reported, ranging from increased protein in the urine to kidney failure.

3.4.19. Nitrogen Oxides

Nitrogen dioxide is an intermediate in the industrial synthesis of nitric acid, millions of tons of which are produced each year. At higher temperatures it is a reddish-brown gas that has a characteristic sharp, biting odor and is a prominent air pollutant

For the public, chronic exposure to NO₂ can cause respiratory effects including airway inflammation in healthy people and increased respiratory symptoms in people with asthma. NO₂ creates ozone which causes eye irritation and exacerbates respiratory conditions, leading to increased visits to emergency departments and hospital admissions for respiratory issues, especially asthma.

A major source of indoor exposure to (NO₂) is from the use of gas stoves for cooking or heating in homes. According to the 2000 census, over half of US households use gas stoves and indoor exposure levels of (NO₂) are, on average, at least three times higher in homes with gas stoves compared to electric stoves with the highest levels being in multifamily homes. Exposure to (NO₂) is especially harmful for children with asthma. Research has shown that children with asthma who live in homes with gas stoves have greater risk of respiratory symptoms such as wheezing, cough and chest tightness. Additionally, gas stove use was associated with reduced lung function in girls with asthma, although this association was not found in boys. Using ventilation when operating gas stoves may reduce the risk of respiratory symptoms in children with asthma.

3.4.20. Ozone

Ozone is formed from dioxygen by the action of ultraviolet light and also atmospheric electrical discharges, and is present in low concentrations throughout the earth's atmosphere (stratosphere). In total, ozone makes up only 0.6 ppm of the atmosphere. Ozone is a powerful oxidant (far more so than dioxygen) and has many industrial and consumer applications related to oxidation. This same high oxidising potential, however, causes ozone to damage mucous and respiratory tissues in animals, and also tissues in plants, above concentrations of about 100 ppb. This makes ozone a potent respiratory hazard and pollutant near ground level. However, the ozone layer (a portion of the stratosphere with a higher concentration of ozone, from two to eight ppm)

is beneficial, preventing damaging ultraviolet light from reaching the earth's surface, to the benefit of both plants and animals.

WHO states: "There are few epidemiological studies on the chronic effects of ozone on human health. Incidence of asthma, a decreased lung function growth, lung cancer and total mortality are the main outcomes studied. At levels currently observed in Europe, the evidence linking O₃ exposure to asthma incidence and prevalence in children and adults is not consistent. Available evidence suggests that long-term O₃ exposure reduces lung function growth in children. There is little evidence for an independent long-term O₃ effect on lung cancer or total mortality.

3.4.21. PCBs (Polychlorinated biphenyls)

The polychlorinated biphenyls (PCBs) are synthetic organochlorine chemicals that were useful industrial products in the past, but their production was ended because they persist in both the environment and living organisms. The PCBs are mixtures of up to 209 different components (congeners), depending on the number and position of chlorines around the biphenyl ring. The PCBs are fat-soluble substances to which everyone is exposed through ingesting animal fats, inhalation, or dermal contact. Exposure to PCBs suppresses the immune system, thereby increasing the risk of acquiring several human diseases.

Exposure to PCBs is associated with an increased risk of certain cancers of the digestive tract, liver and skin. PCB exposure is also associated with reproductive deficiencies, such as reduced growth rates, retarded development, and certain neurological effects which may or may not persist beyond infancy. The immune system can also be affected, leading to increased infection rates, and changes in the skin such as chloracne and pigmentation disturbances.

Human exposures to relatively high levels of PCBs have occurred primarily in persons working in plants that extensively manufactured and used PCBs and PCB-containing equipment. Occupational exposure to PCBs can result in a broad spectrum of effects that includes:

- Increased levels of some liver enzymes, with possible hepatic damage,
- Chloracne and related dermal lesions, and respiratory problems

3.4.22. Pesticides

Pesticides are designed to kill and because their mode of action is not specific to one species, they often kill or harm organisms other than pests, including humans. The World Health Organization estimates that there are 3 million cases of pesticide poisoning each year and up to 220,000 deaths, primarily in developing countries. According to the Environmental Protection Agency (EPA), 60 percent of herbicides, 90 percent of fungicides and 30 percent of insecticides are known to be carcinogenic. Alarming, pesticide residues have been detected in 50 percent to 95 percent of U.S. foods.

Pesticides are any substance used to kill, repel, or control certain forms of plant or animal life that are deemed pests. This includes herbicides, insecticides, fungicides, disinfectants, and compounds used to control rodents. In the US, over 4.5 billion pounds of pesticides are used each year. Most conventional food production uses pesticides, so people are exposed to low levels of pesticide residues through their diets. While the health effects of pesticide residues are not entirely clear, research from the National Institute of Health showed that farmers who use agricultural insecticides experience an increase in headaches, fatigue, insomnia, dizziness, hand tremors, and other neurological symptoms, while licensed pesticide applicators have a 20-200% increased risk of developing diabetes.

Other data found that individuals reporting regular exposure to pesticides had a 70% higher incidence of Parkinson's disease than those reporting no exposure. It also appears that children are particularly susceptible to adverse effects from exposure to pesticides, specifically neuro-developmental problems. This is probably because children eat more food relative to their size. They also play in the dirt and spend time on the ground, where pesticides may linger.

To reduce exposure to pesticides, wash and scrub all fruits and vegetables, organic or conventional. If possible purchase mostly organic fruits and vegetables, particularly the ones consistently found to have the highest pesticide residues – apples, strawberries, celery, peaches and spinach.

3.4.23. Phthalates

Phthalates are chemicals used to soften plastics. They are found in a wide variety of products, including bottles, shampoo, cosmetics, lotions, nail polish, and deodorant. The National Institute of Environmental Health Sciences (NIEHS), part of the National Institute of Health, has found that pre-natal exposure to phthalates is associated with adverse genital development and can significantly reduce masculine behavior in boys. Women with high exposure to phthalates while pregnant report significantly more disruptive behavior in their children, while other research by NIEHS has found phthalate exposure can lead to thyroid dysfunction in adults.

To reduce phthalate exposure, minimize use of plastics. Use PVC-free containers. Buy plastic wrap and bags made from polyethylene and use glass containers. If you do use plastic containers, do not heat or microwave them. Choose phthalate-free toys. Many large toymakers have pledged to stop using phthalates, but be sure to look for toys made from polypropylene or polyethylene.

Purchase phthalate-free beauty products. Avoid nail polish, perfumes, colognes, and other scented products that list phthalates as an ingredient. Many scented products simply list “fragrance” as an ingredient, which often incorporates a number of different chemicals including phthalates. Try to minimize these products, or for more information on phthalate-free cosmetics and personal care products.

3.4.24. Radon

Radon is a colorless, odorless radioactive gas. It comes from the natural decay of uranium or thorium found in nearly all soils and it typically moves up through the ground and into the home through cracks in floors, walls, and foundations. It can also be released from building materials or from well water. Radon breaks down quickly, giving off radioactive particles. Long-term exposure to these particles can lead to lung cancer. The U.S. Environmental Protection Agency estimates that radon causes about 21,000 lung cancer deaths in the United States each year, with 1 in 20 US homes having elevated levels. Radon exposure is the second leading cause of lung cancer after smoking and the leading cause among non-smokers. Many radon-related lung cancer deaths can be prevented by testing for radon and taking the necessary

steps to lower radon levels in homes that have elevated radon. This process is known as radon mitigation.

To reduce radon exposure, get your home air checked. It is simple and inexpensive. If you use a well, check your water also.

3.4.25. Secondhand Smoke

Secondhand smoke is a serious health hazard causing more than 41,000 deaths per year. It can cause or make worse a wide range of damaging health effects in children and adults, including lung cancer, respiratory infections and asthma. Secondhand smoke causes approximately 7,330 deaths from lung cancer and 33,950 deaths from heart disease each year. Between 1964 and 2014, 2.5 million people died from exposure to secondhand smoke, according to a report from the U.S. Surgeon General. The report also concluded that secondhand smoke is a definitive cause of stroke.

There is no risk-free level of exposure to secondhand smoke and even short-term exposure potentially can increase the risk of heart attacks. Secondhand smoke contains hundreds of chemicals known to be toxic or carcinogenic, including formaldehyde, benzene, vinyl chloride, arsenic ammonia and hydrogen cyanide. Secondhand smoke can cause heart attacks; even relatively brief exposure can trigger a heart attack, according to a report by the Institute of Medicine.

3.4.26. Sulfur dioxide

Sulfur dioxide (SO₂) is a colorless reactive gaseous air pollutant with a pungent odor. This gas can pose a threat to human health, animal health, and plant life. The primary sources of sulfur dioxide emissions are from fossil fuel combustion and natural volcanic activity. Hawai'i Volcanoes National Park (NP) is unique in the national park system because it periodically has extremely high concentrations of sulfur dioxide — far higher than any other national park or even most urban areas. It is released naturally by volcanic activity. Inhaling sulfur dioxide is associated with increased respiratory symptoms and disease, difficulty in breathing, and premature death.

Sulfur dioxide irritates the skin and mucous membranes of the eyes, nose, throat, and lungs. High concentrations of SO₂ can cause inflammation and irritation of the respiratory system, particularly during heavy physical

activity. The resulting symptoms may include pain when taking a deep breath, coughing, throat irritation, and breathing difficulties. High concentrations of SO₂ can affect lung function, worsen asthma attacks, and aggravate existing heart disease in sensitive groups. This gas can also react with other chemicals in the air and convert to a small particle that can lodge in the lungs and cause similar health effects.

3.4.27. Volatile Organic Compounds (VOC's)

Volatile Organic Compounds (VOCs) are a large group of chemicals that are found in many products we use to build and maintain our homes such as paint, varnishes, caulks, adhesives, carpet, vinyl flooring, composite wood products, upholstery and foam, air fresheners, cleaning products, Cosmetics, Fuel oil and gasoline. Some of the health effect of VOCs on humans is eye, nose, and throat irritation; headaches, loss of coordination, nausea; damage to liver, kidney, and central nervous system. Some VOCs from testing are known to cause cancer in animals and are suspected or known to cause cancer in humans.

Key signs or symptoms associated with exposure to VOCs include conjunctival irritation, nose and throat discomfort, headache, allergic skin reaction, dyspnea, declines in serum cholinesterase levels, nausea, emesis, epistaxis, fatigue, dizziness.

3.4.28. Microwaves and Electromagnetic Fields (cell phones, electric blankets, computers, TV screens)

A microwave oven usually called as a microwave, is an electrical appliance to heat the cold food items through dielectric heating that is caused by electromagnetic waves produced by microwave. It is used commonly to reheat the previously cooked foods and also used to cook vegetables that are cooked fast and easy. It uses less energy than a stove to reheat the food. Microwaves are used in almost every home these days and there is a need to take some necessary precautionary steps to avoid any mishap occurring due to the use of microwaves. These precautions are to be taken related to the potential exposure to microwaves, food handling and thermal burns.

Microwaves are a form of "electromagnetic" radiation; that is, they are waves of electrical and magnetic energy moving together through space. Microwave cooking can be more energy efficient than conventional cooking because foods cook faster and the energy heats only the food, not the whole oven compartment. Microwave cooking does not reduce the nutritional value of foods any more than conventional cooking. In fact, foods cooked in a microwave oven may keep more of their vitamins and minerals, because microwave ovens can cook more quickly and without adding water

There are certain dangers associated to the usage of microwaved food. The reheated food from microwave can be a cause of very dangerous health hazards like hormonal disruption, brain damage, malnutrition, increased body fat and weakened immune system. Some of the very dangerous side effects associated to the use of microwaved food are as under:

- There are by-products produced in the microwaved food that cannot be broken down that is metabolized in the human body.
- A continuous use of microwaved food can be a cause of brain damage because it shortens the electrical impulse produced from the brain.
- The hormonal production in both males and females is altered or stopped because of the usage of microwaved food.

The by-products of microwaved food have their long term permanent effects on the human body that weakens it slowly but continuously. The minerals present in the vegetables are changed into cancerous radicals when they are cooked in the microwave oven.

- The microwaved food causes the production of cancerous tumors in the human stomach and intestines.
- The rapid increase of colon cancer in people of developed countries in the present era is due to the use of microwaved food which affects the human intestines dangerously enough to cause cancer.
- The human blood develops the cancer causing cells in it if the microwaved food is eaten for a considerable longer period of time.
- The lack of concentration, decrease in intelligence, emotional instability and loss of memory are caused by eating microwaved food.
- The microwaved cereal and milk converts amino acid into carcinogens.
- The meat when heated in a microwave loses its nucleo-proteins.

- The body resistance against the viral and bacterial infections gets lowered because of microwaved food.
- The excessive use of microwaved food can cause serious birth defects in both males and females.
- Microwaves if leaked from the microwave oven can cause serious health issues especially those linked to the eyes because they are the most sensitive of the visible body organs.
- The cholesterol level in the human body is increased due to the intake of microwaved food.
- The white blood cells in the blood are decreased slowly due to the use of reheated food.
- Anemic tendencies are increased in the human body due to the gradual decrease of hemoglobin in the blood that is mainly caused due to the excessive intake of microwaved food.

Health Problems Linked to Electromagnetic Fields (EMF)

Poisoning: The following list represents diseases and health problems that are either a direct result of electromagnetic radiation poisoning or are closely associated with it. Keep in mind that all health issues can be affected to some degree by exposure to EMF radiation, whether electric, magnetic, wireless or ionizing radiation, since it weakens the immune system, stresses the body and damages healthy cells.

- cancer
- alzheimer's
- parkinson's
- fibromyalgia
- chronic fatigue
- daily headaches
- brain cancer
- asthma
- heart problems
- chest pain
- insomnia
- allergies
- multiple sclerosis
- high blood pressure
- brain fog
- forgetfulness

- digestive disorders
- migraine headaches
- brain tumors
- leukemia - general
- lymphoblastic leukemia
- birth defects
- stress
- nausea
- rheumatoid arthritis
- fatigue

In 2011, the WHO IARC spent a year reviewing experiments and health studies and concluded that cell and cordless phone radiation was a “possible human carcinogen”. Those who began using cell phones before age 20 find a 4 to 8 fold increase in brain cancer as well as increases in leukemia. For many young women today, tucking cell phones in the bra has become a cool, hip way to have easy access to these electronic devices. Most women have no idea that cellphones are two-way microwave radios that should not be kept directly on the body and increases their risk of developing breast cancer and other diseases.

3.4.29. Ultraviolet Radiation

Exposure to ultraviolet radiation (UVR) occurs from both natural and artificial sources. The main natural source is the sun. On the other hand, artificial UVR sources are widely used in industry and also used in hospitals, laboratories, etc. because of their germicidal properties. They are even used for cosmetic purposes. The harmful effects of UVR consist of erythema, sunburn, photodamage (photoaging), photocarcinogenesis, damage to the eyes, alteration of the immune system of the skin, and chemical hypersensitivity. Skin cancer is commonly produced by UVR.

3.4.30. Heavy Metals

Metals like arsenic, mercury, lead, aluminum and cadmium, which are prevalent in many areas of our environment, can accumulate in soft tissues of the body.

Risks: Cancer, neurological disorders, Alzheimer's disease, foggy head, fatigue, nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels

Major Sources: Drinking water, fish, vaccines, pesticides, preserved wood, antiperspirant, building materials, dental amalgams, chlorine plants

3.5. Health Effects from Exposure to Toxic Substances

Human health effects caused by exposure to toxic substances fall into two categories: short-term and long-term effects.

Short-term effects (or acute effects) have a relatively quick onset (usually minutes to days) after brief exposures to relatively high concentrations of material (acute exposures). The effect may be local or systemic. Local effects occur at the site of contact between the toxicant and the body. This site is usually the skin or eyes, but includes the lungs if irritants are inhaled or the gastrointestinal tract if corrosives are ingested. Systemic effects are those that occur if the toxicant has been absorbed into the body from its initial contact point, transported to other parts of the body, and cause adverse effects in susceptible organs. Many chemicals can cause both local and systemic effects.

Long-term effects (or chronic effects) are those with a long period of time (years) between exposure and injury. These effects may occur after apparent recovery from acute exposure or as a result of repeated exposures to low concentrations of materials over a period of years (chronic exposure). Health effects manifested from acute or chronic exposure are dependent upon the chemical involved and the organ it effects. Most chemicals do not exhibit the same degree of toxicity for all organs. Usually the major effects of a chemical will be expressed in one or two organs. These organs are known as target organs which are more sensitive to that particular chemical than other organs. The organs of the body and examples of effects due to chemical exposures are listed below.

3.5.1. Respiratory Tract

The respiratory tract is the only organ system with vital functional elements in constant, direct contact with the environment. The lung also has the largest exposed surface area of any organ on a surface area of 70 to 100 square meters versus 2 square meters for the skin and 10 square meters for the digestive system. Many chemicals used or produced in industry can produce acute or chronic diseases of the respiratory tract when they are inhaled (Table 3.1). The toxicants can be classified according to how they affect the respiratory tract.

Asphyxiants: gases that deprive the body tissues of oxygen. **Simple asphyxiants** are physiologically inert gases that at high concentrations displace air leading to suffocation. Examples: nitrogen, helium, methane, neon, argon. **Chemical asphyxiants** are gases that prevent the tissues from getting enough oxygen. Examples: carbon monoxide and cyanide. Carbon monoxide binds to hemoglobin 200 times more readily than oxygen. Cyanide prevents the transfer of oxygen from blood to tissues by inhibiting the necessary transfer enzymes.

Irritants: chemicals that irritate the air passages. Constriction of the airways occurs and may lead to edema (liquid in the lungs) and infection. Examples: hydrogen fluoride, chlorine, hydrogen chloride, and ammonia.

Necrosis producers: Chemicals that result in cell death and edema. Examples: ozone and nitrogen dioxide.

Fibrosis producers: Chemicals that produce fibrotic tissue which, if massive, blocks airways and decreases lung capacity. Examples: silicates, asbestos, and beryllium.

Allergens: Chemicals that induce an allergic response characterized by bronchoconstriction and pulmonary disease. Examples: isocyanates and sulfur dioxide.

Carcinogens: Chemicals that are associated with lung cancer. Examples: cigarette smoke, coke oven emissions, asbestos, and arsenic. Not only can various chemicals affect the respiratory tract, but the tract is also a route for chemicals to reach other organs. Solvents, such as benzene and

tetrachloroethane, anesthetic gases, and many other chemical compounds can be absorbed through the respiratory tract and cause systemic effects.

Table 3.1. Examples of industrial toxicants that produce disease of the respiratory tract.
(UNL Environmental Health and Safety, 2002)

Toxicant	Site of Action	Acute Effect	Chronic Effect
Ammonia	Upper Airways	Irritation, edema	Bronchitis
Arsenic	Upper Airways	Bronchitis, irritation, pharyngitis	Cancer, bronchitis, laryngitis
Asbestos	Lung parenchyma	_____	Fibrosis, cancer
Chlorine	Upper Airways	Cough, irritation, asphyxiant (by muscle cramps in the larynx)	_____
Isocyanates	Lower airways, alveoli	Bronchitis, pulmonary edema, asthma	_____
Nickel Carbonyl	Alveoli	Edema (delayed symptoms)	_____
Ozone	Bronchi, alveoli	Irritation, edema, hemorrhage	Emphysema, bronchitis
Phosgene	Alveoli	Edema	Bronchitis, fibrosis, pneumonia
Toluene	Upper Airways	Bronchitis, edema, bronchospasm	_____
Xylene	Lower airways	Edema, hemorrhage	_____

Not only can various chemicals affect the respiratory tract, but the tract is also a route for chemicals to reach other organs. Solvents, such as benzene and tetrachloroethane, anesthetic gases, and many other chemical compounds can be absorbed through the respiratory tract and cause systemic effects.

3.5.2. Skin.

The skin is, in terms of weight, the largest single organ of the body. It provides a barrier between the environment and other organs (except the lungs and eyes) and is a defense against many chemicals. The skin consists of the epidermis (outer layer) and the dermis (inner layer). In the dermis are sweat glands and ducts, sebaceous glands, connective tissue, fat, hair follicles, and blood vessels. Hair follicles and sweat glands penetrate both the epidermis and dermis. Chemicals can penetrate through the sweat glands, sebaceous glands, or hair follicles. Although the follicles and glands may permit a small amount of chemicals to enter almost immediately, most pass through the epidermis, which constitutes the major surface area. The top layer is the stratum corneum, a thin cohesive membrane of dead surface skin. This layer turns over every 2 weeks by a complex process of cell dehydration and polymerization of intracellular material. The epidermis plays the critical role in skin permeability.

Below the epidermis lies the dermis, a collection of cells providing a porous, watery, nonselective diffusion medium. Intact skin has a number of functions:

- Epidermis: Prevents absorption of chemicals and is a physical barrier to bacteria.
- Sebaceous glands: Secrete fatty acids which are bacteriostatic and fungistatic.
- Melanocytes (skin pigment): Prevent damage from ultraviolet radiation in sunlight.
- Sweat glands: Regulate heat.
- Connective tissue: Provides elasticity against trauma.
- Lymph-blood system: Provide immunologic responses to infection.

The ability of skin to absorb foreign substances depends on the properties and health of the skin and the chemical properties of the substances. Absorption is enhanced by:

- Breaking top layer of skin by abrasions or cuts.
- Increasing hydration of skin.
- Increasing temperature of skin which causes sweat cells to open up and secrete sweat, which can dissolve solids.
- Increasing blood flow to skin.
- Increasing concentrations of the substance.
- Increasing contact time of the chemical on the skin.
- Increasing the surface area of affected skin.
- Altering the skin's normal pH of 5.
- Decreasing particle size of substance.
- Adding agents which will damage skin and render it more susceptible to penetration.
- Adding surface-active agents or organic chemicals. DMSO, for example, can act as a carrier of the substance.
- Inducing ion movement by an electrical charge. Absorption of a toxic chemical through the skin can lead to **local effects** through direct contact, such as irritation and necrosis, and **systemic effects**.

Many chemicals can cause a reaction with the skin resulting in inflammation called dermatitis. These chemicals are divided into three categories:

Primary irritants: Act directly on normal skin at the site of contact (if chemical is in sufficient quantity for a sufficient length of time). Skin irritants include: acetone, benzyl chloride, carbon disulfide, chloroform, chromic acid and other soluble chromium compounds, ethylene oxide, hydrogen chloride, iodine, methyl ethyl ketone, mercury, phenol, phosgene, styrene, sulfur dioxide, picric acid, toluene, xylene.

Photosensitizers: Increase in sensitivity to light, which results in irritation and redness. Photosensitizers include: tetracyclines, acridine, creosote, pyridine, furfural, and naphtha.

Allergic sensitizers: May produce allergic-type reaction after repeated exposures. They include: formaldehyde, phthalic anhydride, ammonia, mercury, nitrobenzene, toluene diisocyanate, chromic acid and chromates, cobalt, and benzoyl peroxide.

3.5.3. Eyes

The eyes are affected by the same chemicals that affect skin, but the eyes are much more sensitive. Many materials can damage the eyes by direct contact:

Acids: Damage to the eye by acids depends on pH and the protein-combining capacity of the acid. Unlike alkali burns, the acid burns that are apparent during the first few hours are a good indicator of the long-term damage to be expected. Some acids and their properties are:

- sulfuric acid. In addition to its acid properties, it simultaneously removes water and generates heat.
- picric acid and tannic acid. No difference in damage they produce in entire range of acidic pHs.
- hydrochloric acid. Severe damage at pH 1, but no effect at pH 3 or greater.

Alkalies: Damage that appears mild initially but can later lead to ulceration, perforation, and clouding of the cornea or lens. The pH and length of exposure have more bearing on the amount of damage than the type of alkali. Some problem alkalies are:

- sodium hydroxide (caustic soda) and potassium hydroxide.
- ammonia penetrates eye tissues more readily than any other alkali; calcium-oxide (lime) forms clumps when it contacts eye tissue and is very hard to remove.

- Organic solvents: Organic solvents (for example, ethanol, toluene, and acetone) dissolve fats, cause pain, and dull the cornea. Damage is usually slight unless the solvent is hot.
- Lacrimators: Lacrimators cause instant tearing at low concentrations. They are distinguished from other eye irritants (hydrogen chloride and ammonia) because they induce an instant reaction without damaging tissues. At very high concentrations lacrimators can cause chemical burns and destroy corneal material. Examples are chloroacetophenone (tear gas) and mace. In addition, some compounds act on eye tissue to form cataracts, damage the optic nerve, or damage the retina. These compounds usually reach the eye through the blood having been inhaled, ingested or absorbed rather than direct contact.

Examples of compounds that can provide systemic effects damaging to the eyes are:

- Naphthalene: Cataracts and retina damage.
- Phenothiazine (insecticide): Retina damage
- Thallium: cataracts and optic nerve damage.
- Methanol: Optic nerve damage.

3.5.4. Central Nervous System

Neurons (nerve cells) have a high metabolic rate but little capacity for anaerobic metabolism. Subsequently, inadequate oxygen flow (anoxia) to the brain kills cells within minutes. Some may die before oxygen or glucose transport stops completely. Because of their need for oxygen, nerve cells are readily affected by both simple asphyxiants and chemical asphyxiants. Also, their ability to receive adequate oxygen is affected by compounds that reduce respiration and thus reduce oxygen content of the blood (barbiturates, narcotics).

Other examples are compounds such as arsine, nickel, ethylene chlorohydrin, tetraethyl lead, aniline, and benzene that reduce blood pressure or flow due to cardiac arrest, extreme hypotension, hemorrhaging, or

thrombosis. Some compounds damage neurons or inhibit their function through specific action on parts of the cell. The major symptoms from such damage include: dullness, restlessness, muscle tremor, convulsions, loss of memory, epilepsy, idiocy, loss of muscle coordination, and abnormal sensations. Examples are:

- Fluoroacetate: Rodenticide.
- Triethyltin: Ingredient of insecticides and fungicides.
- Hexachlorophene: Antibacterial agent.
- Lead: Gasoline additive and paint ingredient.
- Thallium: Sulfate used as a pesticide and oxide or carbonate used in manufacture of optical glass and artificial gems.
- Tellurium: Pigment in glass and porcelain.
- Organomercury compounds: Methyl mercury used as a fungicide, is also a product of microbial action on mercury ions. Organomercury compounds are especially hazardous because of their volatility and their ability to permeate tissue barriers.

Some chemicals are noted for producing weakness of the lower extremities and abnormal sensations (along with previously mentioned symptoms):

- Acrylamide: Soil stabilizer, water proofer.
- Carbon disulfide: Solvent in rayon and rubber industries.
- n-Hexane: Used as a cleaning fluid and solvent. Its metabolic product, hexanedione, causes the effects.
- Organophosphorus compounds: Often used as flame retardants (triorthocresyl phosphate) and pesticides (Leptofor and Mipafox). Agents that prevent the nerves from producing proper muscle contraction and may result in death from respiratory paralysis are DDT, lead, botulinum toxin, and allethrin (a synthetic insecticide). DDT, mercury, manganese, and monosodium glutamate also produce personality disorders and madness.

3.5.5. Liver

Liver injury induced by chemicals has been known as a toxicologic problem for hundreds of years. It was recognized early that liver injury is not a simple entity, but that the type of lesion depends on the chemical and duration of exposure. Three types of response to hepatotoxins can be identified:

Acute. Cell death from:

- carbon tetrachloride: Solvent, degreaser.
- chloroform: Used in refrigerant manufacture solvent.
- trichloroethylene: Solvent, dry cleaning fluid, degreaser.
- tetrachloroethane: Paint and varnish remover, dry cleaning fluid.
- bromobenzene: Solvent, motor oil additive.
- tannic acid: Ink manufacture, beer and wine clarifier.
- kepone: Pesticide.

Chronic. Examples include:

- cirrhosis: a progressive fibrotic disease of the liver associated with liver dysfunction and jaundice. Among agents implicated in cirrhosis cases are carbon tetrachloride, alcohol, and aflatoxin.
- carcinomas: malignant, growing tissue. For example, vinyl chloride (used in chloride production) and arsenic (used in pesticides and paints) are associated with cancers.

Biotransformation of toxicants. The liver is the principal organ that chemically alters all compounds entering the body. For example:

ethanol---> acetaldehyde---> acetic acid---> water + carbon dioxide

This metabolic action by the liver can be affected by diet, hormone activity, and alcohol consumption. Biotransformation in the liver can also lead to toxic metabolites. For example: carbon tetrachloride---> chloroform

3.5.6. Kidneys

The kidney is susceptible to toxic agents for several reasons:

(1) The kidneys constitute 1 percent of the body's weight, but receive 20-25 percent of the blood flow (during rest). Thus, large amounts of circulating toxicants reach the kidneys quickly. (2) The kidneys have high oxygen and nutrient requirements because of their workload. They filter one-third of the plasma reaching them and reabsorb 98-99% of the salt and water. As they are reabsorbed, salt concentrates in the kidneys. (3) Changes in kidney pH may increase passive diffusion and thus cellular concentrations of toxicants. (4) Active secretion processes may concentrate toxicants. (5) Biotransformation is high.

A number of materials are toxic to the kidneys:

- Heavy metals, may denature proteins as well as produce cell toxicity. Heavy metals (including mercury, arsenic, gold, cadmium, lead, and silver) are readily concentrated in the kidneys, making this organ particularly sensitive.
- Halogenated organic compounds, which contain chlorine, fluorine, bromine, or iodine. Metabolism of these compounds, like that occurring in the liver, generates toxic metabolites. Among compounds toxic to the kidneys are carbon tetrachloride, chloroform, 2,4,5-T (a herbicide), and ethylene dibromide (a fumigant).
- Miscellaneous, including carbon disulfide (solvent for waxes and resins) and ethylene glycol (automobile antifreeze).

3.5.7. Blood

The blood system can be damaged by agents that affect blood cell production (bone marrow), the components of blood (platelets, red blood cells, and white blood cells), or the oxygen-carrying capacity of red blood cells.

3.5.7.1. Bone Marrow. Bone marrow is the source of most components in blood. Agents that suppress the function of bone marrow include:

- Arsenic, used in pesticides and paints.
- Bromine, used to manufacture gasoline antiknock compounds, ethylene dibromide, and organic dyes.
- Methyl chloride, used as a solvent, refrigerant, and aerosol propellant.
- Ionizing radiation, produced by radioactive materials and x-rays is associated with leukemia.
- Benzene, a chemical intermediate associated with leukemia.

3.5.7.2. Blood Components. Among platelets (thrombocytes) are blood components that help prevent blood loss by forming blood clots. Among chemicals that affect this action are:

- Aspirin, which inhibits clotting.
- Benzene, which decreases the number of platelets.
- Tetrachloroethane, which increases the number of platelets.

Leukocytes (white blood cells) are primarily responsible for defending the body against foreign organisms or materials by engulfing and destroying the material or by producing antibodies. Chemicals that increase the number of leukocytes include naphthalene, magnesium oxide, boron hydrides, and tetrachloroethane. Agents that decrease the number of leukocytes include benzene and phosphorous. Erythrocytes (red blood cells) transport oxygen in the blood. Chemicals that destroy (hemolyze) red blood cells include arsine (a gaseous arsenic compound and contaminant in acetylene), naphthalene (used to make dyes), and warfarin (a rodenticide).

3.5.7.3. Oxygen Transport. oxygen carrying capabilities of red blood cells. A notable example is carbon monoxide which combines with hemoglobin to form carboxyhemoglobin. Hemoglobin has an affinity for carbon monoxide 200 times greater than that for oxygen. While carbon monoxide combines reversibly with hemoglobin, some chemicals cause the hemoglobin to change such that it cannot combine reversibly with oxygen.

This condition is called methemoglobinemia. Some chemicals that can cause this are:

- Sodium nitrite, used in meat curing and photography.
- Aniline, used in manufacture of rubber accelerators and antioxidants, resins, and varnishes.
- Nitrobenzene and dinitrobenzene, used in manufacture of dyestuffs and explosives.
- Trinitrotoluene (TNT), used in explosives.
- Mercaptans, used in manufacture of pesticides and as odorizers for hazardous odorless gases.
- 2-nitropropane, used as a solvent.

3.5.8. Spleen

The spleen filters bacteria and particulate matter (especially deteriorated red blood cells) from the blood. Iron is recovered from the hemoglobin for recycling. In the embryo, the spleen forms all types of blood cells. In the adult, however, it produces only certain kinds of leukocytes. Examples of chemicals that damage the spleen are:

- Chloroprene, used in production of synthetic rubber.
- Nitrobenzene, used as chemical intermediate.

3.5.9. Reproductive System

Experimental results indicate that certain agents interfere with the reproductive capabilities of both sexes, causing sterility, infertility, abnormal sperm, low sperm count, and/or affect hormone activity in animals. Many of these also affect human reproduction. Some examples of chemicals that have been implicated in reproductive system toxicity include:

- Male: Anesthetic gases (halothane, methoxyflurane) cadmium, mercury, lead, boron, methyl mercury, vinyl chloride, DDT,

kepone, chlordane, PCBs, dioxin, 2,4-D, 2,4,5-T, carbaryl, paraquat, dibromochloropropane, ethylene dibromide, benzene, toluene, xylene, ethanol, radiation, and heat.

- Female: DDT, parathion, carbaryl, diethylstilbestrol (DES), PCBs, cadmium, methyl mercury, hexafluoroacetone, and anesthetic gases.

3.6. Toxic Chemicals in our Food System

Chemicals are used in every step of the process that puts food on our table: production, harvesting, processing, packing, transport, marketing and consumption and can be dangerous to our health. Some of these chemicals remain in our food and many persist in the environment and our bodies for decades to come.

Preservatives are added to many processed foods including breads, cereals, and meat. Studies have found that additives are a source of headaches, nausea, weakness and difficulty breathing. New research has shown that they may damage human nerve cells. We do not fully understand all of the long-term effects that additives could have on our health because synthetic additives are a relatively new invention.

Certain fish contain toxic chemicals called Perchlorinated biphenyls (PCBs-which have been banned but remain in our environment and end up in our food system) or heavy metals such as mercury. PCBs can damage the developing brain and have been linked to behavioral disorders. Heavy metals like mercury may lower IQ and also cause visual or hearing impairment.

Food packaged in plastic may contain phthalates or other harmful chemicals. The chemicals can seep from the packaging into the food itself. Research has linked phthalates to behavioral disorders.

3.6.1. What Chemicals are in our bodies

There is no adequate data to know how many chemicals each of us is exposed to every day, or which ones we will carry in our bodies for the rest of

our lives. Studies of chemical residues in the urine of the U.S. population have shown that most Americans have measurable amounts of pesticides in their bodies (Table 3.2).

**Table 3.2. Human Exposure to Environmental Chemicals
(The U.S. Centers for Disease Control and Prevention
Fourth National Report on Human Exposure to
Environmental Chemicals. 2009.)**

Toxic chemical	Sources of Exposure	Adverse Health Effects
Certain Pesticides & Fungicides	Food residues; contaminated soil; agricultural settings; water contamination	Damage to the developing brain; loss of IQ; respiratory disease; non-Hodgkins lymphoma, childhood leukemia; early breast cancer; asthma; autoimmune disease; thyroid disease
Preservatives: Propyl Gallate, BHA & BHT, Sodium Nitrite & Sodium Nitrate	Preservative-added food	Cancer
PCBs (banned substances)	Certain fish;	Damage to the developing brain; loss of IQ; behavioral disorders
BPA	Canned food; many plastic containers	Damage to the developing brain; behavioral disorders
Phthalates, adipates & organometals	Plastics; other forms of packaging	Behavioral disorders
Arsenic	Chicken, drinking water	Carcinogen; increased risk of cardiovascular disease and diabetes
Mercury	Fish; emissions from coal-powered electric plants	Damage to the developing brain; loss of IQ; behavioral disorders; lower overall function; visual & hearing impairment

A Washington University researcher tested urine samples from local children and found that some pesticides were five to seven times higher in children eating a conventional diet versus those eating an organic one.

Packaging also plays its role as it is likely that dietary ingestion is the reason 90% of people in the U.S. have measurable amounts of BPA in their urine. The U.S. Centers for Disease Control and Prevention studies document that childhood exposure to phthalates is widespread. The CDC found that children aged 6-11 years old excrete higher concentrations of phthalate metabolites than older age groups. Possibly due to higher food consumption related to body weight, mouthing behavior, and/or playing near the ground.

3.6.2. How can we avoid toxic chemicals in food

We cannot avoid toxicants in our food entirely, but we can do several things to reduce our current and future exposure, including:

Choose organic, sustainable and less-toxic options. You can lower your pesticide intake by avoiding the most contaminated fruits and vegetables: apples, strawberries, grapes, celery, peaches, spinach, sweet bell peppers, cucumbers, cherry tomatoes, and potatoes. When possible buy organic for this produce.

Choosing to buy food with less and safer packaging and few or no preservatives is also a good first step in reducing exposure.

Support institutions, such as schools and hospitals, in purchasing more sustainable food. With their large purchasing power, institutions can make a significant impact on the health of their community and the people they serve through the food they purchase. From kids and teachers in schools to patients, staff and visitors at hospitals, millions of people spend money and eat food in institutions every day. Encourage institutions to purchase more sustainable food and support them by ordering it when they do.

Demand national & local food, farm and chemicals policy changes. We need to make a national investment in implementing effective agriculture, environmental and food policy that supports sustainable production practices on farms.

3.7. Ten Dangerous Everyday Things in your Home

Objects that you may be using very day may contain hazardous chemicals, such as lead, mercury, arsenic, and PCBs, that are endangering you and your family. Avoiding these objects completely may be impossible. However, the following products, which are possibly laden with dangerous chemicals, should be avoided: such as farm-raised salmon, commercial animal fats, tap water, and certain mattresses and clothings.

Household consumer products injure 33.1 million people in the United States every year. These incidents cost \$800 billion in related expenses from death, injury or property damages. And many scientists are starting to believe that, in particular, the chemicals found in a wide variety of the goods you use every day may be more toxic than previously thought. Here are 10 of the most common products that may be hazardous to your health:

3.7.1. Mothballs

Since moths chew holes through clothing and other textiles, people pack away these stinky repellents to kill them. But studies on one active ingredient in some repellents, paradichlorobenzene, found that it can cause cancer in animals. Other types of moth balls use naphthalene, which after prolonged exposure can damage or destroy red blood cells, and which can also stimulate nausea, vomiting, and diarrhea.

3.7.2. Pesticides

Ninety percent of households in the United States use some form of pesticide, a broad term that encompasses a variety of chemical formulas that kill everything from tiny microorganisms up to rodents. In 2006, the American Association of Poison Control Centers received nearly 46,000 calls regarding children under 5 years old who had been exposed to potentially toxic levels of pesticides.

3.7.3. Pressed wood products

This faux wood takes bits and pieces of logs and wood leftovers and combines them together. Pressed wood products include paneling, particle board, fiberboard, and insulation, all of which were particularly popular for home construction in the 1970s. However, the glue that holds the wood

particles in place may use urea-formaldehyde as a resin. The U.S. EPA estimates that this is the largest source of formaldehyde emissions indoors. Formaldehyde exposure can set off watery eyes, burning eyes and throat, difficulty breathing, and asthma attacks. Scientists also know that it can cause cancer in animals. The risk is greater with older pressed wood products, since newer ones are better regulated.

3.7.4. Chemicals in carpets

Indoor carpeting has recently come under greater scrutiny because of the volatile organic compounds (VOCs) associated with new carpet installation. The glue and dyes used with carpeting are known to emit VOCs, which can be harmful to your health in high concentrations. However, the initial VOC emissions will often subside after the first few days following.

3.7.5. Laser printers chemicals

A 2007 study found that some laser printers give off ultra-fine particles that can cause serious health problems. Another study confirmed that laser and ink-jet printers can release volatile organic compounds (VOCs) and ozone particulates. All of these have been linked with heart and lung disease.

3.7.6. Lead paint

In 1991, the U.S. government declared lead to be the greatest environmental threat to children. Even low concentrations can cause problems with your central nervous system, brain, blood cells, and kidneys. It's particularly threatening for fetuses, babies, and children, because of potential developmental disorders. Many houses built before 1978 contain lead paint. Once the paint begins to peel away will, it release the harmful lead particles that you can inhale.

3.7.7. Air fresheners and cleaning solutions

Air fresheners and cleaning solutions, when used excessively or in a small, unventilated area, can release toxic levels of pollutants. This comes from two main chemicals called ethylene-based glycol ethers and terpenes. While the EPA regards the ethers as toxic by themselves, the non-toxic terpenes can react with ozone in the air to form a poisonous combination. Air

fresheners in particular are linked to many volatile organic compounds, such as nitrogen dioxide, and some fresheners also contain paradichlorobenzene, the same chemical emitted by mothballs.

3.7.8. Baby bottles and BPA

Canada has taken the first steps to outlaw the sale of baby bottles made from polycarbonate plastics, which are the most common type on the market. It has done so because the plastics are made with a chemical called bisphenol-A (BPA). BPA has a structure very similar to estrogen and for that reason is referred to as a "hormone disruptor." Hormone disruptors can interfere with the natural human hormones, especially for young children.

3.7.9. Flame retardants

Commonly used in mattresses, upholstery, television, and computer casings and circuit boards, flame retardants use poly brominated diphenyl ethers, or PBDEs for short. Two forms of PBDEs were phased out of use in manufacturing in the United States in 2004 because of related health threats, but the products containing them linger on. Studies have linked PBDEs to learning and memory problems, lowered sperm counts and poor thyroid functioning in rats and mice. Other animal studies have indicated that PBDEs could be carcinogenic in humans, although that has not yet been confirmed.

3.7.10. Cosmetic phthalates

Phthalates, also called plasticizers, go into many products including hair spray, shampoos, fragrances, and deodorants. Phthalates bind the color and fragrance in cosmetic products, and are also used to increase the durability and flexibility of plastics. Like BPA, these hormone-like chemicals are linked to reproductive and developmental problems in animals. Because of these findings, California and Washington State have banned the use of phthalates in toys for younger children.

3.8. More Toxic Sources You may not have Considered

Although cutting your exposure to toxic materials down to zero would be a near impossible task, you can significantly lower, and maintain your toxic load as low as possible, by being an informed and vigilant consumer.

Unfortunately, many of the toxins that you're exposed to are from your general environment, which you may have little or no control over. However, you can still make a positive impact on your health by avoiding some of the products that are infamously high in various toxins, and replacing them with healthier alternatives. Products to avoid include:

3.8.1. Farm-raised salmon – High in PCBs (polychlorinated biphenyls). This industrial chemical has been banned in the United States for decades, yet is a persistent organic pollutant that's still present in your environment. Its risks include cancer and impaired fetal brain development. Unfortunately, most farm-raised salmon, which accounts for most of the supply in the United States, are fed meals of ground-up fish that have absorbed PCBs in the environment, and for this reason should be avoided, especially if you are pregnant or nursing.

3.8.2. Commercial animal fats – High in dioxins. Dioxins are chemical compounds formed as a result of combustion processes, such as commercial or municipal waste incineration and from burning fuels (like wood, coal, or oil). These highly toxic compounds pollute our environment where they enter the food chain. Over 95 percent of your exposure to dioxins comes from eating commercial animal fats. Health risks include: cancer, reproductive and developmental disorders, skin rashes, skin discoloration, excessive body hair, and mild liver damage. Grass-fed, organic meats are your healthiest option all around.

3.8.3. Tap water – High in disinfection byproducts (DBPs), chloroform, and fluoride. **Chloroform** and other DBPs are formed when chlorine is added to water. This is the most common form of water disinfection in the U.S. Unfortunately, chloroform can cause cancer, potential reproductive damage, birth defects, dizziness, fatigue, headache, liver and kidney damage.

Another byproduct of water chlorination is trihalomethanes (THMs), which are Cancer Group B carcinogens, meaning they've been shown to cause cancer in laboratory animals. They've also been linked to reproductive problems in both animals and humans. **Fluoride** is another serious danger silently lurking in most people's tap water. Fluoride alters your endocrine function, especially in your thyroid, increases your risk of bone fractures, and can lower IQ. Filtering the water in your home (including the water in your shower) with a reverse osmosis filter is your best option here.

3.8.4. Mattresses – High in Polybrominated diphenyl ethers or PBDEs, antimony, and formaldehyde. PBDEs, which have been banned in Canada, Europe, and several states, build up in your body over time, and what you absorb or inhale does not go away. This is of great concern, since you spend as much as a third of your life in bed, on a potentially toxic mattress.

The health problems associated with PBDE exposure include brain and reproductive damage, decreased sperm quality, thyroid problems, and even cancer at high levels. **Boric acid**, another agent found in many mattresses, is a toxic respiratory irritant used to kill roaches. **Antimony**, a metal that may be more toxic than mercury, and formaldehyde, which causes cancer, are both used in mattresses for flame retardant purposes.

A person sleeping on a chemically treated mattress will absorb 0.8 mg of antimony every night; an amount that is 27 times more than the U.S. Environmental Protection Agency says is safe. Five-year-old children, meanwhile, will absorb 0.5 mg of antimony every night, according to the United States Consumer Product Safety Commission (CPSC), which is 63 times more than the EPA's safety limit.

Shopping for a safe mattress is not an easy task. Mattress manufacturers are not required to label or disclose which chemicals their mattresses contain. However, there are now a few manufacturers that make 100% wool, toxin-free mattresses.

3.8.5. Clothing – May contain **formaldehyde**. Clothing made of rayon, blended cotton, corduroy, wrinkle-resistant 100% cotton, and any synthetic blended polymer are likely to have been treated with formaldehyde resins, which is a "probable carcinogen." You can be exposed to the chemical

both via off-gassing and direct contact with your skin. Formaldehyde has been shown to cause cancer in animals, and may cause cancer in humans. Other common adverse health effects include fatigue, skin rashes, and allergic reactions.

3.9. Reducing Environmental Exposures for Children

Scientists continue to study the relationship between childhood illness or disability and environmental chemicals. In the meantime, applying good habits around the home or school makes good sense. The more you can reduce unnecessary exposures to commonly used chemicals or other hazardous substances, the healthier the environment becomes for you and your children. Providing a healthy environment for our children is a goal we all share. Children are at greater risk from chemicals found in food, water, dirt and air for several reasons. They're more at risk because, for their size, they eat, drink and breathe more than grown-ups. They're more at risk because they crawl on floors, play in dirt and put their hands in their mouths. They may be more at risk because their bodies are still developing.

3.9.1. Keep contaminants out of your surroundings

The exposure can come from...	What you must do
Secondhand smoke	Go outside if you have to smoke and encourage other smokers to do so too.
Contaminants tracked in from outside such as lawn pesticides, lead dust from exterior paint	Use a door mat, remove shoes at the door, plant shrubs and grass to keep dust levels down.
Chemical drift from nearby pesticide applications	Close windows during treatment and then open them about 30 minutes later for fresh air. Cover children's play toys and equipment prior to applications if possible, or hose down exposed items before re-use.
Gasoline and kerosene fumes from lawn mowers, snow blowers and other power equipment	Store equipment in outside shed or garage and use proper storage containers.
Release of mercury from broken thermometers	Clean up spills properly (never vacuum a spill); replace mercury thermometers with digital types and dispose of old ones properly.

3.9.2. Reduce contaminants in the house by ventilating with fresh air

The exposure can come from...	What you must do
Breathing chemicals from paints, wood finishing products and cleaning products	When possible, either use these products outdoors or open windows. Schedule painting and finishing projects during warm months when windows can be left open.
Breathing chemicals from new carpets or building materials	Ask the merchant to air out new carpet before installing or delivering it; keep the windows open if possible until the smell has disappeared.
Some materials used for adult hobbies, such as glues and paints with strong odors, lead for stained glass work, etc.	Keep children away from hobby areas and make sure there is plenty of ventilation when you are using the materials. Store chemicals in tight containers and keep them out of children's reach.

3.9.3. Use less of chemical product

The exposure can come from...	What you must do
Cleaning products	Have as few products as necessary, and use them sparingly. Consider less toxic or lower strength alternatives. Resist the use of anti-bacterial products, as they encourage the growth of stronger germs.
Pesticides	Many pests can be controlled without the use of chemical pesticides. Keep pests under control. Learn about Integrated Pest Management techniques that focus on preventative pest control and use chemical pesticides as a last resort.
Insect repellents	Use the lowest concentration of a repellent that is needed and then use only as much as you need for your situation. Do not let young children apply their own repellent. Spray repellent on your hands and then apply to children, avoiding the face.

3.9.4. Wash hands, toys, fruits, vegetables and surfaces around the home, as it also reduces potential chemical exposures.

The exposure can come from...	What we should do
Contaminants on and in food, such as bacteria, pesticides and other chemicals	Wash fruits and vegetables. Rinse meat, chicken and fish before you cook it. Also, skin and trim fish, since some contaminants concentrate in fatty tissues. For more information about sportfish contamination, see the Department of Health's Chemicals in Sportfish and Game brochure. Check the FDA website to review reports about pesticide residues in food.
Chemical dust and residues	Wash children's hands and toys with soap and water frequently. Damp mop floors. Wash window sills and the area between the sill and the screen or outside window, where dust collects. If clothes get contaminated, wash them separately from children's items.

3.9.5. Decide what may be an "unnecessary" exposure for you and your family

The exposure can come from...	What we should do
Lawn pesticides	Learn about ways to control pests and weeds that do not use chemical pesticides. Appreciate the look of a natural lawn.
Air fresheners, deodorizers and candles	Recognize that if you can smell it, the artificial scent or odor may be made up of chemicals. Nothing freshens a room like fresh air.
Personal care products	Ask yourself if the products you use are really necessary (hair spray? powders? perfumes?).

3.9.6. Keep children away from products that contain hazardous Substances

The exposure can come from...	What we should do
Household products such as glass cleaners, oven cleaners, drain openers, floor and furniture polishes, bleaches, dishwasher detergents, carpet cleaners, etc.	Put locks on cabinets and store products out of children's reach. Do not keep what you will not use again. Dispose of unwanted products properly.
Pesticides such as flea and tick controls, lawn pesticides and indoor pesticides	Read and follow label directions carefully. Keep children away from areas being treated during treatment and for a while after. Remove toys and stuffed animals from areas before treatment. Teach children not to touch flea and tick collars on pets or areas where other products, such as spray and spot treatments, were applied.

3.9.7. Home Safe Home

The exposure can come from...	What we should do
Breathing radon gas	Test your home for radon (using a simple and inexpensive detector); if there is a problem, it can often be fixed quite easily.
Carbon monoxide (CO) and other emissions from furnaces, appliances, space heaters, fireplaces and wood burning stoves	Have your furnace checked about once a year or immediately if you smell fuel or smoke. Clean chimneys. Consider the use of CO detectors.
Molds (which can trigger allergies or asthma)	Find the source of the leak or moisture and fix it. Dehumidifiers can help in damp settings; make sure you empty the reservoir and clean frequently.

3.10. Almost a Quarter of all Disease Caused by Environmental Exposure

As much as 24% of global disease is caused by environmental exposures which can be averted. Well-targeted interventions can prevent much of this environmental risk, the World Health Organization (WHO) demonstrates in a report issued today. The report further estimates that more than 33% of disease in children under the age of 5 is caused by environmental exposures. Preventing environmental risk could save as many as four million lives a year in children alone, mostly in developing countries.

The report, **Preventing disease through healthy environments - towards an estimate of the environmental burden of disease**, is the most comprehensive and systematic study yet undertaken on how preventable environmental hazards contribute to a wide range of diseases and injuries. By focusing on the environmental causes of disease, and how various diseases are influenced by environmental factors, the analysis breaks new ground in understanding the interactions between environment and health. The estimate reflects how much death, illness and disability could be realistically avoided every year as a result of better environmental management.

The report estimates that more than 13 million deaths annually are due to preventable environmental causes. Nearly one third of death and disease in the least developed regions is due to environmental causes. Over 40% of deaths from malaria and an estimated 94% of deaths from diarrhoeal diseases, two of the world's biggest childhood killers, could be prevented through better environmental management.

Diseases with the largest total annual health burden from environmental factors, in terms of death, illness and disability or Disability Adjusted Life Years (DALYs: Disability Adjusted Life Years: The sum of years of potential life lost due to premature mortality and the years of productive life lost due to disability) are:

- Diarrhoea (58 million DALYS per year; 94% of the diarrhoeal burden of disease) largely from unsafe water, sanitation and hygiene
- Lower respiratory infections (37 million DALYs per year; 41% of all cases globally) largely from air pollution, indoor and outdoor.

- Unintentional injuries other than road traffic injuries (21 million DALYs per year; 44 % of all cases globally), classification which includes a wide range of industrial and workplace accidents.
- Malaria (19 million DALYs per year; 42% of all cases globally), largely as a result of poor water resource, housing and land use management which fails to curb vector populations effectively.
- Road traffic injuries (15 million DALYS per year; 40% of all cases globally), largely as a result of poor urban design or poor environmental design of transport systems.
- Chronic Obstructive Pulmonary disease (COPD) -- a slowly progressing disease characterized by a gradual loss of lung function. (COPD, 12 million DALYs per year; 42% of all cases globally) largely as a result of exposures to workplace dusts and fumes and other forms of indoor and outdoor air pollution.
- Perinatal conditions (11 million DALYS per year; 11% of all cases globally).

Most of the same environmentally-triggered diseases also rank as the biggest killers outright -- although they rank somewhat differently in order of lethality. Diseases with the largest absolute number of deaths annually from modifiable environmental factors (these are all parts of the environment amenable to change using available technologies, policies, preventive and public health measure). These diseases include:

- 2.6 million deaths annually from cardiovascular diseases
- 1.7 million deaths annually from diarrhoeal diseases
- 1.5 million deaths annually from lower respiratory infections
- 1.4 million deaths annually from cancers
- 1.3 million deaths annually from chronic obstructive Pulmonary disease
- 470,000 deaths annually from road traffic crashes
- 400,000 deaths annually from unintentional injuries

The report shows that one way or another, the environment significantly affects more than 80% of these major diseases. Moreover, it looks to quantify only those environmental hazards that are modifiable - that is, those that are readily amenable to change through policies or technologies that already exist. The report also spells out for us how much environment-related disease is preventable.

By acting assertively and setting priorities for measures aimed at curbing the most serious killers, millions of unnecessary deaths can be prevented every year. Working with sectors such as energy, transport, agriculture and industry to ameliorate the root environmental causes of ill health is crucial.

3.11. Conclusion

It's impossible in this day and age to avoid all environmental toxins. What you can do, however, is limit your exposure as much as possible with the following tips:

- Buy and eat, as much as possible, organic produce and free-range, organic foods. If you can only purchase one organic product it probably should be free range organic eggs. Fortunately some grocery stores now have these available.
- Rather than eating fish, which is largely contaminated with PCBs and mercury, consume a high-quality purified fish .
- Avoid processed foods -- remember that they're processed with chemicals!
- Switch over to natural brands of toiletries, including shampoo, toothpaste, antiperspirants and cosmetics.
- Avoid spraying insect repellants that contain DEET on your body. There are safe, effective and natural alternatives out there, like Neem-Based Botanical Outdoor Gel.
- Remove any metal fillings as they're a major source of mercury. Be sure to have this done by a qualified biological dentist. Although nearly any dentist is technically qualified to replace your amalgam fillings, far less than 95 percent have any clue on how to do it properly so your risk of mercury exposure is minimized.
- Avoid using artificial air fresheners, dryer sheets, fabric softeners or other synthetic fragrances as they can pollute the air you are breathing.

- Avoid artificial food additives of all kind, including artificial sweeteners.
- Get plenty of safe sun exposure to boost your vitamin D levels and your immune system (you'll be better able to fight disease).
- Have your tap water tested and, if contaminants are found, install an appropriate water filter on all your faucets (even those in your shower or bath).

4. Environmental Pollution and Human Health

4.1. Introduction

Pollution is the introduction of damaging or toxic materials into the natural environment that can cause damage to environment and harm or discomfort to humans or other living species. It is the addition of another form of any substance or form of energy to the environment at a rate faster than the environment can accommodate it by dispersion, breakdown, recycling, or storage in some harmless form.

Environmental pollution has existed for centuries but only started to be significant following the industrial revolution in the 19th century. Pollution occurs when the natural environment cannot destroy an element without creating harm or damage to itself. The elements involved are not produced by nature, and the destroying process can vary from a few days to thousands of years (that is, for instance, the case for radioactive pollutants). In other words, pollution takes place when nature does not know how to decompose an element that has been brought to it in an unnatural way.

When people think of environmental pollution, most focus on fossil fuel and carbon emissions, but there are different contributing factors. Chemical pollution in bodies of water contributes to illnesses. Electromagnetic pollution has effects on human health but is uncommonly considered in present times despite the fact we essentially expose ourselves to it on a daily basis.

Environmental pollution is one the greatest challenges that the world is facing today. It began with the industrial revolution, increasing day by day and causing irreparable damage to Mother Earth. Environmental pollution has its own causes, effects and solutions. Looking into these will help you identify the causes and what steps you can take to mitigate those effects. Broadly, environmental pollution consists of six basic types of pollution, i.e. air, water, land, soil, noise, and light.

4.2. Causes of Environmental Pollution

Air pollution comes from both natural and human-made (anthropogenic) sources. However, globally human-made pollutants from combustion, construction, mining, agriculture and warfare are increasingly significant in the air pollution equation. Agricultural air pollution comes from contemporary practices which include clear felling and burning of natural vegetation as well as spraying of pesticides and herbicides. About 400 million metric tons of hazardous wastes are generated each year. The United States alone produces about 250 million metric tons. Americans constitute less than 5% of the world's population, but produce roughly 25% of the world's CO₂, and generate approximately 30% of world's waste.

In February 2007, a report by the Intergovernmental Panel on Climate Change (IPCC), representing the work of 2,500 scientists, economists, and policymakers from more than 120 countries, said that humans have been the primary cause of global warming since 1950. Humans have ways to cut greenhouse gas emissions and avoid the consequences of global warming, a major climate report concluded. But to change the climate, the transition from fossil fuels like coal and oil needs to occur within decades, according to the final report this year from the UN's Intergovernmental Panel on Climate Change (IPCC).

Some of the more **common soil contaminants** are chlorinated hydrocarbons (CFH), heavy metals (such as chromium, cadmium—found in rechargeable batteries, and lead—found in lead paint, aviation fuel and still in some countries, gasoline), MTBE, zinc, arsenic and benzene. In 2001 a series of press reports culminating in a book called *Fateful Harvest* unveiled a widespread practice of recycling industrial byproducts into fertilizer, resulting in the contamination of the soil with various metals. Ordinary municipal landfills are the source of many chemical substances entering the soil environment (and often groundwater), emanating from the wide variety of refuse accepted, especially substances illegally discarded there, or from pre-1970 landfills that may have been subject to little control in the U.S. or EU. There have also been some unusual releases of polychlorinated dibenzodioxins, commonly called *dioxins* for simplicity, such as TCDD.

Pollution can also be the consequence of a **natural disaster**. For example, hurricanes often involve water contamination from sewage, and petrochemical spills from ruptured boats or automobiles. Larger scale and environmental damage is not uncommon when **coastal oil rigs** or **refineries** are involved. Some sources of pollution, such as **nuclear power plants** or **oil tankers**, can produce widespread and potentially hazardous releases when accidents occur.

In the case of **noise pollution** the dominant source class is the motor vehicle, producing about ninety percent of all unwanted noise worldwide. Let us first take a look at the causes of environmental pollution:

4.2.1. Industries

Industries have been polluting our environment especially since the beginning of the industrial revolution, as mentioned above, notably due to the increasing use of fossil fuels. In the 19th century and for a significant part of the 20th century, coal has been used to make machines work faster, replacing human force. Though pollution by industries mainly causes air pollution, soil and water contamination can also occur. This is particularly the case for power-generating industries, such as plants producing electricity (may they be a dam, a nuclear reactor or some other type of plant).

Principal **stationary pollution sources** include chemical plants, coal-fired power plants, oil refineries, petrochemical plants, nuclear waste disposal activity, incinerators, large livestock farms (dairy cows, pigs, poultry, etc.), PVC factories, metals production factories, plastics factories, and other heavy industry.

4.2.2. Transportation

The transportation of energy can be harmful to the environment. We can take as an example the transportation of petrol through pipelines; if there is a leak in the pipeline, soil will automatically be polluted. At the same time, if the tanker transporting the petrol from its production plant to the place where it will be consumed leaks or sinks, the water will get contaminated.

4.2.3. Agricultural activities

Agriculture is mainly responsible for the contamination of water and soil. This is caused by the increased use of pesticides, as well as by the intensive character of its production. Almost all pesticides are made from chemical substances and are meant to keep diseases and threatening animals away from the crops. However, by keeping these forms of life away, harm is almost always made to the surrounding environment as well. Furthermore, as agriculture gets more and more intensive to feed the increasing world population, more environments and ecosystems are destroyed to make space for the crops. Some of them, like rapeseed –used to make oil – demand a lot of space for a relatively small output.

4.2.4. Trading activities

Trading activities include the production and exchange of goods and services. Concerning goods, pollution can be caused by packaging (which often involves the use of plastic, which is made from fossil fuels) or transport, mainly.

4.2.5. Residences

Residential areas provide their fair share of pollution as well. First, to be able to build homes, natural environment has to be destroyed in one way or another. Wildlife and plants are driven away and replaced by human constructions. As it requires the work of industries, construction itself is also a source of contamination of the environment. Then, when people settle in, they will produce waste every day, including a part that cannot be processed by the environment without harm yet.

4.2.6. Pollution from cars, trucks, and other vehicles

Pollution from cars, trucks, and other vehicles is and has been our major environmental pollution issue for almost a century now. **Motor vehicle emissions** are one of the leading causes of air pollution.

4.2.7. Fossil fuel emissions from power plants

Fossil fuel emissions from power plants which burn coal as fuel contributed heavily, along with vehicles burning fossil fuels, to the production of smog. Smog is the result of fossil fuel combustion combined with sunlight and heat. The result is a toxic gas which now surrounds our once pristine planet. This is known as “ozone smog” and means we have more problems down here than we do in the sky. Carbon dioxide is another product from all of the vehicles on the planet as well as unreformed power plants and other industrial facilities. A continually growing population of humans and **clear cutting of forests** have exacerbated this problem so natural defenses are no longer present and carbon dioxide levels are on the rise.

4.2.8. Water pollution is a major issue

Many industries dump wastes into rivers, lakes, ponds, and streams in an attempt to hide wastes from EPA inspectors. These water sources feed major crops and food becomes contaminated with a variety of chemicals and bacteria, causing rampant health problems.

4.2.9. Radiation

Primarily, there is radiation from the sun. The sun is wonderful, but the only reason we are able to survive on this planet so close to the sun is due to the fact of natural shielding against solar radiation. As the protective ozone layer around the planet has become thinner, ultraviolet radiation has risen significantly, causing increases in skin cancers and other types of cancer in all countries, killing millions of people every year. More radiation is a problem. The sun shining brightly on a naked planet is not the only source of radiation we are exposed to.

Electromagnetic radiation is another insidious culprit. Once upon a time, the major concern around this type of radiation was due to high tension wires which carry huge amounts of electricity to cities. Now, we even carry sources of this radiation with us as cell phones, laptops, tablets and other wireless devices.

4.3. Effects of Environmental Pollution

We have identified the main causes of environmental pollution, let us study the negative effects on the environment.

4.3.1. Effects on human health

The effects of environmental pollution on humans are mainly physical, but can also turn into neuro-affectations in the long term. The best-known troubles to us are respiratory, in the form of allergies, asthma, irritation of the eyes and nasal passages, or other forms of respiratory infections (Figure 4.1). Notably, these well spread affectations can be observed when **air pollution** is high in cities, when the weather gets hot, for instance. On top of that, environmental pollution has been proven to be a major factor in the development of cancer. This can happen for example when we eat remnants of pollutants used in the production of processed foods, or pesticides from the crops. Other, rarer, diseases include hepatitis, typhoid affectations, diarrhoea and hormonal disruptions. **Adverse air quality** can kill many organisms including humans.

Ozone pollution can cause respiratory disease, cardiovascular disease, throat inflammation, chest pain, and congestion. **Water pollution** causes approximately 14,000 deaths per day, mostly due to contamination of drinking water by untreated sewage in developing countries. An estimated 500 million Indians have no access to a proper toilet, over ten million people in India fell ill with waterborne illnesses in 2013, and 1,535 people died, most of them children. 500 million Chinese lack access to safe drinking water. A 2010 analysis estimated that 1.2 million people died prematurely each year in China because of **air pollution**. The WHO estimated in 2007 that air pollution causes half a million deaths per year in India. Studies have estimated that the number of people killed annually in the United States could be over 50,000.

Oil spills can cause skin irritations and rashes. **Noise pollution** induces hearing loss, high blood pressure, stress, and sleep disturbance. **Mercury** has been linked to developmental deficits in children and neurologic symptoms. Older people are majorly exposed to diseases induced by air pollution. Those with heart or lung disorders are at additional risk. Children and infants are also at serious risk. **Lead** and other **heavy metals** have been shown to cause neurological problems. Chemical and **radioactive** substances can cause cancer, as well as birth defects.

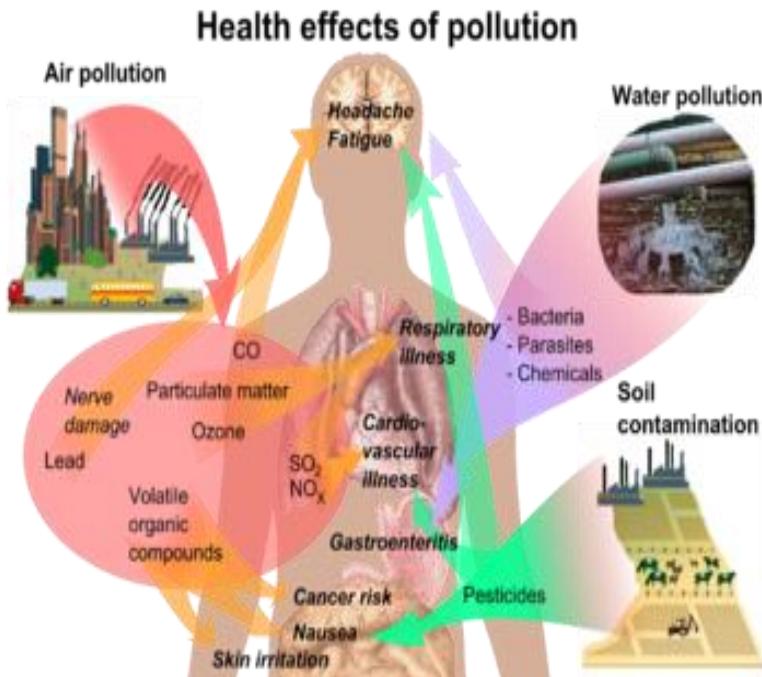


Figure 4.1. Health effects of pollution. Source: WHO

4.3.2. Effects on animal

Environmental pollution mainly affects animal by causing harm to their living environment, making it toxic for them to live in. Acid rains can change the composition of rivers and seas, making them toxic for fishes, an important quantity of ozone in the lower parts of the atmosphere can cause lung problems to all animals. Nitrogen and phosphates in water will cause overgrowth of toxic algae, preventing other forms of life to follow their normal course. Eventually, soil pollution will cause harm and sometimes even

the destruction of microorganisms, which can have the dramatic effect of killing the first layers of the primary food chain.

4.3.3. Effects on plants

As for animals, plants, and especially trees, can be destroyed by acid rains (and this will also have a negative effect on animals as well, as their natural environment will be modified), ozone in the lower atmosphere block the plant respiration, and harmful pollutants can be absorbed from the water or soil.

4.3.4. Effects on the ecosystem

In short, environmental pollution, almost exclusively created by human activities, has a negative effect on the ecosystem, destroying crucial layers of it and causing an even more negative effect on the upper layers.

4.3.5. Effects on environment

Pollution has been found to be present widely in the environment. There are a number of effects of this:

- **Biomagnification** describes situations where toxins (such as **heavy metals**) may pass through trophic levels, becoming exponentially more concentrated in the process.
- **Carbon dioxide emissions** cause ocean acidification, the ongoing decrease in the pH of the Earth's oceans as CO₂ becomes dissolved.
- **The emission of greenhouse gases** leads to global warming which affects ecosystems in many ways.
- **Invasive species** can out compete native species and reduce biodiversity. Invasive plants can contribute debris and biomolecules (allelopathy) that can alter soil and chemical compositions of an environment, often reducing native species competitiveness.
- **Nitrogen oxides** are removed from the air by rain and fertilise land which can change the species composition of ecosystems.
- **Smog** and haze can reduce the amount of sunlight received by plants to carry out photosynthesis and leads to the production of tropospheric ozone which damages plants.

- Soil can become infertile and unsuitable for plants. This will affect other organisms in the food web.
- **Sulfur dioxide** and **nitrogen oxides** can cause acid rain which lowers the pH value of soil.

4.4. Toxic Pollution and Human Health

The WHO has estimated that environmental exposures contribute to 19% of cancer incidence worldwide. Additionally, a WHO Global Health Risks report looked at five environmental exposures, (unsafe water, sanitation and hygiene, urban outdoor air pollution, indoor smoke from solid fuels, lead exposure and climate change), and estimated they account for nearly 10% of deaths and disease burden globally and around one quarter of deaths and disease burden in children under the age of five. The connection between pollution, notably toxic substance pollution, and human health has long been made in the developed world. Incidents such as Love Canal, a hazardous waste site in New York causing illness in the 1970s, brought industry pollutants and their effect on human health to prominence in public health studies. However, these connections between toxic pollution and human health have largely not been made as clearly in the developing world.

The generation of the list of industry sources was based on an analysis of toxic pollutants found at the source sites and a projection of their related human health impacts. The list sets out the most significant industry sectors based on these toxic pollutants, ranked by estimated health impacts. Pollutant types examined in the 2012 report only include those with measurable health outcomes whose contribution to DALYs can be calculated. **Lead, chromium, mercury, and asbestos are the toxic pollutants highlighted below. These pollutants have quantifiable health outcomes that are given disability weights by the WHO. Toxic pollutants without established health outcomes recognized by the WHO cannot be quantified by a DALY measurement and were not included in the burden of disease calculations.**

4.4.1. Lead

Lead is a metal that is found in various ores and is used in many different products. The toxic properties of lead are well documented yet it is

still used in varied and important ways within the world economy because of its dense, corrosion-resistant, and malleable characteristics.

Scope and Nature of Problem. Lead is the most pervasive pollutant found in the Blacksmith Institute's database and is a well-documented health hazard. The Blacksmith Institute has identified over 500 sites polluted by lead, putting an estimated 16 million people at risk. Based on the Blacksmith Institute's investigations, the top sources contributing to lead pollution, by population, are lead smelting, mining and ore processing, industrial estates and lead-acid battery recycling and manufacturing. Lead pollution is also found in polluted sites around product manufacturing sites, e-waste recycling and chemical manufacturing sites. In the U.S., lead is most predominantly used for manufacturing lead-acid batteries. But around the world lead is used in many different industrial-manufacturing processes for plumbing materials, alloys, paints, ammunition, and in a limited amount of countries, as a lubricating agent in gas. This extensive list illustrates the widespread problem of lead pollution.

The majority of lead contaminated sites in the Blacksmith Institute's database are found in Africa, South America, South and Southeast Asia, but the problem of lead pollution plagues most developing countries worldwide. Its uses are varied; in Latin America it has often been utilized for ceramic glazing and in other countries leaded gasoline is still used. In the U.S. lead paint is the cause of a majority of lead exposures and such exposures can be expected in most countries since paint pigments using lead were commonly used worldwide up until a few decades ago. Global production of lead was expected to increase 9% in 2011 to 4.52 million tons, due to increases in China, India and Mexico, with China accounting for one-half of all lead mining production. Increasing quantities of lead are being recycled. But often recycling occurs at uncontrolled or poorly controlled facilities in the informal economic sector, making lead reprocessing itself a significant problem in many countries. Lead enters the environment through the air (as dust) and through water; the specific form of introduction varies depending on the industry or product.

Health Impacts. When humans inhale or ingest lead it is distributed to the brain, liver, kidney and bones and can be stored in the blood, teeth or bones. Because lead is an element, it cannot be broken down or destroyed; it accumulates in the body as long as a person continues to be exposed to it.

Lead accumulation leads to neurological, gastrointestinal, and cardiovascular problems. Lead exposure during pregnancy can lead to miscarriage, stillbirth, low birth weights, premature births and birth defects. The International Agency for Research on Cancer declares it to be a possible human carcinogen. Children are exceptionally vulnerable because their bodies absorb 4-5 times as much lead as adults; even at the lowest levels of exposure lead is toxic to children. The brain damage resulting from lead exposure in children is untreatable and includes mild mental retardation, decreased IQ, shortened attention spans, loss of executive function, increased risk of dyslexia, and diminished productivity. It is estimated that the effects of mild mental retardation and cardiovascular problems alone, caused by lead exposure, amount to almost 1% of the total global burden of disease, with developing countries carrying the largest burden.

4.4.2. Chromium

Chromium is a metallic element that occurs naturally in the environment in the form of trivalent and hexavalent chromium. Trivalent chromium, or chromium-3 can be found in fruits, vegetables, grains and meat and is considered a key part of the human diet. Hexavalent chromium, or chromium-6 is naturally occurring through erosion of ore deposits, or is leaked into the environment by industrial processes. Chromium-6 is used in the manufacturing and processing of steel, alloys, plating, dyes, and leather and can be a very serious health risk. In certain environmental circumstances trivalent chromium can turn into hexavalent chromium, and vice versa, after being released into the environment.

Scope and Nature of Problem. The Blacksmith Institute has identified over 150 sites polluted by chromium, putting more than 5.5 million people at risk of exposure from the sites identified. The top sources of chromium pollution, by at risk population, in the Blacksmith Institute's database are industrial estates, product manufacturing, mining and ore processing, tanneries, industrial dumpsites, chemical manufacturing and the dye industry. It also is found at e-waste recycling sites, petrochemical plants, and heavy industry sites. The majority of the chromium-polluted sites in the Blacksmith Institute's database are in South Asia, mostly within Pakistan and India. However, given the prevalence of tanneries and mining in various African, South American and North Asian countries, Blacksmith expects chromium pollution to be found throughout the developing world. Chromium

enters the environment as dust in the air or is leached into groundwater from unmanaged waste from ore processing sites. Chromium exposure occurs mainly through dermal contact with contaminated soil or water, inhalation of dust or soil, ingestion of food exposed to chromium through contaminated water or soil and direct ingestion of contaminated water.

Health Impacts. The two types of chromium differ drastically in their level of toxicity. Chromium-3 in appropriate amounts is an essential nutrient, but can be harmful in large quantities. Chromium-6 is a known carcinogen and when inhaled has been proven to cause lung cancer in humans. There is less understanding of the human health impacts of ingesting chromium-6 in drinking water. Some recent studies have linked ingestion to an increased risk for stomach and lung cancer, but authorities have not officially recognized the health impacts from ingestion. However, as recognition of the known toxicity of the element the U.S. EPA has issued standards limiting the level of chromium in drinking water.

4.4.3. Mercury

Mercury is a naturally occurring metal that can exist in the elemental form (a liquid at room temperature) or as organic or inorganic mercury. It occurs in different mineral forms, including in association with coal. Emissions from the burning of coal are the largest source of mercury pollution in the air in the U.S. Mercury in the atmosphere is a pollutant that travels globally and is of major concern, but this is outside the scope of Blacksmith Institute's investigations and is not addressed in this report. The use of mercury in mining and industrial operations, however, is a major problem addressed by Blacksmith Institute.

Scope and Nature of Problem. The Blacksmith Institute's database contains almost 350 sites contaminated with mercury, putting close to 10 million people at risk from the identified sites. It is the second most prevalent pollutant in the database. The top sources of mercury pollution are artisanal gold processing, mining and ore processing, coal mining, processing and localized air pollution related to coal combustion at poorly controlled sites, and chemical manufacturing, notably for older chlor-alkali plants making chlorine.

Artisanal mining of gold ores and processing using mercury is common worldwide. Mercury is used to recover gold from ores and is

released into the environment through mine tailings after processing or as a result of evaporating mercury from gold-mercury amalgams to recover the metallic gold. Mercury is a bio accumulative toxin and will persist in the food chain. Under certain environmental conditions inorganic mercury can be transformed into the most toxic form of mercury, methyl mercury. Human populations at polluted sites can be exposed through dermal contact with contaminated soil and water, ingestion of contaminated water, inhalation of dust and vapor and ingestion of contaminated food.

Health Impacts. Mercury health effects depend on the type of mercury to which a person is exposed. In general, health impacts include renal toxicity, damage to the immune system, alteration of genetic and enzyme systems and neurological damage, especially in babies exposed in utero. Methyl mercury is the most toxic form of mercury because it is absorbed quickly in the body and expelled much more slowly. Currently there is not enough human exposure data to make links between mercury and cancer. Mercury health effects are difficult to quantify using WHO's approach because disability weights have not yet been assigned to the types of health impacts mercury causes. However, because of the prevalence and toxicity of mercury we have included it in the report.

4.4.4. Asbestos

Asbestos refers to a group of silicate fibers that are naturally occurring in the earth. These fibers are used for their strength and flexibility, they can be bonded together to create products like insulation, roofing, shingles, tiles, paper products packaging, and car parts. Asbestos is used heavily in building products because of its natural fire retardant features.

Scope and Nature of Problem. Asbestos is recorded in a small number of sites in the Blacksmith Institute's database, but potentially puts over 350,000 people at risk. Asbestos enters the environment through either mining of the mineral or through the use of products containing asbestos. Occupational exposure to asbestos is a major issue for people that work in industries that mine asbestos or make products out of asbestos. Exposure pathways are mostly from inhalation of airborne asbestos fibers.

Because of the large amount of information about the toxic nature of asbestos, it is tightly regulated in most developed countries. All new uses of

asbestos were banned in 1989 in the United States and the use of asbestos in manufacturing, processing and distribution is closely monitored. However, despite bans in 52 countries, asbestos continues to be used in low and middle-income countries. White asbestos is used in cheap building materials in China, India, Russia and Brazil, while blue and brown asbestos are no longer used anywhere. White asbestos is mined and processed in both the developed and developing world, with Russia leading asbestos production in 2008. The World Federation of Public Health Organizations (WFPHA), the International Commission on Occupational Health (ICOH), and the International Trade Union Confederation (ITUC) have called for a global asbestos ban, especially since asbestos mining and processing plants in developing countries are often under regulated and lack necessary pollution controls.

Health Impacts. Asbestos affects the whole respiratory system. There are three serious health impacts, asbestosis, lung cancer and mesothelioma. Asbestosis is a serious, non-cancer form of lung disease. There is no treatment or cure for it and it causes shortness of breath. Lung cancer is the leading cause of death from asbestos exposure. Mesothelioma, another type of cancer, affects the lining of the lungs, abdomen and heart; largely all cases of mesothelioma can be directly linked back to asbestos exposure.

4.5. Various Types of Environmental Pollution

In particular, the natural environments subject to pollution largely include water, air, and land. Presence of substances such as (liquid, gas, solid) or energy such as (heat, light, radiation, noise) whose qualities directly or indirectly changes the natural environmental process in part or in whole, and has the potential to cause or can cause damage to the health or well-being of humans, animals, or plants defines pollution. The major forms of pollution are listed below along with the particular **contaminant** relevant to each of them.

4.5.1. Air Pollution

Air pollution is the release of chemicals and particulates into the atmosphere. Common gaseous pollutants include carbon monoxide, sulfur dioxide, chlorofluorocarbons (CFCs) and nitrogen oxides produced by industry and

motor vehicles (Figure 4.2, 4.3, 4.4, 4.5, 4.6 and 4.7). Photochemical ozone and smog are created as nitrogen oxides and hydrocarbons react to sunlight. Particulate matter, or fine dust is characterized by their micrometre size PM_{10} to $PM_{2.5}$. Air pollution results from both human and natural activities. Emissions from power plants present a perfect example of human activities contributing to air pollution whereas volcanic eruptions and forest fires are some of the natural aspects. Some other examples of air pollution include the burning of fossil fuels, such as coal, oil, or gas, harmful off-gassing from things such as paint, plastic production, and radiation spills or nuclear accidents. Air pollution is linked to asthma, allergies and other respiratory illnesses.

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Sources of Air Pollutants

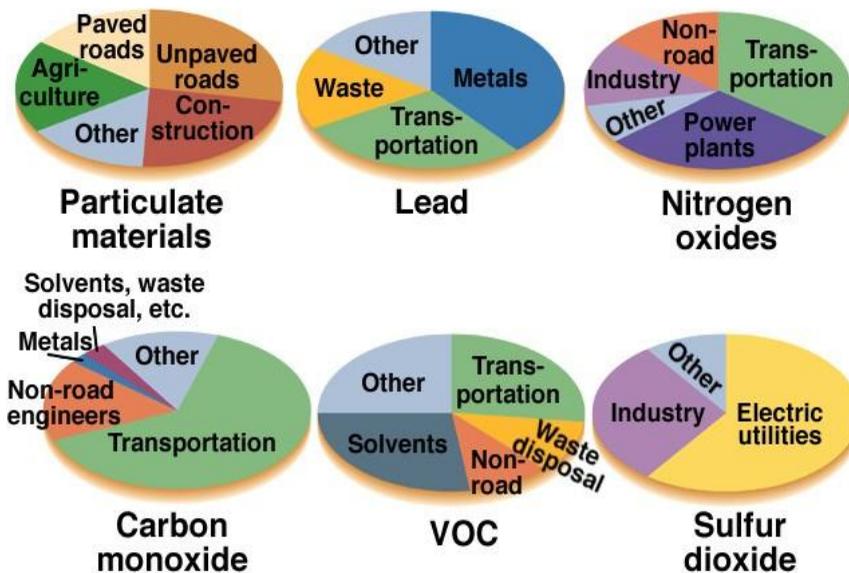


Figure 4.2. Sources of Air Pollutants. Source: The McGraw-Hill Companies, Inc.

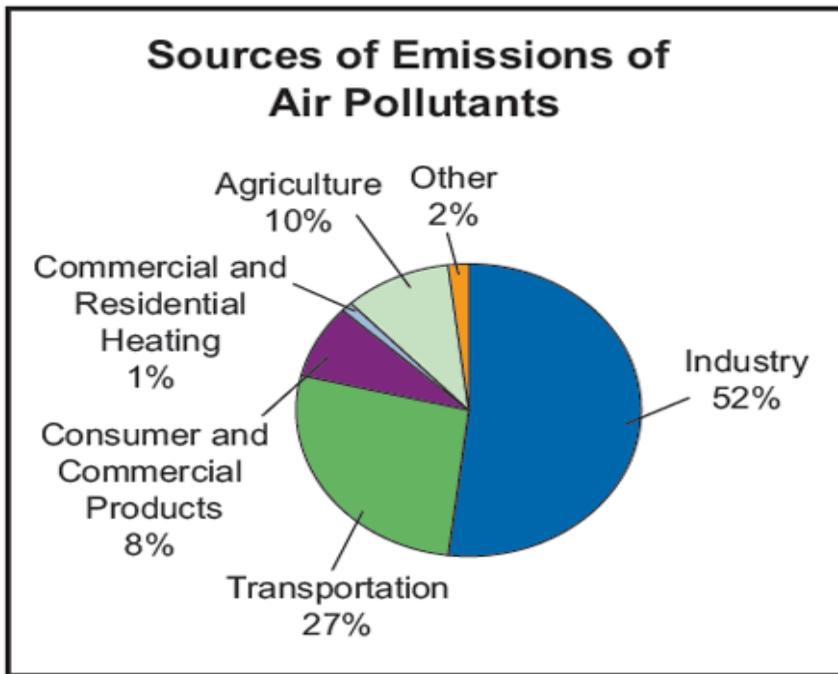


Figure 4.3. Sources of emissions of Air Pollutants.
Source: The McGraw-Hill Companies, Inc.



Figure 4.4. Girl walks through smog in Beijing, where small-particle pollution is 40 times over International Safety Standard.
Source: Natural News



Figure 4.5. Air pollution in India. Source: The Indian Express

HEALTH EFFECTS OF AIR POLLUTION

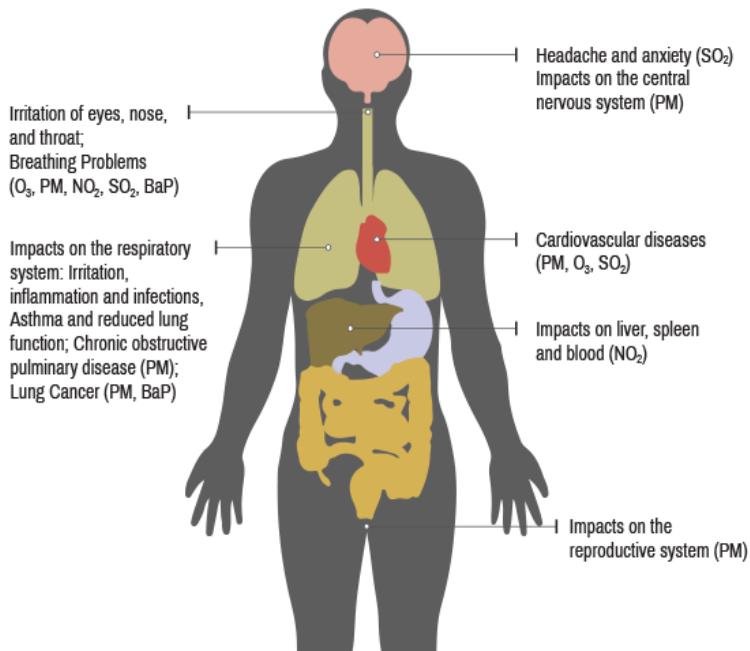


Figure 4.6. Health effects of Air Pollution. Source: WHO

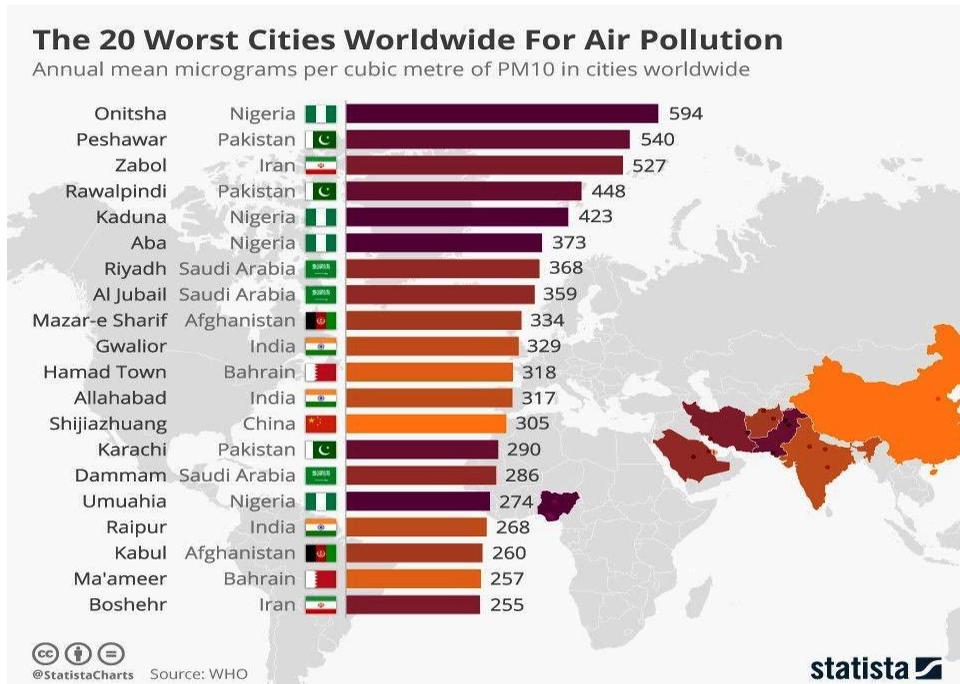


Figure 4.7. The 20 worst cities worldwide for air pollution. Source: WHO 2015

Air pollution can harm us when it accumulates in the air in high enough concentrations. Millions of people live in areas where urban smog, particle pollution, and toxic pollutants pose serious health concerns. People exposed to high enough levels of certain air pollutants may experience:

- Irritation of the eyes, nose, and throat
- Wheezing, coughing, chest tightness, and breathing difficulties
- Worsening of existing lung and heart problems, such as asthma
- Increased risk of heart attack

In addition, long-term exposure to air pollution can cause cancer and damage to the immune, neurological, reproductive, and respiratory systems. In extreme cases, it can even cause death. Air pollution is a problem for all of us.

4.5.2. Water Pollution

Water pollution is the act of contaminating water bodies including rivers, oceans, lakes, streams, aquifers, and groundwater (Figure 4.8, 4.9, 4.10 and 4.11). It occurs when foreign harmful materials like chemicals, waste matter, or contaminated substances are directly or indirectly discharged into water bodies. Any alterations in the chemical, physical, or biological water properties qualify as water pollution. Very often, the primary contributors to water pollution are human activities since they introduce substances that contaminate the water with harmful chemicals and toxic materials. Water pollution is categorized into point source, non-point source, and groundwater.

Point source water pollution occurs when the contaminants enter a water body from a single identifiable source while **non-point source** occurs as a result of cumulative effects of different amounts of contaminants. **Groundwater** pollution occurs through infiltration and affects groundwater sources such as wells or aquifers.

Water is considered the second most polluted environmental resource after air pollution. Some examples of water pollution:

- Raw sewage running into lake or streams
- Industrial waste spills contaminating groundwater
- Radiation spills or nuclear accidents
- Illegal dumping of substances or items within bodies of water
- Biological contamination, such as bacteria growth
- Farm runoff into nearby bodies of water



Figure 4.8. Contamination of water bodies. Source: ENVIS



Figure 4.9. Water pollution killing fish species. Source: d.ibtimes.co.uk

A large number of chemicals that either exist naturally in the land or are added due to human activity dissolve in the water, thereby contaminating it and leading to various diseases.

Pesticides. The organophosphates and the carbonates present in pesticides affect and damage the nervous system and can cause cancer. Some of the pesticides contain carcinogens that exceed recommended levels. They contain chlorides that cause reproductive and endocrinal damage.

Lead. Lead is hazardous to health as it accumulates in the body and affects the central nervous system. Children and pregnant women are most at risk.

Fluoride. Excess fluorides can cause yellowing of the teeth and damage to the spinal cord and other crippling diseases.

Nitrates. Drinking water that gets contaminated with nitrates can prove fatal especially to infants that drink formula milk as it restricts the amount of oxygen that reaches the brain causing the 'blue baby' syndrome. It is also linked to digestive tract cancers. It causes algae to bloom resulting in eutrophication in surface water.

Petrochemicals. Benzene and other petrochemicals can cause cancer even at low exposure levels.

Chlorinated solvents. These are linked to reproduction disorders and to some cancers.

Arsenic. Arsenic poisoning through water can cause liver and nervous system damage, vascular diseases and also skin cancer.

Other heavy metals. –Heavy metals cause damage to the nervous system and the kidney, and other metabolic disruptions.

Salts. It makes the fresh water unusable for drinking and irrigation purposes.

Exposure to polluted water can cause diarrhoea, skin irritation, respiratory problems, and other diseases, depending on the pollutant that is in the water body. Stagnant water and other untreated water provide a habitat for the mosquito and a host of other parasites and insects that cause a large number of diseases especially in the tropical regions. Among these, malaria is undoubtedly the most widely distributed and causes most damage to human health.

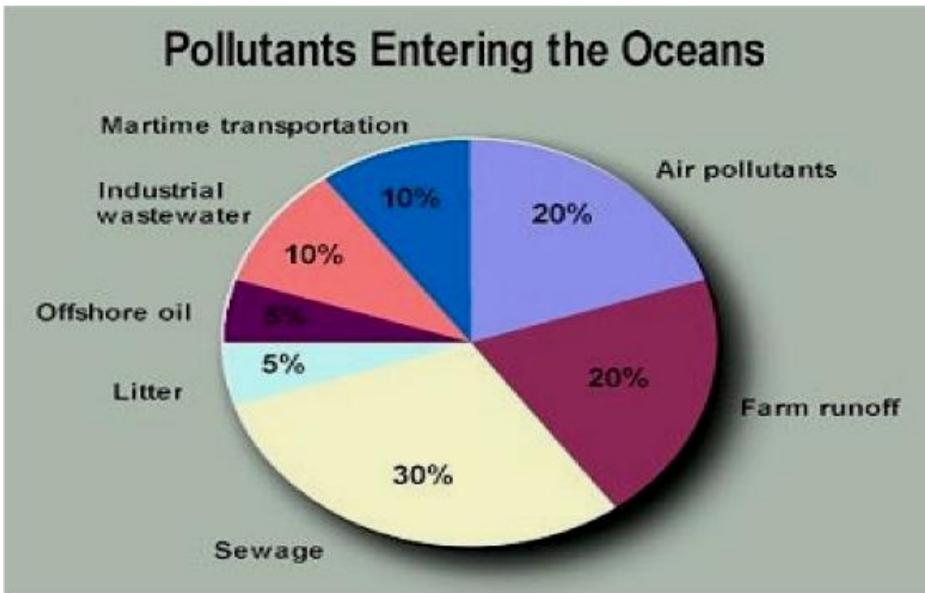


Figure 4.10. Pollutants entering the oceans. Source: Prezi

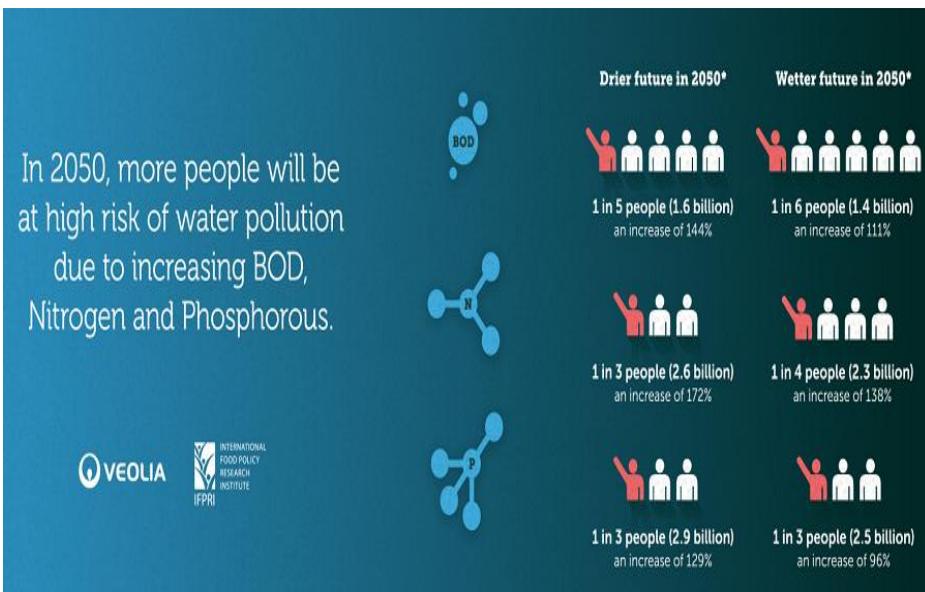


Figure 4.11. In 2050, more people will be at high risk of water pollution due to increasing BOD, Nitrogen and Phosphorous. Source: International Food Policy Research Institute

4.5.3. Land Pollution

Human activities that pollute land include the application of herbicides, use of pesticides and insecticides, inappropriate disposal of garbage and urban over-development. Land pollution is also caused by the dangerous disposal of harmful toxic wastes, from the careless dumping of waste oil from cars to the organized but potentially dangerous dumping of radioactive by-products from nuclear energy plants (Figure 4.12).

Water percolating through garbage dumps and landfills can introduce toxins like arsenic into groundwater, causing a host of illnesses including birth defects. Garbage produced by improper disposal of computers and other electronic devices, introduces toxic chemicals like lead and cadmium into the water tables through leaching.



Figure 4.12. Land Pollution. Source: UN Environment

Businesses large and small have been improperly dumping waste products into the ground for decades. Large factories have been dumping toxic by-products into private landfills, while small businesses like automotive service stations and dry cleaners have been dumping waste out in their own backyards. All these toxic chemicals eventually leach poisons into the water table, where humans consume them through drinking water or eating contaminated foods like freshwater fish.

The toxic materials that pollute the soil can get into the human body directly by:

- coming into contact with the skin
- being washed into water sources like reservoirs and rivers
- eating fruits and vegetables that have been grown in polluted soil
- breathing in polluted dust or particles.

Contaminated lands can cause problems in the human respiratory system and skin. It can also cause various kinds of cancers.

4.5.4. Soil Pollution

Soil pollution happens when human activities directly or indirectly introduce destructive chemicals, substances, or objects into the soil in a way that causes damage to the immediate earthly environment (Figure 4.13). Soil degradation also contributes to soil pollution, and it occurs as a result of over-grazing, over-farming, or mining activities. The notable causes of soil pollution include agricultural farming activities, waste dumping on land, industrial activities, mining, and acid rain. Soil contamination occurs when chemicals are released by spill or underground leakage. Among the most significant soil contaminants are hydrocarbons, heavy metals, herbicides, pesticides and chlorinated hydrocarbons.



Figure 4.13. Soil Pollution. Source: UN Environment

Soil pollution occurs when soil contains chemicals that are toxic or otherwise dangerous for humans and other living things. The chemicals may be foreign to the area, or they may be naturally occurring materials that

pollute the soil by being present in dangerously high amounts. Soil pollution can have a number of harmful effects on human health. The harmful effects of soil pollution may come from direct contact with polluted soil or from contact with other resources, such as water, that have come in direct contact with the polluted soil.

The presence of heavy metals in soil in toxic amounts can cause irreversible developmental damage in children. Although lead and mercury may be found naturally in soil, high concentrations of either metal may cause damage to the developing brains of young children, which in turn may lead to neurological problems. Humans of any age may also suffer kidney or liver damage from exposure to excessive mercury in soil.

Soil that is not significantly polluted may still harm humans indirectly, according to Pollution issues. One way such soil pollution can harm humans is by bioaccumulation. Plants that are grown in lightly polluted soil continuously absorb molecules of the pollutants. Since the plants cannot get rid of these molecules, they accumulate in the plant, causing higher amounts of pollution to exist in the plant than in the soil. Animals who eat many of these polluted plants take on all the pollution those plants have accumulated. Larger animals that eat the plant-eating animals take on all the pollution from the animals they eat. Humans who eat plants or animals that have accumulated large amounts of soil pollutants may be poisoned, even if the soil itself does not contain enough pollution to harm human health.

Many common soil pollutants are carcinogenic, or cancer-causing. According to the U.S. Environmental Protection Agency, humans who are exposed to these pollutants are far more likely to develop cancer than humans who are not exposed to them. For example, regular exposure to benzene is known to cause leukemia in both children and adults. Exposure to polychlorinated biphenyls (PCBs) is linked to liver cancer.

Soil pollution can leach into the groundwater and end up in drinking supplies, according to the World Health Organization. Directly consuming the contaminated water can cause health effects associated with the types of chemicals that are in the water.

Human health can be severely affected by direct contact with contaminated soils. For example, building a playground on a contaminated

site can be disastrous since the children will tend to come into heavy contact with the contaminated soil and their development can be drastically harmed. Chromium has been linked to cancer. Lead has been linked to brain damage and kidney damage. Mercury can lead to both liver and kidney damage.

While adults can be harmed by soil pollution, children are at a much higher risk of contamination. Children's smaller bodies mean soil pollution can get ingested into their bodies at much higher relative quantities. Also, children do not take many of the precautions that adults take, including eating harmful substances. Children are still developing, and their development can be harmed by pollution.

4.5.5. Noise Pollution

Noise pollution is mostly an undesirable sound or sound which generates horrible discomfort on the ears (Figure 4.14). Noise pollution is defined as unpleasant and undesirable sound levels that cause serious discomfort to all living things. It is measured in decibels (dB).

Sound levels beyond 100 dB can cause permanent hearing loss, and noise of around 90 dB causes auditory weakness. The industrial sound limit according to the World Health Organization (WHO) is 75 dB. Noise pollution may increase your risk of hearing loss, stress, sleep disturbances, and heart disease. It merely occurs when sound waves of intense pressure reach the human ears and may even affect the body muscles due to sound vibrations. Noise pollution can be disruptive to humans' stress levels and may be harmful to unborn babies. Noise pollution similarly affects marine and wildlife animals in the same manner it affects humans, and can even cause their death. Some examples of noise pollution:

- Airplanes, helicopters, and motor vehicles
- Construction or demolition noise
- Human activities such as sporting events or concerts
- high-intensity sonar.



Figure 4.14. Noise pollution in India. Source: The Indian Express

Noise-induced hearing loss (NIHL), which can occur from one very loud noise exposure (such as an explosion) or continuous exposure to loud noise over time (such as working in a factory), affects about 15 percent of Americans. Long or repeated exposure to sounds at or above 85 decibels can cause hearing loss. The louder the sound, the shorter the amount of time it takes for NIHL to happen. Here are the average decibel ratings of some familiar sounds:

- The humming of a refrigerator: 45 decibels
- Normal conversation: 60 decibels
- Noise from heavy city traffic: 85 decibels
- Motorcycles: 95 decibels
- An MP3 player at maximum volume: 105 decibels
- Sirens: 120 decibels
- Firecrackers and firearms: 150 decibels

Your distance from the source of the sound and the length of time you are exposed to the sound are also important factors in protecting your hearing. The best way to prevent NIHL is to reduce the noise if possible, and if not wear earplugs or other protective devices to protect your hearing. If you can't do either of these, move away from the noise. . In the contemporary society, noise has become a permanent aspect owing to the daily activities such as transportation, industrial manufacturing, and technology. In contrast to the other types of pollution, noise pollution lacks the element of accumulation in the environment.

A new analysis conducted an environmental assessment of US noise pollution as a cardiovascular health hazard. The analyses suggested that a 5-decibel noise reduction would reduce the prevalence of high blood pressure by 1.4 percent and coronary heart disease by 1.8 percent. According to research published in *Environmental Health Perspectives*, long-term exposure to traffic noise may account for approximately 3 percent of coronary heart disease deaths (or about 210,000 deaths) in Europe each year.

4.5.6. Thermal Pollution

Production and Manufacturing plants are biggest source of thermal pollution. These plants draw water from nearby source to keep machines cool and then release back to the source with higher temperature. When heated water returns to the river or ocean, the water temperature rises sharply (Figure 4.15). When oxygen levels are altered in the water, this can also degrade the quality and longevity of life in wildlife that lives underwater. This process can also wipe away streamside vegetation, which constantly depends on constant levels of oxygen and temperature. By altering these natural environments, industries are essentially helping decrease the quality of life for these marines-based life forms and can ultimately destroy habitats if they are not controlled and careful about their practices.

Soil erosion is another major factor that causes thermal pollution. Consistent soil erosion causes water bodies to rise, making them more exposed to sunlight. The high temperature could prove fatal for aquatic biomes as it may give rise to anaerobic conditions.

Trees and plants prevent sunlight from falling directly on lakes, ponds or rivers. When deforestation takes place, these water bodies are directly exposed to sunlight, thus absorbing more heat and raising its temperature. Deforestation is also a main cause of the higher concentrations of greenhouse gases i.e. global warming in the atmosphere.

Urban runoff discharged to surface waters from paved surfaces like roads and parking lots can make water warmer. During summer seasons, the pavement gets quite hot, which creates warm runoff that gets into the sewer systems and water bodies.

Natural causes like volcanoes and geothermal activity under the oceans and seas can trigger warm lava to raise the temperature of water bodies. Lightning can also introduce massive amount of heat into the oceans. This means that the overall temperature of the water source will rise, having significant impacts on the environment.

Thermal pollution is not thought to directly affect human health, but it may have a dramatic impact on aquatic organisms, including plants, insects, microorganisms, and fish. The most important impact of warm water thermal pollution is a decrease in dissolved oxygen. An increase in temperature actually encourages rapid growth of algae which in turn leads to a higher B.O.D. value. The algae that may negatively affect human health include cyanobacteria, which may contaminate drinking water and have been associated with gastroenteritis, skin irritation, and liver damage. Harmful marine algae produce toxins that can build up in shellfish. When ingested, these toxins may cause neurological or gastrointestinal problems or even death. Breathing the toxins may contribute to asthma attacks in susceptible individuals. In 2007, the Nassau County Health Department in northeastern

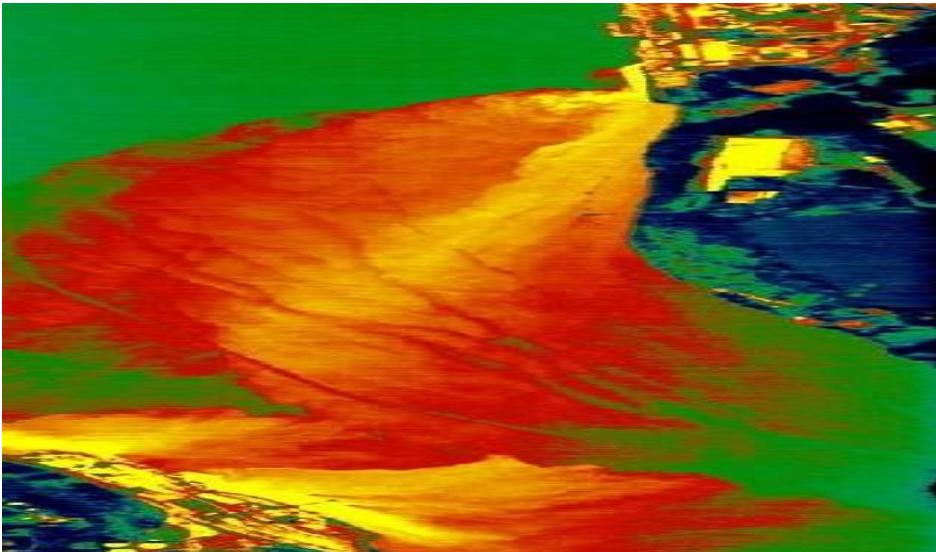


Figure 4.15. When Manufacturing plants release back heated water to the river or ocean, the water temperature rises sharply.
Source: U.S. EPA

Florida found that harmful algal blooms along the coastline were likely causes of respiratory illness in people working on a beach restoration.

Water temperature can affect the level of dissolved oxygen. When the temperature of water increases, oxygen becomes less soluble in water. With the constant flow of high temperature discharge from industries, there is a huge increase in toxins that are being regurgitated into the natural body of water. These toxins may contain chemicals or radiation that may have harsh impact on the local ecology and make them susceptible to various diseases.

For some species, a sudden increase in temperature can lead to instant death because many aquatic species can only survive within a certain range of temperature. The death of these species may have a negative effect on the food chain, causing the entire ecosystem to collapse. If the temperature increase continues over a long period, this may result in permanent changes in the species composition of the aquatic ecosystem.

4.5.7. Industrial Pollution

Industrial pollution is the release of wastes and pollutants generated by industrial activities into the natural environment including air, water, and land. The pollutants and wastes from industries encompass air emissions, deposit of used water into water resources, landfill disposal, and injection of toxic materials underground. Industrial pollution can adversely damage plants, kill animals, cause ecosystem imbalance, and degrade the quality of life.

Leading industries such as power plants, steel mills, sewage treatment plants, heating plants, and glass smelting among other production, processing and manufacturing companies are the contributors to industrial pollution. They release smoke, effluents, material wastes, toxic byproducts, contaminated residues, and chemical consumer products that eventually end up in the environment thereby causing pollution (Figure 4.16 and 4.17).



Figure 4.16. The releasing of smoke, material wastes and toxic byproducts that eventually end up in the environment thereby causing pollution. Source: Wikipedia



Figure 4.17. The releasing of wastes and pollutants generated by industrial activities into the natural environment. Source: Wikipedia

The health of humans and animals is a primary concern with industrial pollution. Air pollution that impacts the ground-level ozone can weaken respiratory systems, reduce lung function and increase respiratory inflammation in humans and animals alike. When exposed to air pollution, people may experience nausea, pulmonary congestion and chest pains.

Industrial pollution affects the growth of plants, crops and animals, thus reducing natural resources. When air quality is low due to industrial pollution, the ozone damages the forest ecosystems and crops. Bodies of water that are polluted by industrial chemicals can infect drinking water and also the natural habitats of aquatic species. Industrial pollution events such as fires,

radioactive material leaks and oil spills directly affect the level of pollution within water, the soil and the air. Natural habitats, such as forests and oceans, remain polluted, which has a negative impact on the species that live within the habitats.

4.5.8. Light Pollution

Light pollution occurs due to lengthened and excessive use of artificial lights, such that it results in the brightening of the skies at night (Figure 4.18 and 4.19). As a consequence, it upsets the activities and natural cycles of wildlife and also affects the welfare of humans. Light pollution is the brightening of the night sky inhibiting the visibility of stars and planets by the use of improper lighting of communities. Some examples of what causes light pollution:

- Street lamps that shine light in all directions, instead of with a hood to point light downward toward the street.
- Extra, unnecessary lights around the home.
- Cities that run lights all night long.

Light pollution uses more energy (by shining more light up instead of down, meaning you need brighter bulbs for the same amount of light), and may affect human health and our sleep cycles, and most importantly, corrupts our kids telescopes and their curiosity. Research suggests that artificial light at night can negatively affect human health, increasing risks for obesity, depression, sleep disorders, diabetes, breast cancer and more. Light pollution also has a major impact on the human mind. Nighttime light exposure creates inflammation in the brain's hippocampus, according to The Huffington Post. This frequently causes depression. Both depression and



Figure 4.18. Excessive lighting. Source: The Weather Network

excessive lighting can prevent people from obtaining sufficient sleep. Inadequate sleep causes auto accidents, heart disease, diabetes and high blood pressure. It promotes obesity by creating a false sense of hunger. A person's skin will age rapidly if he or she doesn't sleep enough.

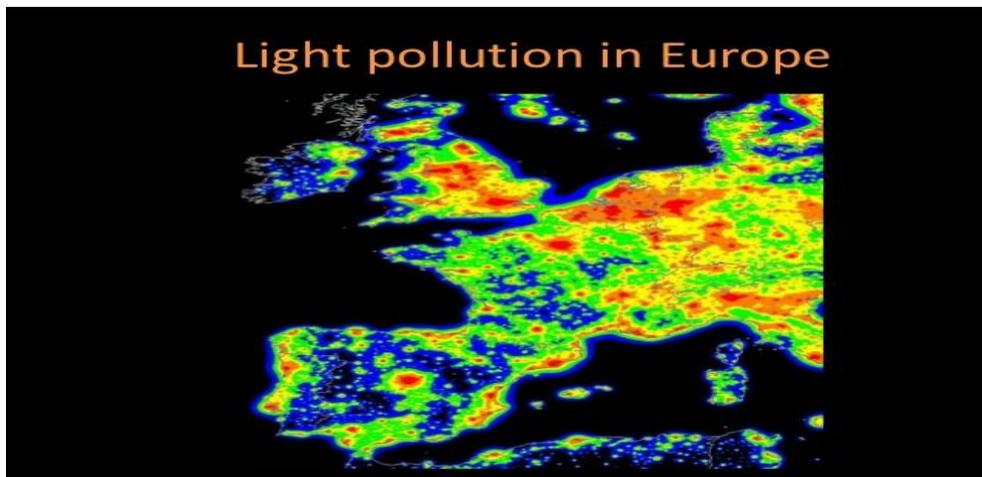


Figure 4.19. Light pollution in Europe. Source: The Weather Network

4.5.9. Radioactive Pollution

Radioactive pollution is increasing because of the increased use of radioactivity (Figure 4.20). It occurs mostly from the waste products that are left behind after the use of radioactive substances. Radioactive waste is usually the product of a nuclear process such as nuclear fission, which is extensively used in nuclear reactors, nuclear weapons and other nuclear fuel-cycles.

Radioactive wastes are disposed off without any precautionary measures to isolate the emissions which then contaminate the air, soil and water. Large amounts of radioactive waste is generated from nuclear reactors used in nuclear power plants and for many other purposes. It occurs during extraction and refining of the radioactive material. Nuclear accidents and nuclear explosions are the two worst man-made sources of radioactive pollution.



Figure 4.20. Large amount of radioactive waste is generated from nuclear reactors used in nuclear power plants.

Source: Universal Information

The major sources where radioactive wastes are generated and are responsible for radioactive pollution are as follows:

1. Uranium mining
2. Production of nuclear fuel
3. Nuclear power reactors

4. Use of radionuclides in industries for various application
5. Nuclear tests carried out by the Defense Personnel; and
6. Disposal of nuclear waste.

Radioactive pollution is not a constant or regular phenomenon and hence the duration and frequency of pollution will vary with time and conditions. Three major types of conditions exist.

1. Continuous pollution: This type of condition exists in Uranium mines, nuclear reactors and laboratories where the humans are under continuous exposure to radioactive contaminants.
2. Accidental pollution: This type of condition exists during accidental exposure to radiations by virtue of equipment failure, radiation leak, faulty protective equipment .
3. Occasional pollution: This condition exists during isolated experiment or test of nuclear substance.

Exposure to any type of radiation can prove harmful and even lethal. The two types of effects are genetic and nongenetic or body damage. In **genetic damage**, genes and chromosomes get altered. Its effect may become visible as deformations in the offspring (children or grandchildren). Alterations or breaks in the genetic material, that is DNA (deoxyribonucleic acid)- the molecule containing genetic information, is called **mutation**. In **nongenetic effects**, the harm is visible immediately in the form of birth defects, burns, some type of leukemia, miscarriages, tumors, cancer of one or more organs and fertility problems.

4.5.10. Plastic Pollution

Plastics accumulate in garbage dumps and landfills and are sully the world's oceans in ever-greater quantity (Figure 4.21, 4.22 and 4.23). Human exposure to plastic particles can occur via consumption of contaminated food and water, but also through the air.

Plastics pollution has a direct and deadly effect on wildlife. Thousands of seabirds and sea turtles, seals and other marine mammals are killed each year after ingesting plastic or getting entangled in it. Endangered wildlife like Hawaiian monk seals and Pacific loggerhead sea turtles are among nearly 300 species that eat and get caught in plastic litter.



Figure 4.21. Plastic Pollution. Source: Environment News South Africa

Different plastics spread throughout the ocean. As Styrofoam breaks into smaller parts, polystyrene components in it sink lower in the ocean, so that the pollutant spreads throughout the sea column. In fact, not only do the toxins in plastic effect the ocean, but acting like sponges, they soak up other toxins from outside sources before entering the ocean. As these chemicals are ingested by animals in the ocean, this is not good for humans. We as humans ingest contaminated fish and mammals.



Figure 4.22. Albatross killed by excessive plastic ingestion in Midway Islands (North Pacific). Source: Environmental Health Perspectives

There are different types of ways that plastic is dangerous for humans. Direct toxicity from plastics comes from lead, cadmium, and mercury. These toxins have also been found in many fish in the ocean, which is very dangerous for humans. Diethylhexyl phthalate (DEHP) contained in some plastics, is a toxic carcinogen. Other toxins in plastics are directly linked to cancers, birth defects, immune system problems, and childhood developmental issues.



Figure 4.23. Plastic pollution by numbers. Source: Plastic Oceans

Other types of toxic plastics are BPA or health-bisphenol-A, along with phthalates (mentioned above). Both of these are of great concern to human health. BPA is used in many things including plastic bottles and food packaging materials. Over time the polymer chains of BPA break down, and

can enter the human body in many ways from drinking contaminated water to eating a fish that is exposed to the broken down toxins. Specifically, BPA is a known chemical that interferes with human hormonal function.

4.6. World's Top 10 Toxic Pollution Problems

Every day synthetic, toxic chemicals are released into the environment. It affects our water, land and air. Water is our most vital resource but also our most threatened. Without water, there is no life. Our land is where we live and thrive upon. The air is what we breathe; what travels through the air is what we inhale. As it ultimately affects the future of our planet and us, it is considered to be a global threat at huge cost to the environment.

The toxic pollution problems impacts more to the people who live near to the sources of pollution. These pollutants may cause serious health effects such as birth defects, development disorders, respiratory problems, cancer and in some cases can lead to death. Apart from this, it can also have adverse effect on wildlife and environment.

Here is a list of the Top 10 Toxic Pollution Problems our world faces today, in no particular order:

4.6.1. Lead-Acid Battery Recycling

These rechargeable batteries are composed of lead plates and sulphuric acid in a plastic case. The battery recycling business is a very large industry, and although it aims to reduce the number of disposable batteries as solid waste, batteries contain a high number of toxic metals and chemicals like lead oxide that lead to the pollution of our water and contamination of soil.

4.6.2. Mercury and Lead Pollution from Mining

More than two million people globally are affected by mining and ore processing. These mining sites provide various minerals and metals to produce variety of products and minerals. The most hazardous chemicals that are found near these sites are lead, chromium, asbestos, arsenic, cadmium and mercury.

4.6.3. Coal Mining (Sulphur Dioxide and Mercury Pollution)

Though it's often overlooked, the high levels of mercury in the air are a serious threat to human health. Power plants fired by coal are located very close to large urban areas and cities in America. It can also travel exceptionally far (as in thousands of miles) through the air. Mercury is extremely damaging to human health as it severely damages the brain and nervous system when inhaled or made contact with. It is also estimated that a high percentage of pregnant woman in America are affected by high mercury levels that affect a foetus's brain development. All in all, mercury is one of the most deadly toxic pollutants in the air.

Not only is Sulphur Dioxide (SO₂) a substantial pollutant in our air and a direct result of coal power plants, it is also one of the causes of some serious health problems. It can be a root cause of lung cancer, asthma, emphysema, and bronchitis. As a result, thousands of people are tragically hospitalized or die each year. It is seriously toxic to human health. SO₂ originates primarily from fossil fuel combustion at power plants and coal power plants.

4.6.4. Artisanal Gold Mining (Mercury Pollution)

The production process of retrieving gold from mined ores releases more mercury than any other global sector. The mining process is usually done in the open air, putting people living nearby at risk either through contaminated water or soil. The vaporized mercury is a potent neurotoxic element that causes development disorders and affects the central nervous system.

4.6.5. Lead Smelting

Each year millions of people are affected by the toxic chemicals, primarily iron, limestone, pyrite and zinc that are released into the air by the dozens of lead smelting sites around the world. Lead smelting uses furnaces and other chemical agents to remove impurity from lead ores. Lead Smelting puts approximately 2.5 million people at risk at 70 polluted lead smelting sites worldwide, according to Blacksmith Institute.

4.6.6. Pesticides Pollution from Agriculture and Storage

Pesticides are substances necessary for agriculture to destroy targeted pests. An approximate 2 million metric tonnes of pesticides are used annually on fields. As a result, millions of tons of pesticides are dumped every year on our fields. Unfortunately, the health effects pesticides have on us are disastrous, from simple skin irritation to hurting to nervous system to even causing cancer.

Apart from this, stockpiles of old and outdated pesticides add to the trouble. Most of the farmers are illiterate and use expired products. An estimated six to nine million metric tonnes of such pesticides are improperly stored.

4.6.7. Arsenic in Ground Water

Arsenic in Ground Water is naturally occurring pollution problem that affects some 750,000 people, mostly in south Asia. Contaminated ground water is still used by many people which can lead to cancer, blood vessel damage, abnormal heart beat and some other ill effects.

4.6.8. Industrial Waste Water

Waste water is water that has been harmfully affected by outside influence and that flows from an open drain. Waste water may or may not be affected by any of the following, but certainly not limited to, batteries, smelting, toxins, organic particles, pathogens, methane and carbon dioxide. This water ends up in the environment where it is much more harmful to humans than irrigation water.

4.6.9. Chromium Pollution (Dye Industry)

Believe it or not, the dye industry actually contains numerous health hazards. Dye is used to add color to material, but the additions they have to pollution are more than noticeable. While chromium, which is used in dye, is critical to the human diet and generally speaking causes no damage to the human body, Cr IV Chromium is dangerous and highly toxic, enough to cause death in humans.

4.6.10. Chromium Pollution (Tanneries)

Chromium is primarily used to turn animal hides into leather for consumers, in places called tanneries, which are primarily centered in South-East Asia. Such tanneries are still operating with little control and produce daily 7.7 million litres of waste water and 88 million tons of solid waste. Again, Cr IV is dangerous and can cause health problems as in respiratory and heart failure and cancer in the brain and kidneys.

4.7. Industrial Pollution in Developing Countries

Industry in developing countries tends to be less efficient than in developed countries. This lack of efficiency is a chronic problem in developing economies, reflecting untrained human resources, the cost of importing equipment and technology, and the inevitable wastage that occurs when some parts of the economy are more developed than others.

This inefficiency is also based in part on the need to rely on outdated technologies which are freely available, do not require an expensive licence or do not cost as much to use. These technologies are often more polluting than the state-of-the-art technologies available to industry in developed countries. An example is the refrigeration industry, where the use of chlorofluorocarbons (CFCs) as refrigerant chemicals is much cheaper than the alternatives, despite the serious effects of these chemicals in depleting ozone from the upper atmosphere and thereby reducing the earth's shield from ultraviolet radiation; some countries have been very reluctant to agree to prohibit the use of CFCs because it would then be economically impossible for them to manufacture and purchase refrigerators. Technology transfer is the obvious solution, but companies in developed countries who developed or hold the license for such technologies are understandably reluctant to share them. They are reluctant because they have spent their own resources developing the technology, wish to retain the advantage they have in their own markets by controlling such technology, and may make their money from using or selling the technology only during the limited term of the patent.

Another problem faced by developing countries is lack of expertise in and awareness of the effects of pollution, monitoring methods and the technology of pollution control. There are relatively few experts in the field in

developing countries, in part because there are fewer jobs and a smaller market for their services even though the need may actually be greater. Because the market for pollution control equipment and services may be small, this expertise and technology may have to be imported, adding to the costs. General recognition of the problem by managers and supervisors in industry may be lacking or very low. Even when an engineer, manager or supervisor in industry realizes that an operation is polluting, it may be difficult to persuade others in the company, their bosses or the owners that there is a problem that must be solved.

Industry in most developing countries competes at the low end of international markets, meaning that it produces products that are competitive on the basis of price and not quality or special features. Few developing countries specialize in making very fine grades of steel for surgical instruments and sophisticated machinery, for example. They manufacture lesser grades of steel for construction and manufacturing because the market is much larger, the technical expertise required to produce it is less, and they can compete on the basis of price as long as the quality is good enough to be acceptable. Pollution control reduces the price advantage by increasing the apparent costs of production without increasing output or sales. The central problem in developing countries is how to balance this economic reality against the need to protect their citizens, the integrity of their environment, and their future, realizing that after development the costs will be even higher and the damage may be permanent.

4.7.1. Air Pollution

The problem of air pollution has grown steadily since the Industrial Revolution began 300 years ago. Four major factors have exacerbated air pollution: growing industrialization; increasing traffic; rapid economic development; and higher levels of energy consumption. The available information shows that the WHO guidelines for the major air pollutants are regularly exceeded in many major urban centres. Although progress has been made in controlling air pollution problems in many industrialized countries over the last two decades, air quality—particularly in the larger cities in the developing world—is worsening. Of major concern are the adverse health effects of ambient air pollutants in many urban areas, where levels are sufficiently high to contribute to increased mortality and morbidity, deficiency

in pulmonary function and cardiovascular and neurobehavioural effects (Romieu, Weizenfeld and Finkelman 1990; WHO/UNEP 1992). Indoor air pollution due to domestic combustion products is also a major issue in developing countries (WHO 1992b), but it is not part of this review, which considers only the sources, dispersion and health effects of outdoor air pollution, and includes a case study of the situation in Mexico.

4.7.1.1. Source of Air Pollutants

The most common air pollutants in urban environments include sulphur dioxide (SO₂), suspended particulate matter (SPM), the nitrogen oxides (NO and NO₂, collectively termed NO_x), ozone (O₃), carbon monoxide (CO) and lead (Pb). Combustion of fossil fuels in stationary sources leads to the production of SO₂, NO_x and particulates, including sulphate and nitrate aerosols formed in the atmosphere following gas to particle conversion. Petrol-fuelled motor vehicles are the principal sources of NO_x, CO and Pb, whereas diesel-fuelled engines emit significant quantities of particulates, SO₂ and NO_x. Ozone, a photochemical oxidant and the main constituent of photochemical smog, is not emitted directly from combustion sources but is formed in the lower atmosphere from NO_x and volatile organic compounds (VOCs) in the presence of sunlight (UNEP 1991b). Table. 4.1 presents the major sources of outdoor air pollutants.

Table 4.1. Major sources of outdoor air pollutants (UNEP 1991b)

Pollutants	Sources
Sulphur oxides	Coal and oil combustion, smelters
Suspended particulate	Combustion products (fuel, biomass), tobacco smoke matter
Nitrogen oxides	Fuel and gas combustion
Carbon monoxide	Incomplete petrol and gas combustion
Ozone	Photochemical reaction
Lead	Petrol combustion, coal combustion, producing batteries, cables, solder, paint
Organic substances	Petrochemical solvents, vaporization of unburnt fuels

4.7.1.2. Dispersion and Transport of Air Pollutants

The two major influences on the dispersion and transport of air pollutant emissions are meteorology (including microclimate effects such as “heat islands”) and topography in relation to the population distribution. Many cities are surrounded by hills which may act as a downwind barrier, trapping pollution. Thermal inversions contribute to a particulate problem in temperate and cold climates. Under normal dispersion conditions, hot pollutant gases rise as they come into contact with colder air masses with increasing altitude. However, under certain circumstances the temperature may increase with altitude, and an inversion layer forms, trapping pollutants close to the emission source and delaying their diffusion. Long-range transport of air pollution from large urban areas may have national and regional impacts. Oxides of nitrogen and sulphur may contribute to acid deposition at great distances from the emission source. Ozone concentrations are often elevated downwind of urban areas due to the time lag involved in photochemical processes (UNEP 1991b).

4.7.1.3. Health Effects of Air Pollutants

Pollutants and their derivatives can cause adverse effects by interacting with and impairing molecules crucial to the biochemical or physiological processes of the human body. Three factors influence the risk of toxic injury related to these substances: their chemical and physical properties, the dose of the material that reaches the critical tissue sites and the responsiveness of these sites to the substance. The adverse health effects of air pollutants may also vary across population groups; in particular, the young and the elderly may be especially susceptible to deleterious effects. Persons with asthma or other pre-existing respiratory or cardiac diseases may experience aggravated symptoms upon exposure (WHO 1987).

Sulphur Dioxide and Particulate Matter .During the first half of the twentieth century, episodes of marked air stagnation resulted in excess mortality in areas where fossil-fuel combustion produced very high levels of SO₂ and SMP. Studies of long-term health effects have also related the annual mean concentrations of SO₂ and SMP to mortality and morbidity. Recent epidemiological studies have suggested an adverse effect of inhalable particulate levels (PM₁₀) at relatively low concentrations (not exceeding the standard guidelines) and have shown a dose-response relationship between

exposure to PM₁₀ and respiratory mortality and morbidity (Dockery and Pope 1994; Pope, Bates and Razienne 1995; Bascom et al. 1996) as shown in table 4.2.

Table 4.2. Summary of short-term exposure-response relationship of PM₁₀ with different health effects indicators (Dockery and Pope 1994; Pope, Bates and Razienne 1995; Bascom et al. 1996)

Health effect	% changes for each 10mg/m ³ increase in PM ₁₀	% changes for each 10 mg/m ³ increase in PM ₁₀
	Mean	Range
Total Mortality	1.0	0.5-1.5
Cardiovascular	1.4	0.8-1.8
Respiratory	3.4	1.5-3.7
Hospital admission for respiratory condition	1.1	0.8-3.4
Emergency visits for respiratory conditions	1.0	0.5-4
Symptom exacerbations among asthmatics	3.0	1.1-11.5
Changes in peak expiratory flow	0.08	0.04-0.25

Nitrogen Oxides. Some epidemiological studies have reported adverse health effects of NO₂ including increased incidence and severity of respiratory infections and increase in respiratory symptoms, especially with long-term exposure. Worsening of the clinical status of persons with asthma, chronic obstructive pulmonary disease and other chronic respiratory conditions has also been described. However, in other studies, investigators have not observed adverse effects of NO₂ on respiratory functions (WHO/ECOTOX 1992; Bascom et al. 1996).

Photochemical Oxidants and Ozone. The health effects of photochemical oxidants exposure cannot be attributed only to oxidants, because photochemical smog typically consists of O₃, NO₂, acid and sulphate

and other reactive agents. These pollutants may have additive or synergistic effects on human health, but O₃ appears to be the most biologically active.

Table 4.3. Health outcomes associated with changes in peak daily ambient ozone concentration in epidemiological studies (Bascom et al. 1996).

Health outcome	Changes in 1-h O ₃ (µg/m ³)	Changes in 8-h O ₃ (µg/m ³)
Symptom exacerbations among healthy children and adults or asthmatics-normal activity		
25% increase	200	100
50% increase	400	200
100% increase	800	300
Hospital admissions for respiratory conditions ^a		
5%	30	25
10%	60	50
20%	120	100

^a Given the high degree of correlation between the 1-h and 8-h O₃ concentrations in field studies, an improvement in health risk associated with decreasing 1- or 8-h O₃ levels should be almost identical.

Source: WHO 1995.

Health effects of ozone exposure include decreased pulmonary function (including increased airway resistance, reduced air flow, decreased lung volume) due to airway constriction, respiratory symptoms (cough, wheezing, shortness of breath, chest pains), eye, nose and throat irritation, and disruption of activities (such as athletic performance) due to less oxygen availability (WHO/ECOTOX 1992). Table 4.3 summarizes the major acute health effects of ozone (WHO 1990a, 1995). Epidemiological studies have suggested a dose-response relationship between exposure to increasing ozone

levels and the severity of respiratory symptoms and the decrement in respiratory functions (Bascom et al. 1996)

Carbon Monoxide. The main effect of CO is to decrease oxygen transport to the tissues through the formation of carboxyhaemoglobin (COHb). With increasing levels of COHb in blood, the following health effects can be observed: cardiovascular effects in subjects with previous angina pectoris (3 to 5%); impairment of vigilance tasks (>5%); headache and dizziness ($\geq 10\%$); fibrinolysis and death (WHO 1987).

Lead. Lead exposure principally affects haem biosynthesis, but also may act on the nervous system and other systems such as the cardiovascular system (blood pressure). Infants and young children less than five years old are particularly sensitive to lead exposure because of its effect on neurological development at blood lead levels close to 10 $\mu\text{g}/\text{dl}$ (CDC 1991).

Several epidemiological studies have investigated the effect of air pollution, especially ozone exposure, on the health of the population of Mexico City. Ecological studies have shown an increase in mortality with respect to exposure to fine particulates (Borja-Arburto et al. 1995) and an increase in emergency visits for asthma among children (Romieu et al. 1994). Studies of the adverse effect of ozone exposure conducted among healthy children have shown an increase in school absenteeism due to respiratory illnesses (Romieu et al. 1992), and a decrease in lung function after both acute and subacute exposure (Castillejos et al. 1992, 1995). Studies conducted among asthmatic children have shown an increase in respiratory symptoms and a decrease in peak expiratory flow rate after exposure to ozone (Romieu et al. 1994) and to fine particulate levels (Romieu et al. in press). Although, it seems clear that acute exposure to ozone and particulates is associated with adverse health effects in the population of Mexico City, there is a need to evaluate the chronic effect of such exposure, in particular given the high levels of photo-oxidants observed in Mexico City and the ineffectiveness of control measures.

Case study: Air pollution in Mexico City

The metropolitan area of Mexico City (MAMC) is situated in the Mexican Basin at a mean altitude of 2,240 metres. The basin covers 2,500 square kilometres and is surrounded by mountains, two of which are over 5,000

metres high. The total population was estimated at 17 million in 1990. Due to the particular geographic characteristics and the light winds, ventilation is poor with a high frequency of thermic inversions, especially during the winter.

More than 30,000 industries in the MAMC and the three million motor vehicles circulating daily are responsible for 44% of the total energy consumption. Since 1986, air pollution has been monitored, including SO₂, NO_x, CO, O₃, particulate matter and non-methane hydrocarbon (HCNM). The main air pollutant problems are related to ozone, especially in the southwest part of the city (Romieu et al. 1991). In 1992 the Mexican norm for ozone (110 ppb one-hour maximum) was exceeded in the southwest part more than 1,000 hours and reached a maximum of 400 ppb. Particulate levels are high in the northeast section of the city, close to the industrial park. In 1992, the annual average of inhalable particulate (PM₁₀) was 140 µg/m³. Since 1990, important control measures have been taken by the government to decrease air pollution, including a programme that prohibits use of cars one day a week depending on their terminating licence plate number, the closure of one of the most polluting refineries located in Mexico City, and the introduction of unleaded fuel. These measures have led to a decrease in various air pollutants, mainly SO₂, particulate matter, NO₂, CO and lead. However the ozone level remains a major problem (see figure 4.24, figure 4.25 and figure 4.26).

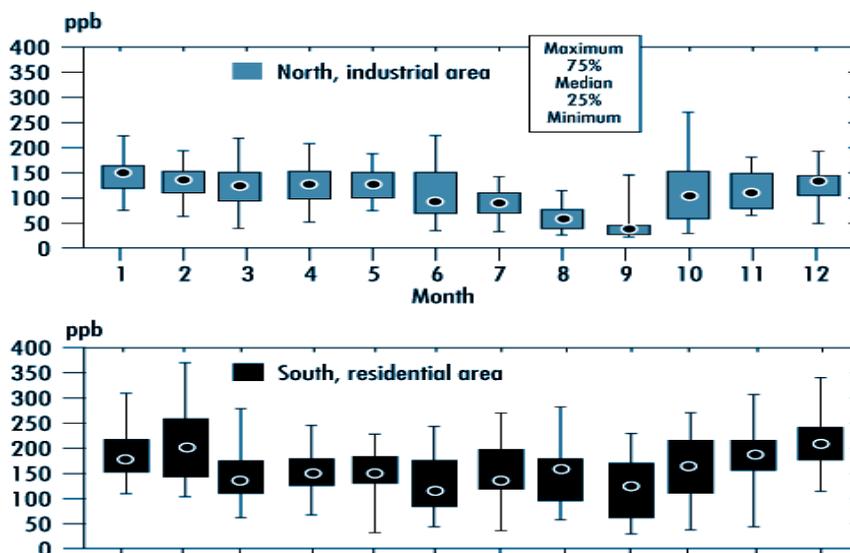


Figure 4.24. Ozone levels in two zones of Mexico City. One-hour daily maximum by month, 1994 (Borja-Arburto et al. 1995)

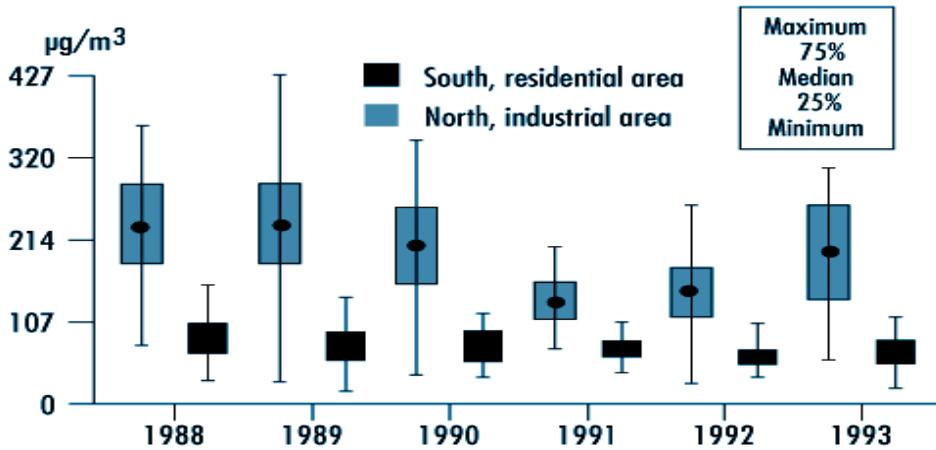


Figure 4.25. Particulates (PM₁₀) in two zones of Mexico City, 1988-1993 (Borja-Arburto et al. 1995)

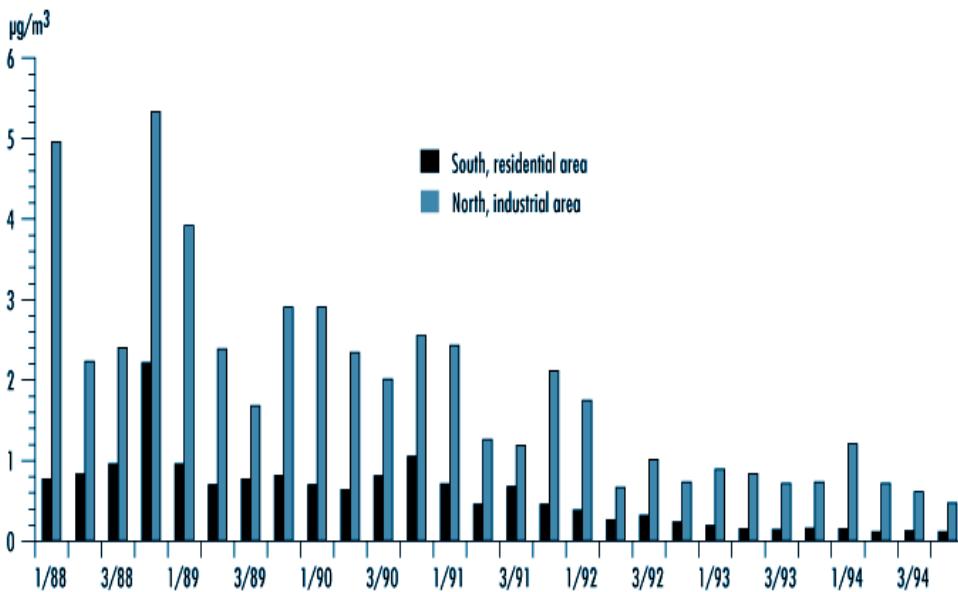


Figure 4.26. Air lead levels in two zones of Mexico City, 1988-1994 (Borja-Arburto et al. 1995)

4.7.2. Water Pollution

Since the middle of the twentieth century, and concurrent with the onset of accelerated industrial growth, various types of water pollution problems have occurred in rapid succession.

4.7.2.1. Types and Sources of Pollution

There are a large number of microbial agents, elements and compounds which may cause water pollution. They can be classified as: microbiological organisms, biodegradable organic compounds, suspended matter, nitrates, salts, heavy metals, nutrients and organic micro pollutants.

Microbiological organisms. Microbiological organisms are common in freshwater bodies polluted particularly by discharges of untreated domestic wastewater. These microbial agents include pathogenic bacteria, viruses, helminths, protozoa and several more complex multicellular organisms that can cause gastro-intestinal illness. Other organisms are more opportunistic in nature, infecting susceptible individuals through body contact with contaminated water or by inhalation of poor quality water droplets in aerosols of various origins.

Biodegradable organic compounds. Organic substances of either natural origin (allochthonous terrestrial detritus or autochthonous debris of aquatic plants) or from anthropogenic sources (domestic, agricultural and some industrial wastes) are decomposed by aerobic microbes as the river continues its course. The consequence is a lowering of the oxygen level downstream of the wastewater discharge, impairing the quality of the water and the survival of the aquatic biota, particularly of high-quality fish.

Particulate matter. Particulate matter is a major carrier of organic and inorganic pollutants. Most toxic heavy metals, organic pollutants, pathogens and nutrients, such as phosphorus, are found in suspended matter. An appreciable amount of the biodegradable organic material responsible for consumption of dissolved oxygen from rivers is also found in suspended particles. Particulate matter comes from urbanization and road construction, deforestation, mining operations, dredging operations in rivers, natural sources which are linked to continental erosion, or natural catastrophic events.

Coarser particles are deposited on river beds, in reservoirs, in the flood plain and in wetlands and lakes.

Nitrates. The concentration of nitrates in unpolluted surface waters ranges from less than 0.1 to one milligrams per litre (expressed as nitrogen), so nitrate levels in excess of 1 mg/l indicate anthropogenic influences such as discharge of municipal wastes and urban and agricultural run-off. Atmospheric precipitation is also an important source of nitrate and ammonia to river basins, particularly in areas not affected by direct pollution sources—for example, some tropical regions. High concentrations of nitrate in drinking water may lead to acute toxicity in bottle-fed infants during their first months of life, or in the elderly, a phenomenon called methaemoglobinaemia.

Salts. Water salinization may be caused by natural conditions, such as geochemical interaction of waters with salty soils or by anthropogenic activities, including irrigated agriculture, sea water intrusion due to excessive pumping of groundwaters in islands and coastal areas, disposal of industrial wastes and of oilfield brines, highway de-icing, landfill leaches and leaking sewers.

While hampering beneficial uses, particularly for irrigation of sensitive crops or for drinking, salinity in itself may not, at even quite high levels, be directly harmful to health, but the indirect effects can be dramatic. The loss of fertile agricultural land and reduced crop yields caused by waterlogging and soil salinization of irrigated areas destroy the livelihood of whole communities and cause hardships in the form of food shortages.

Heavy metals. Heavy metals such as lead, cadmium and mercury are micro-pollutants and of special interest as they have health and environmental significance due to their persistence, high toxicity and bio-accumulation characteristics.

There are basically five sources of heavy metals contributing to water pollution: geological weathering, which provides the background level; industrial processing of ores and metals; the use of metal and metal compounds, such as chromium salts in tanneries, copper compounds in agriculture, and tetraethyl lead as an anti-knock agent in gasoline; leaching of heavy metals from domestic wastes and solid waste dumps; and heavy metals in human and animal excretions, particularly zinc. Metals released into the air

from automobiles, fuel burning and industrial process emissions may settle on land and ultimately run off to surface waters.

Nutrients. Eutrophication is defined as the enrichment of waters with plant nutrients, primarily phosphorus and nitrogen, leading to enhanced plant growth (both algae and macrophytes) which results in visible algae blooms, floating algal or macrophyte mats, benthic algae and submerged macrophyte agglomerations. When decaying, this plant material leads to the depletion of the oxygen reserves of water bodies, which, in turn, causes an array of secondary problems such as fish mortality and liberation of corrosive gases and other undesirable substances, such as carbonic gas, methane, hydrogen sulphide, organoleptic substances (causing taste and odour), toxins and so on.

The source of phosphorus and nitrogen compounds is primarily untreated domestic wastewater, but other sources such as drainage of artificially fertilized agricultural land, surface run-off from intensive livestock farming and some industrial wastewaters can also substantially increase the trophic level of lakes and reservoirs, particularly in tropical developing countries.

The main problems associated with eutrophication of lakes, reservoirs and impoundments are: oxygen depletion of the bottom layer of lakes and reservoirs; water quality impairment, leading to treatment difficulties, particularly for the removal of taste- and odour-causing substances; recreational impairment, increased health hazards to bathers and unsightliness; fisheries impairment due to fish mortality and the development of undesirable and low-quality fish stocks; ageing and reducing the holding capacity of lakes and reservoirs by silting; and increase of corrosion problems in pipes and other structures.

Organic micropollutants. Organic micropollutants can be classified in groups of chemical products on the basis of how they are used and consequently how they are dispersed in the environment:

Pesticides are substances, generally synthetic, that are deliberately introduced into the environment to protect crops or control disease vectors. They are found in various distinct families, such as organochloride insecticides, organophosphate insecticides, herbicides of the plant hormone type, triazines, substituted ureas and others.

Materials for widespread household and industrial use comprise volatile organic substances used as extraction solvents, solvents for degreasing metals and dry-cleaning clothes, and propellants for use in aerosol containers. This group also includes halogenated derivatives of methane, ethane and ethylene. As they are widely used their rates of dispersion in the environment, compared with the amounts produced, are generally high. The group also contains the polycyclic aromatic hydrocarbons, whose presence in the environment results from the extraction, transport and refining of petroleum products and the dispersion of combustion products resulting from their use (petrol and heating oil).

Materials used essentially in industry include substances which are direct or intermediate agents of chemical synthesis, such as carbon tetrachloride for synthesizing freons; vinyl chloride for polymerizing PVC; and chlorinated derivatives of benzene, naphthalene, phenol and aniline for manufacturing dyestuffs. The group also contains finished products used in closed systems, such as heat-exchange fluids and dielectrics.

Organic micropollutants are generated from point and diffuse sources, either urban or rural. The largest part originates in major industrial activities such as petrol refining, coal mining, organic synthesis and the manufacture of synthetic products, the iron and steel industries, the textile industry and the wood and pulp industry. Effluents from pesticides factories may contain considerable quantities of these manufactured products. A significant proportion of organic pollutants are discharged into the aquatic environment as run-off from urban surfaces; and in agricultural areas, pesticides applied to crops may reach surface waters through rainwater run-off and artificial or natural drainage. Also, accidental discharges have led to severe ecological damage and temporary closure of water supplies.

4.7.2.2. Urban Pollution

Owing to this continuously expanding, aggressive and multi-faceted pollution scenario, the problem of maintaining the quality of water resources has become acute, particularly in the more urbanized areas of the developing world. Maintaining water quality is hampered by two factors: failure to enforce pollution control at the main sources, especially industries, and inadequacy of sanitation systems and of garbage collection and disposal

(WHO 1992b). See some examples of water pollution in different cities in developing countries.

Karachi (Pakistan)

The Lyari river, which runs through Karachi, Pakistan's largest industrial city, is an open drain from both the chemical and the microbiological point of view, a mixture of raw sewage and untreated industrial effluents. Most industrial effluents come from an industrial estate with some 300 major industries and almost three times as many small units. Three-fifths of the units are textile mills. Most other industries in Karachi also discharge untreated effluents into the nearest water body.

Alexandria (Egypt)

Industries in Alexandria account for around 40% of all Egypt's industrial output, and most discharge untreated liquid wastes into the sea or into Lake Maryut. In the past decade, fish production in Lake Maryut declined by some 80% because of the direct discharge of industrial and domestic effluents. The lake has also ceased to be a prime recreational site because of its poor condition. Similar environmental degradation is taking place along the seafront as a result of the discharge of untreated wastewater from poorly located outfalls.

Shanghai (China)

Some 3.4 million cubic metres of industrial and domestic waste pour mostly into the Suzhou Creek and the Huangpu River, which flows through the heart of the city. These have become the main (open) sewers for the city. Most of the waste is industrial, since few houses possess flush toilets. The Huangpu has essentially been dead since 1980. In all, less than 5% of the city's wastewater is treated. The normally high water table also means that a variety of toxins from industrial plants and local rivers find their way into groundwater and contaminate wells, which also contribute to the city water supply.

São Paulo (Brazil)

The Tiete River, as it passes through Greater São Paulo, one of the world's largest urban agglomerations, receives 300 tonnes of effluents each day from 1,200 industries located in the region. Lead, cadmium and other heavy metals are among the main pollutants. It also receives 900 tonnes of sewage each day, of which only 12.5% is treated by the five sewage treatment stations located in the area.

Source: Based on Hardoy and Satterthwaite 1989.

4.8. Common Indoor Air Pollutants

When we think of polluted air, the first images that come to our minds are often big factories pumping out smelly clouds or old cars puffing out grey exhaust. However, indoor air pollutants can be even more dangerous than outdoor air pollution. The first step to keeping your home and family safe is learning more about the problem. This article will help by covering 7 common indoor air pollutants.

4.8.1. Carbon monoxide (CO)

Carbon monoxide is an odorless, invisible gas. It is produced by the incomplete combustion of fossil fuels.

Risks: Carbon monoxide stops your body from using the oxygen it needs to work normally. You may experience tiredness, headaches, dizziness, nausea, confusion, and a fast heart rate. If the concentration of carbon monoxide is high enough, you could die.

What you can do: The Environmental Assistance and Protection Department of Forsyth County, North Carolina, recommends having your heating systems checked by a professional yearly. Make sure that combustion appliances, such as heaters, have been installed correctly. Do not use combustion appliances without vents inside and never use a gas stove to heat your house.

4.8.2. Radon

Radon is an odorless, colorless gas that is found everywhere in low levels. It is made naturally as the uranium in the Earth breaks down.

Risks: Being exposed to elevated levels of radon increases your risk of getting lung cancer.

What you can do: You should screen your home for elevated radon levels with a kit or have it tested by a qualified professional. Radon screening kits are easy to use and can be purchased online with your credit card. For example, the company Radon Environmental, Inc., offers a radon homeowner's testing kit for \$35 via radon-environmental.com. You can pay using a Visa, Mastercard, or American Express card. If your home does have elevated levels of radon, you must hire a qualified professional to remove it.

4.8.3. Nitrogen dioxide (NO₂)

Nitrogen dioxide (NO₂) is a common oxide of nitrogen. It is a toxic and corrosive gas. Please note that nitrogen dioxide (NO₂) is different from *nitrous* oxide (N₂O), an oxide of nitrogen that is medically useful when administered by trained professionals, such as dentists.

Risks: Nitrogen dioxide (NO₂) irritates the throat, eyes, nose, and respiratory tract. Exposure to very high doses of NO₂, such as at the site of a building fire, can lead to pulmonary edema (potentially fatal liquid build-up in the lungs) or lung injury. Moderate exposure can lead to acute or chronic bronchitis. Low-level exposure can impair lung function for people who are already at risk, such as asthmatics, people with chronic obstructive lung disease, and children.

What you can do: According to the U.S. Environmental Protection Agency, it's important to ensure that combustion appliances, such as heaters, are installed correctly, used as directed, and kept in good condition. Make sure the air from these appliances can flow outdoors. Do not idle your car in the garage.

4.8.4. Secondhand smoke

Secondhand smoke, also called environmental tobacco smoke, comes from incompletely burned tobacco products. According to the Environmental Assistance and Protection Department of Forsyth County, secondhand smoke contains over 4,700 chemical ingredients.

Risks: In the short term, exposure to secondhand smoke can cause eye, nose, and throat irritation. In the long-term, it can cause many of the same health problems as smoking, like wheezing, pneumonia, bronchitis, and lung cancer. Asthma attacks may be triggered by secondhand smoke exposure.

What you can do: Do not smoke cigarettes, cigars, or pipe tobacco inside your home and do not allow others to do so.

4.8.5. Lead particles

Lead is a natural, soft metal that is very toxic if consumed. Lead was widely used in house paint until it was banned in 1978. Lead particles and dust can become airborne, leading to dangerous indoor air pollution.

Risks: Exposure to lead can damage the brain, nervous system, kidneys, and red blood cells. If children are exposed, they may develop short attention spans, behavioral problems, lower IQ levels, and delayed growth.

What you can do: If you live in a home painted before 1978, the Environmental Assistance and Protection Department of Forsyth County advises keeping play areas clean, mopping floors frequently, and using damp cloths to wipe window ledges and flat areas often. Keep kids away from chipped or peeled paint, clean their toys often, and make sure they wash their hands before eating.

4.8.6. Asbestos

Asbestos is the name used for a group of minerals found naturally all over the world. The U.S. Environmental Protection Agency declared asbestos unsafe in 1971, listing it as a hazardous air pollutant. Although asbestos is not

hazardous when intact, disturbing asbestos fibers causes them to become airborne, where they could potentially enter the lungs.

Risks: In the long-term, exposure to asbestos can lead to various lung disorders, including lung cancer and asbestosis. Asbestosis is an inflammatory condition of the lungs that causes coughing, trouble breathing, and permanent lung damage.

What you can do: If products in your home contain asbestos, but are in good condition, the Environmental Assistance and Protection Department of Forsyth County recommends just keeping them in good condition. Otherwise, have them removed by a trained professional.

4.8.7. Mold

Molds are types of fungi that grow indoors and outdoors. Some types of mold are harmless, while others are dangerous.

Risks: Mold can trigger an allergic reaction in some people. Symptoms can include nasal stuffiness, eye or throat irritation, swelling, coughing or wheezing, headaches, or skin irritation. Severe reactions can lead to fever and trouble breathing. Mold can also trigger asthma attacks.

What you can do: According to the Environmental Assistance and Protection Department of Forsyth County, the key to fighting mold is keeping moisture and humidity levels in check. Fix leaks and clean up spills ASAP. Make sure appliances that create moisture are vented. Keep the bathroom fan on or the window open when taking a shower.

4.9. Top 10 Countries Killing the Planet

The study, evaluating the Relative Environmental Impact of Countries, uses seven indicators of environmental degradation: natural forest loss, habitat conversion, marine captures, fertilizer use, water pollution, carbon emissions and species threat. Unlike existing rankings, this study deliberately avoided human health and economic data, and instead focused on environmental impact only. Other variables—bush meat harvest, coral reef habitat quality, sea grass loss, freshwater habitat degradation, illegal

fishing, invertebrate threat patterns, and some forms of greenhouse gas emission—were excluded due to a lack of country-specific data.

Two rankings were created: a “proportional” environmental impact ranking, where impact is measured against total resource availability, and an “absolute” environmental impact ranking which measures total environmental degradation at a global scale. Listed here are the top ten worst offending countries for absolute environmental impact, those that are just doing the most damage, regardless of per capita calculations.

4.9.1. Brazil

In all seven categories considered for the report, Brazil ranked within the top ten for all but marine capture: **1st place for natural forest loss, 3rd place for natural habitat conversion, 3rd place for fertilizer use, 4th place for threatened species, 4th place for CO₂ emissions, and 8th place for water pollution.** What’s to account for these areas of intense environmental impact? A large portion of deforestation in Brazil can be attributed to the expansive Amazon rain forest land clearing for pastureland by commercial and speculative interests, misguided government policies, inappropriate World Bank projects, and commercial exploitation of forest resources. Soy and cocoa crops, as well as cattle ranching, have had a far-reaching effect.

In the Atlantic forests of Brazil, some of the world’s most diverse ecosystems have been converted to fast growing plantations (mostly non-native eucalyptus) for paper pulp.

4.9.2. USA

You’d think with all of the smarts and resources this country has, it would rank a bit better than Number 2—afraid not. Although it did rank a respectable 211 for natural habitat conversion—that honor is pretty much negated by the country’s abysmal ratings in other areas. Ringing in at **1st place for fertilizer use**, this country’s excessive application of nitrogen, phosphorous and potassium (NPK) fertilizers can result in the leaching of these chemicals into water bodies and remove, alter or destroy natural habitats. The USA also ranks in **1st place for CO₂ emissions, 2nd place for**

water pollution, 3rd place for marine captures, and 9th place for threatened species. Not feeling all that proud to be American at the moment.

4.9.3. China

China's coastal waters are increasingly polluted by everything from oil to pesticides to sewage, helping China earn its **1st place ranking for water pollution**. In China, 20 million people lack access to clean drinking water; over 70 percent of lakes and rivers are polluted; and major pollution incidents happen on a near daily basis—the World Health Organization recently estimated that nearly 100,000 people die annually from water pollution-related illnesses.

China isn't doing much better in terms of overfishing—they take **1st place for marine capture**. Add to that **2nd place for CO₂ emissions** and **6th place for threatened species**, and we can see how China takes the bronze for most environmental impact. Chinese environmental protection agencies lack sufficient authority, financial resources and manpower. When there are conflicts between environmental protection and economic development, the former often loses to the latter.

4.9.4. Indonesia

According to Global Forest Watch, Indonesia was still densely forested as recently as 1950—yet 40 percent of the forests existing in 1950 were cleared in the following 50 years. In round numbers, forest cover fell from 162 million ha to 98 million ha. For this, Indonesia ranks **2nd in natural forest loss**. Indonesia is ranked **3rd for CO₂ emissions, 6th for marine capture, 6th for fertilizer use, and 7th for water pollution**.

4.9.5. Japan

Japan ranks **4th for marine capture**. By 2004, the number of adult Atlantic bluefin tuna capable of spawning had dropped to roughly 19 percent of the 1975 level in Japan, which has a quarter of the world supply of the five big species of tuna: bluefin, southern bluefin, bigeye, yellowfin and albacore. Japan ranks **5th for both natural habitat conversion and water pollution, and 6th for CO₂ emissions**.

4.9.6. Mexico

Mexico holds more species of plants and animals than just about any other country: 450 mammals (Brazil, which is more than twice Mexico's size has only 394 mammals); about 1,000 birds, 693 reptiles; 285 amphibians, and more than 2,000 fish. As of the mid-1990s, many species were known to be already threatened: 64 mammals, 36 birds, 18 reptiles, 3 amphibians, and about 85 fish. Mexico did not join the Convention on International Trade in Endangered Species (CITES), the chief international agreement to stop trade in threatened and endangered plants and animals, in effect since 1975, until 1991, the last Latin American nation to do so. It is perhaps because of these factors that Mexico ranks **1st for threatened species**. The country ranks **9th for natural forest loss**.

4.9.7. India

According to the Wall Street Journal, in an effort to boost food production, win farmer votes and encourage the domestic fertilizer industry, the government has increased its subsidy of urea fertilizer over the years, and now pays about half of the domestic industry's cost of production. The overuse of urea is so degrading the soil that yields on some crops are falling—landing India is **2nd place for environmental impact due to fertilizer use**. India ranks **3rd for water pollution** as increasing competition for water among various sectors, including agriculture, industry, domestic, drinking, energy generation and others, is causing this precious natural resource to dry up—while increasing pollution is also leading to the destruction of the habitat of wildlife that lives in waterways. India comes in **8th for another three areas: threatened species, marine capture and CO₂ emissions**.

4.9.8. Russia

Less than half of Russia's population has access to safe drinking water. While water pollution from industrial sources has diminished because of the decline in manufacturing, municipal wastes increasingly threaten key water supply sources, and nuclear contamination poses immense problems for key water sources as well—landing Russia in **4th place for worst water pollution**. Russia ranks **5th in worst CO₂ emissions**—air quality is almost as poor as water quality, with over 200 cities often exceeding Russian pollution limits. The country ranks **7th for marine capture**.

4.9.9. Australia

About 11.5 percent of the the total land area of Australia is protected, which leaves a lot left (although much of it is arid desert) for unbridled usage, which is how the country ranks **7th worst in habitat conversion**. It also ranks **9th for fertilizer use**, and **10th for natural forest loss**.

4.9.10. Peru

Although Peru hardly seems capable of the harmful environmental impact that larger industrialized countries are capable of, the South America country ranks number 10 overall of countries creating negative environmental impact. Of 179 countries, Peru ranks **2nd for marine capture** and **7th for threatened species**. Over fishing and illegal trade of endangered species seem to be the culprit: Convention on International Trade in Endangered Species (CITIES) lists ten animal species as critically endangered (like the short-tailed chinchilla) the last step before extinction, 28 as endangered, and 99 as vulnerable in Peru.

The proportional index, which takes into consideration the impact as proportional to the resources available in the country, ranks these as the top ten countries creating the most negative environmental impact: Singapore, Korea, Qatar, Kuwait, Japan, Thailand, Bahrain, Malaysia, Philippines and Netherlands. According to the study from which both of these rankings were taken, “continued degradation of nature despite decades of warning, coupled with the burgeoning human population (currently estimated at nearly 7 billion and projected to reach 9 to 10 billion by 2050), suggest that human quality of life could decline substantially in the near future. Increasing competition for resources could therefore lead to heightened civil strife and more frequent wars. Continued environmental degradation demands that countries needing solutions be identified urgently so that they can be assisted in environmental conservation and restoration.”

4.10. Pollution is the Leading Cause of Death in the Developing World

Pollution is the leading cause of death in low- and middle-income countries, according to a report from the Global Alliance on Health and Pollution (GAHP), an organization whose members include the World Bank,

Columbia University's Earth Institute, and various United Nations' bodies and national governments.

In 2012, pollution – in the form of contaminated soil, water, and both indoor and outdoor air – was responsible for 8.4 million deaths in developing countries. That is almost three times more deaths than those caused by malaria, HIV/AIDS and tuberculosis combined: Malaria claimed 600,000 lives in 2012, HIV/AIDS caused 1.5 million deaths and tuberculosis killed 900,000 individuals. And the Ebola outbreak that had American legislators shaking in their suits, while ignoring more pressing national issues? Last year, fewer than 8,000 individuals died from the Ebola virus, the report says.

Worldwide, pollution is responsible for 8.9 million deaths – or 1 in 7 deaths globally. But 94 percent of the burden of pollution falls on lower-income countries “who are the least equipped to deal with the problem,” according to the report. Of the 8.4 million pollution-caused deaths in developing countries, air pollution was the leading offender, the report finds. Forty-four percent of pollution-caused deaths resulted from household air pollution, such as cook stoves that contaminate the air, and 38 percent were caused by ambient air pollution, including particulates from power plants, cars and trucks. The contamination of soil and food from heavy metals released by industry and mining accounted for 10 percent of pollution-caused deaths, while local water systems, polluted by sewage and industrial waste, made up 8 percent. Cancers, strokes, and heart and respiratory diseases are just some of the fatal health conditions that can result from exposure to pollution, the report says. GAHP's findings are based on **World Health Organization data on global deaths from polluted air and water**, as well as GAHP's own analysis of deaths related to toxic chemical and industrial wastes in low-income countries.

4.11. Conclusion

Here are the solutions to Environmental Pollution,

- Gas emission pollution is being mitigated in a variety of ways with car emission control, **electric and hybrid vehicles and public transportation systems**. Not all major cities have successful implementation and decent public transportation in place, but the world is working on this issue constantly and we have managed to reduce

emissions profoundly over the last decade. There is much catching up to do.

- The cost of radioactive power plants is becoming apparent and the days of coal power plants are nearly dead. Radiation is a serious issue. Radioactive leakage from power plants and nuclear testing have already contaminated oceanic life to such a degree that it will take hundreds of years to return to normal. More radiation solutions are in the works with various **ecologically friendly power technologies** being built every day.
- **Solar power is a fantastic solution.** Now that solar radiation is at a climactic peak, we can reap power from the sun using solar panel systems. These range from home systems to larger scale systems powering entire communities and cities.
- **Wind power is coming into play.** This may not seem like much at first, but when you get about 100 feet off the ground, there is a great deal of wind up there. By building wind turbines to harvest natural wind energy, electricity is produced. Wind turbine power and solar power are both powerful forces against fossil fuel power and radioactive power. The one problem here is power companies. They want to stay with radioactive power plants because they actually can't be removed. It has become the crusades of many individuals and small corporations to make the switch and there are plenty of people following this as populations cry out for help
- **Electromagnetic radiation (ER) reduction.** Once major manufacturers of computers and electronic devices realized the blatant potential for huge ER emissions directly into the eyes and brains of users, they started to implement hardware protocols to minimize risks and reduce ER production significantly. Newer devices are in the lead to knock this problem out and, fortunately, this is working.

Also, the Environmental Protection Agency (EPA) is well aware of all leaks and tricks industries are using to dump wastes. This agency now has extremely strict protocols and testing procedures implemented against such facilities so populations are not affected. Additionally, the EPA is measuring air pollution and implementing regulatory procedures for vehicle emissions.

They also monitor pollen issues and, with the help of the Centers for Disease Control (CDC), they implement solutions to reduce pollen in the air.

Dropping pollen counts is a major focus for EPA and CDC activities. Asthma and other allergic conditions are flooding medical care facilities and pharmaceutical companies with serious public health problems. The response has been swift and various methods to control emissions and reduce pollen counts are in the works. Children and elderly people are at the highest risk for environmental pollution related health problems. The good news is we are directly on the horizon to cut down the causes and risks while providing practical health solutions for the general public throughout the world.

5. Environmental Disasters

5.1. Introduction.

A disaster is a sudden, calamitous event that seriously disrupts the functioning of a community or society and causes human, material, and economic or environmental losses that exceed the community's or society's ability to cope using its own resources. Though often caused by nature, disasters can have human origins. In contemporary academia, disasters are seen as the consequence of inappropriately managed risk. These risks are the product of a combination of both hazards and vulnerability. Hazards that strike in areas with low vulnerability will never become disasters, as in the case of uninhabited regions. Developing countries suffer the greatest costs when a disaster hits. More than 95 percent of all deaths caused by hazards occur in developing countries, and losses due to natural hazards are 20 times greater (as a percentage of GDP) in developing countries than in industrialized countries.

Researchers have been studying disasters for more than a century, and for more than forty years disaster research. The studies reflect a common opinion when they argue that all disasters can be seen as being human-made, their reasoning being that human actions before the strike of the hazard can prevent it developing into a disaster. All disasters are hence the result of human failure to introduce appropriate disaster management measures. Hazards are routinely divided into natural or human-made, although complex disasters, where there is no single root cause, are more common in developing countries. A specific disaster may spawn a secondary disaster that increases the impact. A classic example is an earthquake that causes a tsunami, resulting in coastal flooding.

Natural Types of Disasters. A natural hazard is a natural process or phenomenon that may cause loss of life, injury or other health impacts, property damage, loss of livelihoods and services, social and economic disruption, or environmental damage. Various phenomena like earthquakes, landslides, volcanic eruptions, floods, hurricanes, tornadoes, blizzards, tsunamis, and cyclones are all natural hazards that kill thousands of people and destroy billions of dollars of habitat and property each year. However, the rapid growth of the world's population and its increased concentration often in hazardous environments has escalated both the frequency and severity of

disasters. With the tropical climate and unstable land forms, coupled with deforestation, unplanned growth proliferation, non-engineered constructions which make the disaster-prone areas more vulnerable, tardy communication, and poor or no budgetary allocation for disaster prevention, developing countries suffer more or less chronically from natural disasters. Asia tops the list of casualties caused by natural hazards.

Natural Types of Disasters

- | | |
|--|--|
| <ul style="list-style-type: none"> • Agricultural diseases & pests • Damaging Winds • Drought and water shortage • Earthquakes • Emergency diseases (pandemic influenza) • Extreme heat • Floods and flash floods • Hail | <ul style="list-style-type: none"> • Hurricanes and tropical storms • Landslides & debris flow • Thunderstorms and lightning • Tornadoes • Tsunamis • Wildfire • Winter and ice storms • Sinkholes |
|--|--|

Hurricanes and tropical storms are among the most powerful natural disasters because of their size and destructive potential. Tornadoes are relatively brief but violent, potentially causing winds in excess of 200 mph. Both earthquakes and tornadoes strike suddenly without warning.

Flooding is the most common of natural hazards, and requires an understanding of the natural systems of our environment, including floodplains and the frequency of flooding events. Wildfires are more prevalent in the event of a drought. Disasters impacting food supply can be extremely costly; American officials say that a food contamination scare similar to the one that hit the Belgian poultry industry in the 1990's could jeopardize U.S. agricultural exports in excess of \$140 billion.

Human-Made and Technological Types of Disasters. Human-instigated disasters are the consequence of technological hazards. Examples include stampedes, fires, transport accidents, industrial accidents, oil spills

and nuclear explosions/radiation. War and deliberate attacks may also be put in this category. As with natural hazards, man-made hazards are events that have not happened—for instance, terrorism. Man-made disasters are examples of specific cases where man-made hazards have become reality in an event. Airplane crashes and terrorist attacks are examples of man-made disasters: they cause pollution, kill people, and damage property. This example is the September 11 attacks in 2001 at the World Trade Center in New York.

Human-Made and Technological Types of Disasters

- | | |
|---|--|
| <ul style="list-style-type: none"> • Hazardous materials • Power service disruption & blackout • Nuclear power plant and nuclear blast • Radiological emergencies | <ul style="list-style-type: none"> • Chemical threat and biological weapons • Cyber attacks • Explosion • Civil unrest |
|---|--|

High-risk targets include military and civilian government facilities, international airports, large cities and high-profile landmarks. Cyber-terrorism involves attacks against computers and networks done to intimidate or coerce a government or its people for political or social objectives.

5.2. Different Type of Environmental Disasters

Environmental disasters are the realization of hazards to serious impacts, damages and losses, initiating in some or the other environmental systems or resources. These hazards are related to various environmental processes and systems including geo-morphological, topographical/landscape, atmospheric/ climatic, geo-chemical, edaphic, hydrological, hydrodynamic, features, along human interventions including industrial/urban, etc.

5.2.1. Agricultural Disasters

- Mismanagement and shrinking of the **Aral Sea**
- **Salinity in Australia**
- Salinization of the Fertile Crescent
- The **Dust Bowl** in Canada and the United States (1934–1939)
- The Great sparrow campaign; sparrows were eliminated from Chinese farms, which caused locusts to swarm the farms and contributed to a famine which killed 38 million people.
- **Africanized bees**, known colloquially as "killer bees"
- **"Dirty dairying"** in New Zealand
- **Salton Sea** California, U.S.

5.2.2. Industrial Environmental Disasters

- Spring Valley, a neighborhood in Washington, D.C. which was used as a chemical weapons testing ground during World War I.
- **Minamata disease** - mercury poisoning in Japan (1950s & 1960s)
- **Ontario Minamata disease** in Canada
- **Itai-itai disease**, due to cadmium poisoning in Japan
- **Love Canal** toxic waste site
- **Seveso disaster** (1976), chemical plant explosion, caused highest known exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in residential populations
- **Times Beach, Missouri** (1983) the town was completely evacuated due to a dioxin contamination
- **Bhopal disaster** (December 3, 1984, India), leak of methyl isocyanate that took place in 1984 resulted in more than 22,000 deaths.
- **Sandoz chemical spill** into the Rhine river (1986)
- United States Environmental Protection Agency Superfund sites in the United States
- AZF Explosion at a Toulouse chemical factory (2001)
- 2005 Jilin chemical plant explosions
- The Sydney Tar Ponds and Coke Ovens sites in the city of Sydney, Nova Scotia, Canada, known as the largest toxic waste site in North America.
- Release of lead dust into Esperance Harbour.

- Release of cyanide, heavy metals and acid into the Alamosa River, Colorado from the Summitville mine, causing the death of all aquatic life 17 miles downstream.
- Release of 20,000 gallons of lethal chemicals (metam sodium, tradename Vapam) into the Upper Sacramento River near Dunsmuir, causing the death of all aquatic life within a 38-mile radius.
- Release of CFCs resulting in ozone depletion
- Release of sulfur dioxide after a fire at the Al-Mishraq plant in Iraq
- The Phillips Disasters
- Health issues on the Aamjiwnaang First Nation due to chemical factories
- Environmental issues with the Three Gorges Dam
- Kingston Fossil Plant coal fly ash slurry spill
- The Great Smog in London in 1952

5.2.3. Mining Disasters

- 1947 Centralia mine disaster, Illinois
- Centralia mine fire, Pennsylvania, 1962
- Phosphate mining in Nauru
- Phosphate mining in St. Pierre Island
- Talvivaara gypsum pond leak, Finland, 2012
- Mount Polley mine disaster, British Columbia, 2014
- Bento Rodrigues dam disaster, Samarco mine tailings dam failure, which spread for over 2 states, Minas Gerais and Espirito Santo, all the way to the Atlantic sea. Brazil, 2015
- The "**Door to Hell**" (also known as the **Gate to Hell**, the **Crater of Fire**, **Darvaza Crater**), a natural gas field in Derweze, Turkmenistan continuously burning.

5.2.4. Oil Industry

- Environmental issues in the Niger Delta relating to the oil industry
- Lago Agrio oil field issues
- Exxon Valdez oil spill
- Arctic Refuge drilling controversy
- Deepwater Horizon oil spill
- Sidoarjo mud flow triggered by Lapindo Brantas gas exploration in 2006; East Java, Indonesia

- Leaded gasoline introduced 1920s; phased out globally by 2012.
- Vila Parisi (Brazil)

5.2.5. Nuclear

- **Chernobyl disaster** in 1986 in Chernobyl, Ukraine, "killed at least 4056 people and damaged almost \$7 billion of property". Radioactive fallout from the accident concentrated near Belarus, Ukraine and Russia and at least 350,000 people were forcibly resettled away from these areas. After the accident, "traces of radioactive deposits unique to Chernobyl were found in nearly every country in the northern hemisphere".
- **Fukushima Daiichi nuclear disaster:** Following an earthquake, tsunami, and failure of cooling systems at Fukushima I Nuclear Power Plant and issues concerning other nuclear facilities in Japan on March 11, 2011, a nuclear emergency was declared. This was the first time a nuclear emergency had been declared in Japan, and 140,000 residents within 20 km of the plant were evacuated. Explosions and a fire have resulted in dangerous levels of radiation, sparking a stock market collapse and panic-buying in supermarkets.
- **Mayak** nuclear waste storage tank explosion, (Chelyabinsk, Soviet Union, 29 September 1957), 200+ people died and 270,000 people were exposed to dangerous radiation levels. Over thirty small communities had been removed from Soviet maps between 1958 and 1991.
- **Windscale fire**, United Kingdom, October 8, 1957. Fire ignites plutonium piles and contaminates surrounding dairy farms.
- Soviet submarine K-431 accident, August 10, 1985 (10 people died and 49 suffered radiation injuries).
- Soviet submarine K-19 accident, July 4, 1961. (8 deaths and more than 30 people were over-exposed to radiation).
- Nuclear testing at Moruroa and Fangataufa in the Pacific Ocean
- Fallout from the Castle Bravo nuclear test at Bikini Atoll in the Marshall Islands
- The health of Downwinders
- Atomic bombings of Hiroshima and Nagasaki Within the first two to four months of the bombings, the acute effects killed 90,000–166,000 people in Hiroshima and 60,000–80,000 in Nagasaki, with roughly half of the deaths in each city occurring on the first day.

- Hanford Nuclear, 1986 – The U.S. government declassifies 19,000 pages of documents indicating that between 1946 and 1986, the Hanford Site near Richland, Washington, released thousands of US gallons of radioactive liquids. Radioactive waste was both released into the air and flowed into the Columbia River (which flows to the Pacific Ocean). In 2014, the Hanford legacy continues with billions of dollars spent annually in a seemingly endless cleanup of leaking underground

5.2.6. Air

- The Donora Smog of 1948 in Donora, Pennsylvania in the United States
- The Great Smog of 1952, which killed 4,000 Londoners
- The 1983 Melbourne dust storm
- The 1997 Southeast Asian haze
- The 2005 Malaysian haze
- The 2006 Southeast Asian haze
- Yokkaichi asthma in Japan
- Health problems due to the Jinkanpo Atsugi Incinerator in Japan
- Kuwaiti oil fires

5.2.7. Land

- The Dust Bowl of Canada and the United States
- Contaminated soils in Mapua, New Zealand due to the operation of an agricultural chemicals factory
- Basin F, a disposal site in the United States for contaminated liquid wastes from the chemical manufacturing operations of the Army and its lessee Shell Chemical Company
- Exide lead contamination in southeast Los Angeles County, California, United States, from a battery recycling plant that emitted lead, arsenic and other dangerous pollutants
- 2006 Côte d'Ivoire toxic waste dump
- Nigeria gully erosion crisis

5.2.8. Water

- Sandoz chemical spill, severely polluting the Rhine in 1986
- Selenium poisoning of wildlife due to farm runoff used to create Kesterson National Wildlife Refuge, and the artificial wetland
- The Jiyeh Power Station oil spill in the Mediterranean region
- Effects of polluted water in the Berkeley Pit in the United States
- Ignition and conflagration (13 times from 1868 to 1969) of the Cuyahoga River in Ohio, United States
- Cheakamus River derailment which polluted a river with caustic soda
- Draining and development of the Everglades
- Loss of Louisiana Wetlands due to Mississippi River levees, saltwater intrusion through manmade channels, timber harvesting, subsidence, and hurricane damage.
- Lake Okeechobee is heavily polluted and during extreme events releases large volumes of polluted water into the St. Lucie River estuary and the Caloosahatchee River estuary.

5.2.9. Marine

- Coral bleaching
- Gulf of Mexico Dead Zone due to high-nutrient fertilizer runoff from the Midwest that is drained through the Mississippi River.
- The artificial Osborne Reef off the coast of Fort Lauderdale, Florida in the United States
- Dumping of conventional and chemical munitions in Beaufort's Dyke, a sea trench between Northern Ireland and Scotland
- Marine debris
- Environmental threats to the Great Barrier Reef
- Nurdles, plastic pellet typically under 5mm in diameter
- Friendly Floatees
- The Great Pacific Garbage Patch
- Minamata disease, mercury poisoning in Japan
- Mercury in fish

5.3. Twenty five Biggest Man made Environmental Disasters in History

Our environment has been the victim of all sorts of attacks. Some of these attacks are natural such as hurricanes and earthquakes. However, there

are attacks that are unnatural and man-made such as wars, explosions, chemical spills, etc. These attacks usually carry with them heavy price tags as property and lives are damaged beyond full compensation and repair. To see these effects firsthand (or as close to first hand as possible) I present 25 biggest environmental disasters in history list for your personal edification.

5.3.1. The Great Smog of London, 1952

Thousands died and a hundred thousand fell ill because of a blanket of smog that covered London for 5 days in 1952 (Figure 5.1). Cold weather, combined with windless conditions collected airborne pollutants from the use of coal to form a thick layer of smog over the city. Government medical reports in the following weeks, however, estimated that up until 8 December, 4,000 people had died as a direct result of the smog and 100,000 more were made ill by the smog's effects on the human respiratory tract. More research suggested that the total number of fatalities was considerably greater, about 12,000. The Great Smog was known to be the worst air-pollution event in the history of the United Kingdom, and the most significant in terms of its effect on environmental research, government regulation, and public awareness of the relationship between air quality and health. It led to several changes in practices and regulations, including the Clean Air Act 1956.



Figure 5.1. The Great Smog of London, 1952. Source: BBC.com

5.3.2. The Chernobyl Nuclear Explosion, 1986

On April 26, 1986, one of the reactors at the Chernobyl power plant in Ukraine exploded, resulting in a nuclear meltdown that sent massive amounts of radiation into the atmosphere, reportedly more than the fallout from Hiroshima and Nagasaki (Figure 5.2 and 5.3). That radiation drifted westward, across what was then Soviet Russia, toward Europe. Since then, thousands of surviving kids have been suffered blindness remains off-limits.

Thirty-one deaths were directly attributed to the accident, all among the reactor staff and emergency workers. An UNSCEAR report placed the total number of confirmed deaths from radiation at 64 as of 2008. The Chernobyl Forum predicted that the eventual death toll could reach 4,000 among those exposed to the highest levels of radiation (200,000 emergency workers, 116,000 evacuees and 270,000 residents of the most contaminated areas); this figure was a total causal death toll prediction, combining the deaths of approximately 50 emergency workers who died soon after the accident from acute radiation syndrome, nine children who had died of thyroid cancer and a future predicted total of 3940 deaths from radiation-induced cancer and leukaemia.



Figure 5.2. A picture of a nuclear blast. Chernobyl witnesses reported seeing 'blue twinkles' in the wreckage of Reactor No.4. Chernobyl nuclear explosion, 1986. Source: redflagnews.com

More than 4,000 cancer deaths were attributed to the widespread radioactive contamination, which hit Belarus the hardest. Elevated atmospheric radiation levels were found as far away as the British Isles.



**Figure 5.3. The Chernobyl nuclear reactor after the disaster.
Reactor No.4 (centre). Source: en.wikipedia.org**

Chernobyl was known as the worst nuclear power plant incident in history. Cancers, deformities and other long term illnesses were the scars of not only human inhabitants but of animals as well.

5.3.3. The Bhopal Disaster, 1984

Around midnight on Dec. 2, 1984, an accident at a Union Carbide pesticide plant in Bhopal, India, resulted in 45 tons of poisonous methyl isocyanate escaping from the facility. Thousands died within hours. More followed over subsequent months — about 15,000 in all. In total, about half a million people were affected in some way. Many of those who survived suffered blindness (Figure 5.4), organ failure and other awful bodily malfunctions.



Figure 5.4. Survivors suffering blindness after Bhopal Disaster, 1984
Source: Boston.com

A shockingly high number of children in the area have been born with all manner of birth defects. In 1989, Union Carbide paid out about half a billion dollars to victims, an amount the afflicted say is not nearly enough to deal with the decades-long consequences. Bhopal remains the worst industrial disaster ever.

5.3.4. The Shrinking of the Aral Sea, 1989 - 2009

Dubbed as "one of the planet's worst environmental disasters", 10% of Aral sea's 68,000 square kilometers has disappeared due to the diversion of rivers for irrigation. That percentage that was once a part of the fourth largest inland body of water is now a plain of highly saline soil with depleting marine life.

In early April 2010, United Nations Secretary-General Ban Ki-moon traveled to Central Asia, where he laid eyes upon a "graveyard of ships" — rusting fishing trawlers and other vessels stranded in a desert that stretched for miles in all directions. It was the Aral Sea ... or what used to be the Aral Sea. Situated between Uzbekistan and Kazakhstan, the Aral was once the fourth largest lake on earth, as big as Ireland. Since the 1960s, however, when Soviet irrigation projects diverted several of its source waterways, the Aral has shrunk 90% (Figure 5.5). What was once a vibrant, fish-stocked lake is now a

massive desert that produces salt and sandstorms that kill plant life and have negative effects on human and animal health for hundreds of miles around.

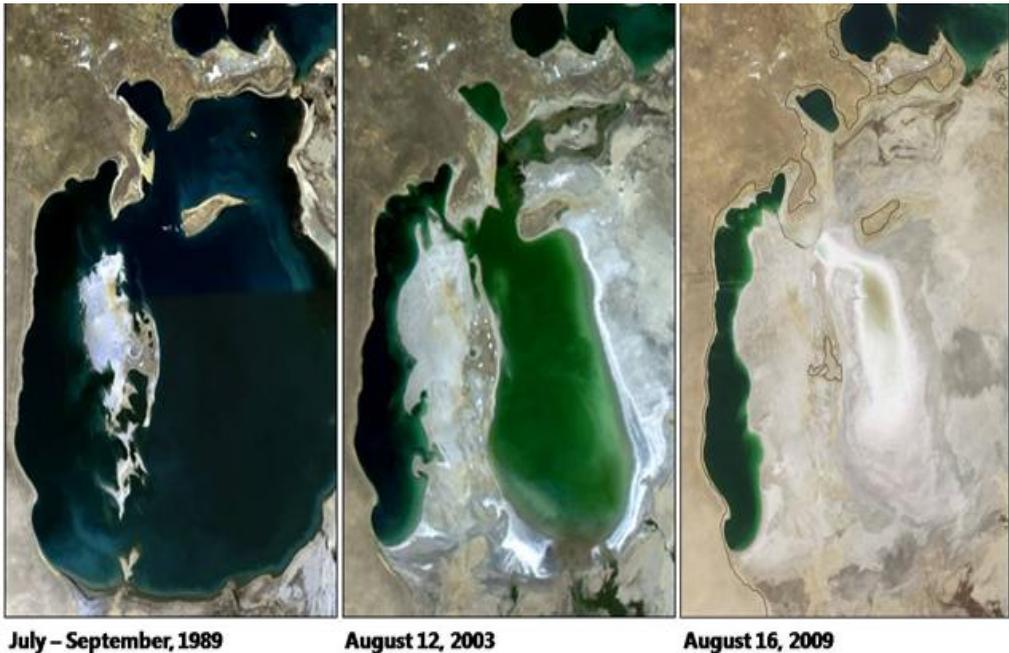


Figure 5.5. Comparisons of the shrinking of Aral Sea (1989, 2003 and 2009).
Source: www.columbia.edu

5.3.5. Baia Mare Cyanide Spill, 2000

After the Chernobyl incident in Russia, this cyanide spill in Baiba Mare, Romania is aptly called the worst environmental disaster in Europe. On January 30, 2000, 100,000 cubic meters of cyanide-contaminated water leaked out from a dam, spewing out 100 tons of cyanide and incredible amount of fish and aquatic plants were killed and up to 100 people were hospitalized after eating contaminated fish (Figure 5.6 and 5.7).



Figure 5.6. Cyanide spill in Baiba Mare, Romania, 2000.
Source: Emaze



Figure 5.7. Fishermen hauling dead fish ashore following the Baia Mare cyanide spill in 2000. Source: MTI Archives

Due to a speedy response by the Romanian government, no casualties were reported (although some children were sickened from eating fish from the contaminated rivers). Test of river water and sediments after the accident, found cyanide levels between 300 and 700 times above pollution standards. Copper and zinc concentrations also exceeded by many times “safe” pollution thresholds

Exposed metal ore tailings, when dry, can produce toxic dust. To reduce this problem, and to extract any remaining gold, the process of **gold cyanidation** is employed, which uses cyanide to extract any gold traces from mineral ores. The by-product of this controversial process is vast quantities of cyanide-laced water and heavy metal waste. Cyanide is a highly lethal chemical. There are thousands of mining waste dumps (many without dam containment) scattered all over the world, most situated near streams, rivers and large bodies of water.

5.3.6. E-waste in Guiyu, China, 2005

Guiyu, in Guangdong Province, China, is an agglomerate of four adjoined villages widely perceived as the largest electronic waste (e-waste) site in the world. In 2005 there were 60,000 e-waste workers in Guiyu who processed the more than 100 truckloads that were transported to the 52 square kilometre area every day. As a result 88% of the children in the area suffer from lead poisoning and there is more than the average rate of miscarriage. The constant movement into and processing of e-wastes in the area leading to the harmful and toxic environment and living conditions, coupled with inadequate facilities, have led to the Guiyu town being nicknamed the "electronic graveyard of the world" (Figure 5.8).



**Figure 5. 8. Chinese town of Guiyu built its economy on recycling waste from overseas. Largest electronic waste (e-waste) site in the world.
Source: faranaz.com**

Many of the primitive recycling operations in Guiyu are toxic and dangerous to workers' health with 80% of children suffering from lead poisoning (Figure 5.9). Above-average miscarriage rates are also reported in the region. Workers use their bare hands to crack open electronics to strip away any parts that can be reused—including chips and valuable metals, such as gold, silver, etc. Workers also "cook" circuit boards to remove chips and solders, burn wires and other plastics to liberate metals such as copper; use highly corrosive and dangerous acid baths along the riverbanks to extract gold from the microchips; and sweep printer toner out of cartridges.

Children are exposed to the dioxin-laden ash as the smoke billows around Guiyu, and finally settles on the area. The soil has been saturated with lead, chromium, tin, and other heavy metals. Discarded electronics lie in pools of toxins that leach into the groundwater, making the water undrinkable to the extent that water must be trucked in from elsewhere. Lead levels in the river sediment are double European safety levels, according to the Basel Action Network. Lead in the blood of Guiyu's children is 54% higher on average than that of children in the nearby town of Chendian.



Figure 5.9. Primitive recycling operations in Guiyu are toxic and dangerous to workers' health with 80% of children suffering from lead poisoning. Source: Greenpeace USA

5.3.7. The Seveso Disaster, 1976

In July of 1976, a pesticide plant in the town of Seveso, Italy, unintentionally released a large plume of a dioxin-based gas-vapor (TCDD, or tetrachlorodibenzoparadioxin), blanketing some 37,000 residents with ultra-high levels of the chemical which is known to be carcinogenic even in small dosages. Shortly thereafter 3,300 animals died and many more were put down in order to prevent the spread of contamination into the food chain. Children were hospitalized with skin inflammation and nearly 500 people were found to have skin disease known as chloracne (Figure 5.10). Hundreds were evacuated and thousands treated for dioxin poisoning (the most obvious sign of which was the horrible, pustular form of acne that dioxin exposure causes). Tens of thousands of animals were subsequently slaughtered to prevent TCDD from entering the food chain.

HAZARDOUS EFFECTS



Figure 5.10. Hazardous effect of the Seveso Disaster. 1976. Source: Toxipedia

The fear of birth defects caused thirty-four women to obtain therapeutic abortions, with the permission of the Catholic Church. One estimate said that another 120 women in the area may have had abortions without the permission of the church. The fear of birth defects has proven justified. Babies born with birth defects in the Seveso area in 1975, the year before the contamination, numbered three. In 1978, they totaled fifty-three. On July 25, 1976, over two weeks after the dioxin contamination, the Seveso regional authorities finally ordered the evacuation of 739 residents from the 253 acres (102 hectares) immediately adjacent to the plant.

5.3.8. Methylmercury poisoning (Minamata Disease), 1956

Minamata disease was first discovered in Minamata city in Kumamoto prefecture, Japan, in 1956. Symptoms included convulsions, slurred speech, loss of motor functions and uncontrollable limb movements. In 1968 it was determined that the Chisso Corporation, a petrochemical plant, had been dumping a toxic mercury compound into the bay. In total, the company dumped 27 tons of poison into the water. Minamata disease is methylmercury (MeHg) poisoning that occurred in humans who ingested fish and shellfish contaminated by MeHg discharged in waste water from a chemical plant (Chisso Co. Ltd.). The marine products in Minamata Bay displayed high

levels of Hg contamination (5.61 to 35.7 ppm). The fish passed on the Mercury built up in their bodies to the fish-consuming residents nearby. Scientists estimate that the **Biomagnification of the Mercury** was as great as a millionfold. Initial symptoms included uncoordinated movement and numbness of the lips and extremities, followed by constricted vision. The effect upon the infants was even more severe as many were born with a wide range of disabilities (Figure 5.11, 5.12 and 5.13).



Figure 5.11. A severe case of Minamata Disease. Source: Japan Focus

Three years later, an investigation concluded that the affliction was a result of industrial poisoning of Minamata Bay by the Chisso Corp., which had long been one of the port town's biggest employers. As a result of wastewater pollution by the plastic manufacturer, large amounts of mercury and other heavy metals found their way into the fish and shellfish that comprised a large part of the local diet. Thousands of residents have slowly suffered over the decades and died from the disease.



Figure 5.12. Shinobu Sakamoto a sufferer of fetal Minamata disease speaks at a news conference. Source: Getty Images

Residents of the town began to experience symptoms as well—including tremors, brain damage and vision problems. Repercussions included death, insanity, birth defects (like mangled limbs) and deformities, including vision and hearing. As of March 2001, 2,265 victims had been officially recognised as having Minamata disease (1,784 of whom had died) and over 10,000 had received financial compensation from Chisso Corporation.



Figure 5.13. Minamata disease suffering patients got loss of motor functions and uncontrollable limb movements. Source: Time Magazin

5.3.9. Gulf of Mexico Dead Zone disaster

The most infamous hypoxic zone in the United States, the Gulf of Mexico's 'dead zone' is the dumping area for nitrogen and phosphorus, just two of the many high nutrient run offs. These substances come from the Mississippi River, which is the drainage area for almost half of the continental America. Nutrient overloading and algal blooms lead to eutrophication, which has been shown to reduce benthic biomass and biodiversity. Hypoxic water supports fewer organisms and has been linked to massive fish kills in the Black Sea and Gulf of Mexico (Figure 5.14). To blame are fertilizers, pesticides and nitrogen-rich livestock waste that have seeped into the water from farms along the Mississippi River.

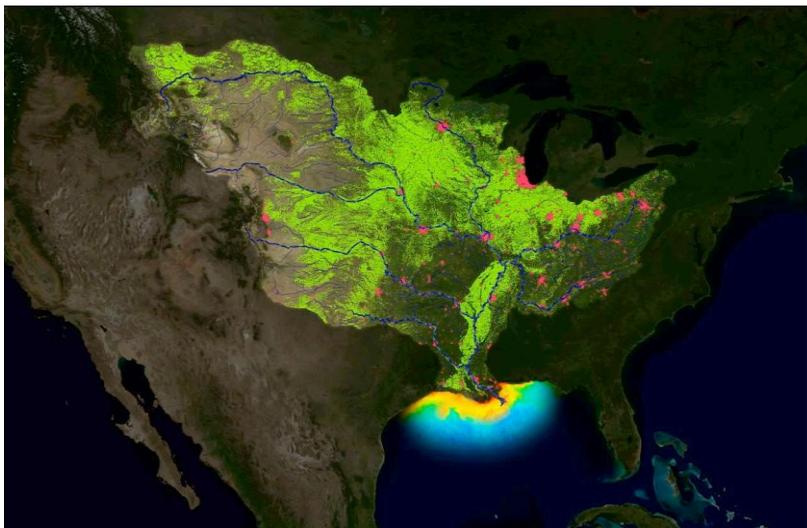


Figure 5.14. The Gulf of Mexico “Dead Zone” Source: People's World

Scientists have determined this year's Gulf of Mexico “dead zone,” an area of low oxygen that can kill fish and marine life, is 8,776 square miles, an area about the size of New Jersey. It is the largest measured since dead zone mapping began there in 1985 (Figure 5.15).

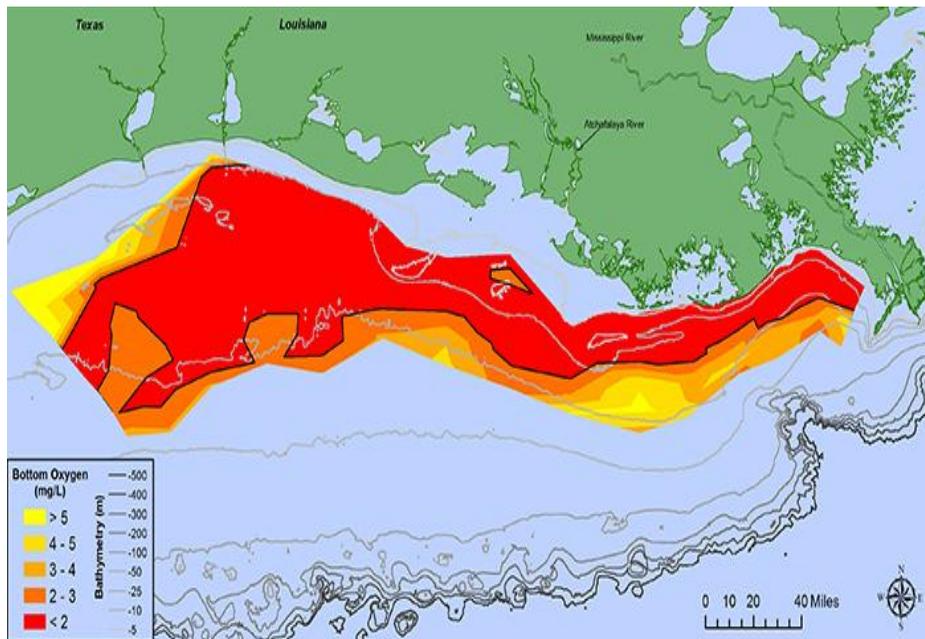


Figure 5.15. Gulf of Mexico dead zone in July 2017. At 8,776 square miles, this year's dead zone in the Gulf of Mexico is the largest ever measured. Source: National Oceanic and Atmospheric Administration (NOAA) of the United States.

The Gulf of Mexico is a major source area for the seafood industry. The Gulf supplies 72% of U.S. harvested shrimp, 66% of harvested oysters, and 16% of commercial fish. Consequently, if the hypoxic zone continues or worsens, fishermen and coastal state economies will be greatly impacted.

5.3.10. The Love Canal, 1978

In 1978, Love Canal, located near Niagara Falls in upstate New York, was a nice little working-class enclave with hundreds of houses and a school. It just happened to sit atop 22,000 tons of toxic industrial waste that had been buried underground in the 1940s and '50s by a local company. Over the years, the waste began to bubble up into backyards and cellars (Figure 5.16).

By 1978, the problem was unavoidable, and hundreds of families sold their houses to the federal government and evacuated the area. According to the United States Environmental Protection Agency (EPA) in 1979, residents exhibited a "disturbingly high rate of miscarriages ... Love Canal can now be

added to a growing list of environmental disasters involving toxics, ranging from industrial workers stricken by nervous disorders and cancers to the discovery of toxic materials in the milk of nursing mothers." In one case, two out of four children in a single Love Canal family had birth defects; one girl was born deaf with a cleft palate, an extra row of teeth, and slight retardation, and a boy was born with an eye defect.

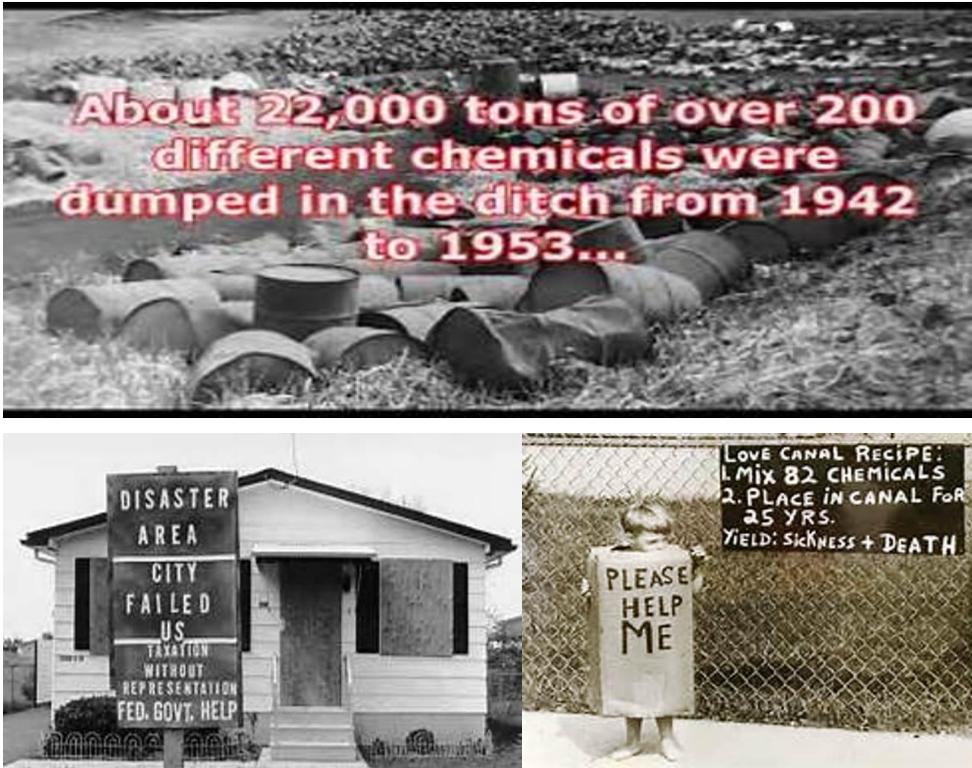


Figure 5.16. Love Canal dump site located near Niagara Fall, 1978.

Source: Getty Images

Finally, in 1978 President Carter declared a state of emergency at Love Canal, making it the first human-caused environmental problem to be designated that way. The Love Canal incident became a symbol of improperly stored chemical waste. The disaster led to the formation in 1980 of the Superfund program, which helps pay for the cleanup of toxic sites. Clean up of Love Canal, which was funded by Superfund and completely finished in 2004, involved removing contaminated soil, installing drainage pipes to

capture contaminated groundwater for treatment, and covering it with clay and plastic.

5.3.11. The Al-Mishraq Fire, 2003

Al-Mishraq is a state run sulfur plant near Mosul, Iraq. In June 2003, it was the site of the largest human-made release of sulfur dioxide ever recorded when a fire (thought to have been deliberately started) gained control and burned for about three weeks. At its height, the fire was putting 21,000 tons of sulfur dioxide a day into the atmosphere. The pollution in Mosul, which is about 45 kilometres from Mishraq, reached a catastrophic level. For over 48 hours the white smoke from sulfur dioxide could be seen in the air. Two civilians died and nearly 1,000 people were treated for toxic gas inhalation. Shifting winds sent the gas to Qayyarah Airfield West, where U.S. and coalition forces were forced to use gas masks (Figure 5.17).



Figure 5.17. Al Mishraq Fire 2003 Man-Made Disaster.
Source: Screencast-O-Matic.com

Sulfur dioxide can cause respiratory problems, wipe out crops and lead to acid rain. Many people were hospitalized, and most of the area's vegetation was destroyed.

5.3.12. Ecocide in Vietnam during the Vietnam War

During the Vietnam War, destruction of the farmland and rice paddies that fed the enemy was promulgated by the American military strategists. Other than these areas which were the source of food and livelihood of the Vietnamese folk, the jungle along with its flora and fauna was also devastated.



Figure 5.18. Destruction of the farmland and rice paddies by spraying a variety of herbicides during the Vietnam War.
Source: greenpeace.org

The term **ecocide**, which refers to any large-scale destruction of the natural environment, was coined after the herbicide disaster in Southeast Asia during the Vietnam War (Figure 5.18 and 5.19). In order to prevent Communists from hiding in and subsisting on the jungles' vegetation, the



Figure 5.19. Herbicide disaster in Southeast Asia during the Vietnam War
Source: greenpeace.org

U.S. Army sprayed a variety of herbicides, like Agent Orange, into the foliage. The side effects of the toxic spray were devastating: Cancer, birth defects and disabilities are among the myriad health problems that plague survivors and their children to this day.

5.3.13. Amoco Cadiz Oil Spill, 1978

A huge crude carrier bearing the flag of Liberia split into three parts and sank, releasing 1,604,500 barrels (219,797 tons) of light crude oil and 4,000 tons of fuel oil making it the largest oil spill of its kind at that time and resulted in the largest loss of marine life ever recorded from an oil spill (Figure 5.20).

At the time (March 1978), **Amoco Cadiz** incident resulted in the largest loss of marine life ever recorded from an oil spill. Mortalities of most animals occurred over the two months following the spill. Two weeks following the accident, millions of dead molluscs, sea urchins, and other bottom dwelling organisms washed ashore.



Figure 5.20. Sinking of the Amoco Cadiz in Portsall, France in March, 1978.

Source: Wikipedia



Figure 5.21. Amoco Cadiz spill (Black coloured area in photo) was the largest recorded spill in history, 1978. Source: Wikipedia

Diving birds constituted the majority of the nearly 20,000 dead birds that were recovered. The oyster mortality from the spill was estimated at 9,000 tons. Fishermen in the area caught fish with skin ulcerations and tumors.

Some of the fish caught in the area reportedly had a strong taste of petroleum. Although echinoderm and small crustacean populations almost completely disappeared, the populations of many species recovered within a year. Cleanup activities on rocky shores, such as pressure-washing, also caused habitat impacts.

The **Amoco Cadiz** spill was the largest recorded spill in history and was the first spill in which estuarine tidal rivers were oiled (Figure 5.21). No follow-up mitigation existed to deal with asphalt formation and problems that resulted after the initial aggressive cleanup.

Additional erosion of beaches occurred in several places where no attempt was made to restore the gravel that was removed to lower the beach face. Many of the affected marshes, mudflats, and sandy beaches, were low-energy areas. Evidence of oiled beach sediments can still be seen in some of these sheltered areas. Layers of sub-surface oil still remain buried in many of the impacted beaches.

5.3.14. Deep water horizon (BP) oil spill, 2010

One of the most recent disasters, the explosion and massive leak (beneath the **Deep Water Horizon** oil rig) resulted from failed sensors and shut-off valves nearly one mile under the sea (April 20, 2010). Eleven workers were killed in the initial explosion and fire. The rig was owned by Trans Ocean, but leased and controlled by BP. The leak of an estimated 60 million barrels of mixed-grade oil from the unplugged well continued for more than four months (Figure 5.22).



Figure 5.22. BP oil spill explosion , 2010. Source: BBC.com



Figure 5.23. Total extent of oil spill, 68,000 square miles in the Gulf of Mexico, 2010. Source: Skytruth.org



Figure 5.24. Large numbers of dead dolphins began washing ashore in Alabama and Mississippi in 2010. Source: Nature

The deep water horizon oil spill (also referred to as the BP oil spill) in the Gulf of Mexico is considered the largest accidental marine oil spill in the history of the petroleum industry (Figure 5.23). Over 34, 000 birds (including egrets and blue heron), hundreds of sea turtles, some six dozen dolphins and other marine vertebrates and invertebrates were poisoned, suffocated and died in the surface layers of the spill (Figure 5.24). As of September, 2010, when much of the surface spill had been dispersed (using millions of gallons of a harmful dispersant called Corexit), reports of underwater (mid ocean depth) oil plumes extending for dozens of miles were being reported. The long-term effects on marine life and the regional economy caused by this spill — considered the worst in history — will be felt and studied for decades to come.

5.3.15. Libby, Montana Asbestos Contamination, 1979-2011

The W.R. Grace plant in Libby, Montana, spewed asbestos over the small town for decades, sickening more than 1,000 people and killing more than 200 (Figure 5.25). Smoke from the factory coated the town in tremolite asbestos, a particularly toxic form linked to numerous diseases including mesothelioma.

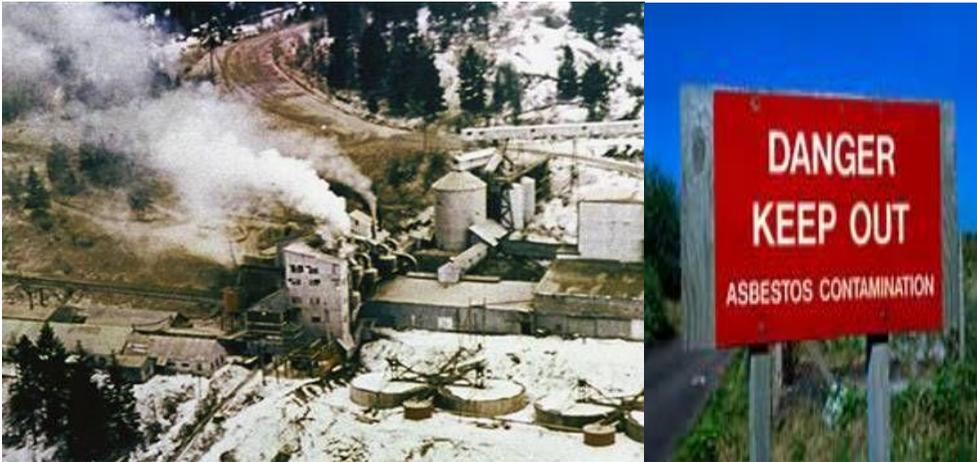


Figure 5.25. Libby asbestos contamination, 1979-2011.

Source: Mother Nature Network

Dust from the plant became part of the residents' lives in 1919, and for years it covered lawns, dusted cars and drifted through the air. Tailings from the plant were used as fill for driveways, gardens, playgrounds and even the Libby junior high and high school tracks. Family members of mine workers were also exposed to asbestos that employees brought home on their work clothing. The mine closed in 1990, and the company is now bankrupt after facing more than 270,000 asbestos-related lawsuits, but asbestos remains in Libby. No one knows exactly how many people have been affected, but a local health clinic that specializes in asbestos-related diseases says it has 1,400 patients and sees about 20 new patients each month. It was reported that asbestos-related mortality in Libby lasted for 33 years (1979–2011).

5.3.16. The Sidoarjo Mudflow and the Muddiness of an Environmental Disaster, 2006

The Sidoarjo mudflow, also known as “Lusi,” a contraction of its Indonesian name Lumpur Sidoarjo, is one of the longest ongoing disasters in recent memory. A mud volcano in the Sidoarjo district, East Java, Indonesia, has been spewing hot mud and gases since 29 May 2006. Sidoarjo (the largest mud volcano in the world) also known as the Lapindo mud, exists today because of gas blowout wells drilled by PT Lapindo Branta. Branta denies this however and claims that the mud flows were created by an earthquake. 180,000 m³ of mud per day is spewed at its peak and has been in eruption

since May, 2006. The Sidoarjo Mudflow — the biggest mud volcano in the world — has buried entire villages and left 40,000 people homeless in Surabaya, a major port city in East Java (Figure 5.26, 5.27 and 5.28).



Figure 5.26. Mud Flow buried entire villages, East Java, Indonesia, 2006. Source: Sott



Figure 5.27. Mud and steam are spewing from the earth on the island of Java, Indonesia, leaving thousands of people homeless, 2006. Source: volcanocafe.wordpress.com

From the time the eruption occurred, geologists debated the cause of the eruption. Some geologists pointed to a minor earthquake in the region. **In June 2008, however, a report released by British, American, Indonesian, and Australian scientists concluded that the volcano was not a natural disaster, but the result of oil and gas drilling.** At the time the report was released, the volcano was producing roughly 100,000 cubic meters (3.5 million cubic feet) of mud per day— enough to fill 53 Olympic swimming pools.



Figure 5.28. Statues honouring the mudflow victims, Java, Indonesia, 2006
Picture Source: Getty Images

5.3.17. The Palomares Incident, 1966

The **1966 Palomares B-52 crash**, or the **Palomares incident**, occurred on 17 January 1966, when a B-52G bomber of the United States Air Force's Strategic Air Command collided with a KC-135 tanker during mid-air refuelling at 31,000 feet (9,450 m) over the Mediterranean Sea, off the coast of Spain (Figure 5.29). The KC-135 was completely destroyed when its fuel

load ignited, killing all four crew members. The B-52G broke apart, killing three of the seven crew members aboard.



Figure 5.29. The B52G was carrying four Mk28 hydrogen bombs. Two of the non-nuclear explosives on the bombs went off when they hit the ground. This caused two quarter square miles of contaminated area by plutonium. Source: History on Air.

Of the four Mk28-type hydrogen bombs the B-52G carried, three were found on land near the small fishing village of Palomares in the municipality of Cuevas del Almanzora, Almería, Spain. The non-nuclear explosives in two of the weapons detonated upon impact with the ground, resulting in the contamination of a 2-square-kilometer (490-acre) (0.78 square mile) area by plutonium. The fourth, which fell into the Mediterranean Sea, was recovered intact after a 2½-month-long search.

5.3.18. Darvasa gas crater (Door to Hell), 1971

In Derweze, Turkmenistan, a drilling rig made by Soviet geologists in 1971 gave way to a large hole measuring 70 meters in diameter, exposing a large methane gas reservoir (Figure 5.30). Fearing the environmental impact due to the substantial methane gas release; the geologists decided to burn it off. Unfortunately, the gas is still burning today. At the heart of

Turkmenistan's Karakum Desert sits a crater of fire the size of a football field that's been perpetually burning now for almost fifty years. Locals have suitably dubbed it the 'Door To Hell', officially it's known as the Darvaza Gas Crater. It's not volcanic, that's not magma, **in fact, this sinister flame pit was man-made and thought to be the result of a Soviet-era gas drilling accident.**



**Figure 5.30. Darvasa gas crater, nicknamed the 'door to hell', in Turkmenistan has been burning for over 40 years.
Source: Huffington Post**

5.3.19. The Kuwait Oil Fires, 1991

The **Kuwaiti oil fires** were caused by Iraqi military forces setting fire to a reported 605 to 732 oil wells along with an unspecified number of oil filled low-lying areas, such as oil lakes and fire trenches, as part of a scorched earth policy while retreating from Kuwait in 1991 due to the advances of Coalition military forces in the Persian Gulf War. The fires were started in January and February 1991, and the first well fires were extinguished in early April 1991, with the last well capped on November 6, 1991 (Figure 5.31).

Around 6 million barrels of oil were lost from January to November, 1991. 600 oil wells were set afire as part of the scorched earth policy by the retreating Iraqi military forces. \$1.5 billion was spent by Kuwait to extinguish the fires that caused heavy pollution to the soil and air. When oilfields burn, they release pollutants like sulphur dioxide, carbon monoxide, and lead. These pollutants adversely affect human health and are particularly damaging to those who are already suffering from respiratory illnesses.



Figure 5.31. The Kuwait Oil Fires, 1991. Source: timesunion.com

As the 1991 Persian Gulf War drew to a close, Hussein sent men to blow up Kuwaiti oil wells. Approximately 600 were set ablaze, and the fires — literally towering infernos — burned for seven months. The Gulf was awash in poisonous smoke, soot and ash. Black rain fell. Lakes of oil were created. As NASA wrote, "The sand and gravel on the land's surface combined with oil and soot to form a layer of hardened 'tarcrete' over almost 5 percent of the country's area." Scores of livestock and other animals died from the oily mist, their lungs blackened by the liquid.

3.3.20. The Three Mile Island Nuclear Explosion, 1979

The three mile island accident was a partial nuclear meltdown which occurred in one of the two United States nuclear reactors on March 28, 1979. Located on the three mile island in Dauphin County, Pennsylvania; it was the

worst accident in U.S. commercial nuclear power plant history with the partial meltdown resulting in the release of small amounts of radioactive gases and radioactive iodine into the environment (Figure 5.32). Worries were expressed by anti-nuclear movement activists; however, epidemiological studies analyzing the rate of cancer in and around the area since the accident, determined there was a small statistically non-significant increase in the rate and thus no causal connection linking the accident with these cancers has been substantiated. Cleanup started in August 1979, and officially ended in December 1993, with a total cleanup cost of about \$1 billion.



Figure 5.32. An accident at Three Mile Island, a nuclear power plant located near Harrisburg, Pennsylvania, led to the meltdown of the core reactor, 1979. Source: lancasteronline.com

5.3.21. Castle Bravo, 1954

The code name **Castle Bravo** was given to the first United States test of a dry fuel thermonuclear hydrogen bomb. The bomb was detonated on Bikini Atoll, Marshall Islands on March 1, 1954, as the first test of Operation Castle Bravo and was the most powerful nuclear device ever detonated by the United States at that time (Figure 5.33). The bomb was over 1000 times more powerful than those exploded over Hiroshima Nagasaki in 1945.



Figure 5.33. Cloud top rose and peaked at 130,000 feet (almost 40 km) after only six minutes after explosion of hydrogen bomb, 1954. Source: Wikipedia

The Bravo crater in the atoll reef had a diameter of 6,510 ft, with a depth of 250 ft. The cloud top rose and peaked at 130,000 feet (almost 40 km) after only six minutes. Eight minutes after the test the cloud had reached its full dimensions with a diameter of 100 km, a stem 7 km thick, and a cloud bottom rising above 55,000 feet (16.5 km), and after 10 minutes had a diameter of more than 60 miles.

Intense radioactive fallout from the cloud was carried eastward and severely contaminated a Japanese commercial fishing boat and the atolls of Rongelap, Alinginea, Rongerik, and Utirik, some 200 miles away. About five hours after detonation, fallout began to deposit on the Rongelap Atoll. The fallout was so heavy that the Rongelap people, who had never seen snow, thought it was snowing. In the early morning of March 1, 1954, residents of Adelaide, Australia were awakened to a violent shaking in their beds. When they went outside, they saw a brilliant glow in the east. The United States had just set off the Casyle Bravo nuclear bomb on Bikini Isiang, 3,600 miles away.

5.3.22. Jilin Chemical Plant Explosions in China, 2005

The Jilin chemical plant explosions were a series of explosions which occurred on November 13, 2005 in the No.101 Petro chemical plant in Jilin City, Jilin Province, China (Figure 5.34). These explosions were responsible for the deaths of six workers and injured dozens causing the evacuation of tens of thousands of residents. To add insult to injury, these explosions severely polluted the Songhua River with an estimated 100 tons of pollutants containing benzene and nitrobenzene whose exposure reduces white blood cell count and is linked to leukemia.



Figure 5.34. Smoke rised into the sky after explosions at a chemical plant in Jilin City in northeast China, 2005. Source: notiaactual.com

5.3.23. Pacific Gyre Garbage Patch, 1985

The Great Pacific garbage patch, also described as the Pacific trash vortex, is a gyre of marine debris particles in the central North Pacific Ocean discovered between 1985 and 1988. It is located roughly between 135°W to 155°W and 35°N to 42°N (Figure 5.35). This patch which is characterized by high concentrations of pelagic plastics, chemical sludge and other debris formed gradually as a result of the marine pollution gathered by oceanic currents.

Twice the size of Texas, the Great Pacific Garbage Patch stretches for hundreds of miles across the North Pacific Ocean and is one of the most frightening examples of just how much human activity is violating the planet.



Figure 5.35. The Great Pacific Garbage Patch.

Source: captain-charles-moore.org

Affected species include sea turtles and the black-footed albatross. Midway Atoll receives substantial amounts of marine debris from the patch. These toxin-containing plastic pieces are also eaten by jellyfish, which are then eaten by fish. Many of these fish are then consumed by humans, resulting in their ingestion of toxic chemicals.

5.3.24. The Exxon Valdez Oil Spill, 1989

On March 24, 1989, 260,000 to 750,000 barrels of crude oil was spilled in Prince William Sounds, Alaska by the oil tanker Exxon Valdez after it ran into Bligh Reef. It would eventually spread almost 500 miles from the original crash site and stain thousands of miles of coastline (Figure 5.36). It is considered to be one of the most devastating human-caused environmental disasters with both the long-term and short-term effects of the oil spill having been studied. Immediate effects included the deaths of 100,000 to as many as

250,000 seabirds, at least 2,800 sea otters, 300 harbor seals, 247 Bald Eagles, and 22 Orcas, and an unknown number of salmon and herring (Figure 5.37).



Figure 5.36. An oil skimming operation works in a heavy oil slick near Latouche Island on April 1, 1989. Source: The Atlantic



Figure 5.37. The Exxon Valdez Oil Spill caused deaths as many as 250,000 seabirds, at least 2,800 sea otters, 300 harbor seals, 247 Bald Eagles, and 22 Orcas, and an unknown number of salmon and herring. Source: Pinterest

The research team from the National Oceanic and Atmospheric Administration estimated that 26,000 gallons (55, 000 liters) of Valdez oil remain scattered along 11 hectares of beach and coastline. Some of these oil patches (comprising only 4% of the total spilled) show no signs of decaying. While the *Exxon Valdez* oil leak is considered to be the largest man-made environmental disaster in U.S. history, the Gulf of Mexico spill may eventually surpass it in severity.

5.3.25. TVA Kingston Fossil Plant Coal Fly Ash Slurry Spill, 2008

The **TVA Kingston Fossil Plant coal fly ash slurry spill** occurred just before 1 a.m. on Monday December 22, 2008, when an ash dike ruptured at an 84-acre (0.34 km²) solid waste containment area at the Tennessee Valley Authority's Kingston Fossil Plant in Roane County, Tennessee, USA. 1.1 billion US gallons (4,200,000 m³) of coal fly ash slurry was released.

The coal-fired power plant, located across the Clinch River from the city of Kingston, uses ponds to dewater the fly ash, a byproduct of coal combustion, which is then stored in wet form in dredge cells. The slurry (a mixture of fly ash and water) traveled across the Emory River and its Swan Pond embayment, on to the opposite shore, covering up to 300 acres (1.2 km²) of the surrounding land, damaging homes and flowing up and down stream in nearby waterways such as the Emory River and Clinch River (tributaries of the Tennessee River). It was the largest fly ash release in United States history (Figure 5.38 and 5.39).

Analysis of coal ash has revealed high concentrations of heavy metals, including lead, arsenic, selenium, mercury, chromium, nickel and boron. In high concentrations, these metals can cause cancers and various neurological problems. The long-term, health hazards posed by these sites can last more than a century, as heavy metals take long periods of time to leach out into the soil and then sink into water table.



Figure 5.38. Aerial photograph of site taken the day after the event of Coal Fly Ash Slurry Spill, 2008 (Photo courtesy Tennessee Valley Authority)



Figure 5.39. The wet fly ash engulfed this house, one of 12 damaged in the spill, 2008. (Photo courtesy Tennessee Valley Authority)

5.4. Disaster Risk Reduction & Disaster Risk Management

Historically, dealing with disasters focused on emergency response, but towards the end of the 20th century it was increasingly recognised that disasters are not natural (even if the associated hazard is) and that it is only by reducing and managing conditions of hazard, exposure and vulnerability that we can prevent losses and alleviate the impacts of disasters. Since we cannot reduce the severity of natural hazards, the main opportunity for reducing risk lies in reducing vulnerability and exposure. Reducing these two components of risk requires identifying and reducing the underlying drivers of risk, which are particularly related to poor economic and urban development choices and practice, degradation of the environment, poverty and inequality and climate change, which create and exacerbate conditions of **hazard, exposure and vulnerability**. Addressing these underlying risk drivers will reduce disaster risk, lessen the impacts of climate change and, consequently, maintain the sustainability of development (UNISDR, 2015a).

Disaster risk reduction (DRR) is a part of sustainable development, so it must involve every part of society, government, non-governmental organizations and the professional and private sector. It therefore requires a people-centred and multi-sector approach, building resilience to multiple, cascading and interacting hazards and creating a culture of prevention and resilience.

Consequently **disaster risk management (DRM)** includes strategies designed to:

- avoid the construction of new risks
- address pre-existing risks
- share and spread risk to prevent disaster losses being absorbed by other development outcomes and creating additional poverty

Although disaster risk management (DRM) includes disaster preparedness and response activities, it is about much more than managing disasters (UNISDR, 2015a). Successful DRR results from the combination of top-down, institutional changes and strategies, with bottom-up, local and community-based approaches. DRM programmes should not be stand alone but instead be integrated within development planning and practice, since

disasters are an indicator of failed or skewed development, of unsustainable economic and social processes, and of ill-adapted societies (UNISDR, 2009b and 2015a). Approaches need to address the different layers of risk (from intensive to extensive risk), underlying risk drivers, as well as be tailored to local contexts. There is no ‘one-size fits all’ approach to DRM, but there exist a number of approaches and frameworks, which have been effectively implemented to reduce disaster risk. But, before being able to reduce risk, we need to understand the hazards, and the exposure and vulnerability of people and assets to those hazards.

Disaster risk management involves activities related to:

Prevention. The outright avoidance of adverse impacts of hazards and related disasters (often less costly than disaster relief and response). For instance, relocating exposed people and assets away from a hazard area.

Mitigation. The lessening or limitation of the adverse impacts of hazards and related disasters. For instance, constructing flood defences, planting trees to stabilize slopes and implementing strict land use and building construction codes.

Transfer. The process of formally or informally shifting the financial consequences of particular risks from one party to another whereby a household, community, enterprise or state authority will obtain resources from the other party after a disaster occurs, in exchange for ongoing or compensatory social or financial benefits provided to that other party. For instance, insurance.

Preparedness. The knowledge and capacities of governments, professional response and recovery organisations, communities and individuals to effectively anticipate, respond to, and recover from the impacts of likely, imminent or current hazard events or conditions. For instance, installing early warning systems, identifying evacuation routes and preparing emergency supplies. Implementation of these activities and measures is rarely done in isolation and includes a number of associated activities, including:

- Identification and measuring disaster risk
- Education and knowledge development

- Informing people about their risk (awareness raising)
- Incorporating DRM into national planning and investment
- Strengthening institutional and legislative arrangements
- Providing financial protection for people and businesses at risk (finance and contingency planning)
- Integrating DRR across multiple sectors, including health, environment, etc.

Response activities for reducing risk can be described as structural, for instance land use planning and implementation of building codes, and non-structural, for instance awareness raising, policy-making and legislation. How governments, civil society and other actors organise DRM, for example through institutional arrangements, legislation and decentralisation, and mechanisms for participation and accountability is termed risk governance. There is clear evidence to suggest that low-income countries with weak governance are more vulnerable and less resilient to disaster risk (UNISDR, 2015a).

Fundamentally, DRR succeeds in reducing risk by building the strengths, attributes and resources available within a community, society or organization – collectively known as their capacity. DRM activities are designed to increase the resilience of people, communities, society and systems to resist, absorb, accommodate and to recover from and improve well-being in the face of multiple hazards. Activities for reducing and managing risks can therefore provide a way for building resilience to other risks. In addition to development, DRM should therefore be integrated across a number of sectors, including climate change and conflict.

Awareness, identification, understanding and measurement of disaster risks are all clearly fundamental underpinnings of disaster risk management (UNISDR, 2015a). Disaster risk reduction is about decisions and choices, including a lack of, so risk information has a role in five key areas of decision making:

Risk identification. Because the damages and losses caused by historical disasters are often not widely known, and because the potential damages and losses that could arise from future disasters (including infrequent but high-impact events) may not be known at all, DRM is given a low priority.

Appropriate communication of robust risk information at the right time can raise awareness and trigger action.

Risk reduction. Hazard and risk information may be used to inform a broad range of activities to reduce risk, from improving building codes and designing risk reduction measures (such as flood and storm surge protection), to carrying out macro-level assessments of the risks to different types of buildings (for prioritizing investment in reconstruction and retrofitting, for example).

Preparedness. An understanding of the geographic area affected, along with the intensity and frequency of different hazard events, is critical for planning evacuation routes, creating shelters, and running preparedness drills. Providing a measure of the impact of different hazard events—potential number of damaged buildings, fatalities and injuries, secondary hazards—makes it possible to establish detailed and realistic plans for better response to disasters, which can ultimately reduce the severity of adverse natural events.

Financial protection. Disaster risk analysis was born out of the financial and insurance sector's need to quantify the risk of comparatively rare high-impact natural hazard events. Governments need to seek to manage their sovereign financial risk or support programs that manage individual financial risks (e.g., micro-insurance or household earthquake insurance).

Resilient reconstruction. Risk assessment can play a critical role in impact modelling before an event strikes (in the days leading up to a cyclone, for example), or it can provide initial and rapid estimates of human, physical, and economic loss in an event's immediate aftermath. Moreover, risk information for resilient reconstruction needs to be available before an event occurs, since after the event there is rarely time to collect the information needed to inform resilient design and land-use plans.

If those exposed to hazards are unaware of the risks they face, it is difficult to see how or why households, businesses or governments would invest in reducing their risk levels. However, while risk awareness may be a precondition, the importance people attach to managing their risks can only be understood in the context of the full range of social, economic, territorial and environmental constraints and opportunities they face (UNISDR, 2015a).

Governments need to invest in the collection, management and dissemination of risk information, including disaster loss and impact statistics, hazard models, exposure databases and vulnerability information. At the same time, they need to put standards and mechanisms in place to ensure openness and transparency so that users not only have access to the information they need but are aware of its underlying assumptions and limitations (UNISDR, 2015a). The generation of understandable and actionable risk information needs to be particularly sensitive to extensive risk, which, because it is configured to a large extent by social, economic and environmental vulnerability, can be reduced effectively through risk management and sustainable development practices (UNISDR, 2015a).

6. Case Studies of some Man made Environmental Disasters

An environmental disaster is defined as a specific event caused by human activity that results in a seriously negative effect on the environment. Sometimes a natural disaster can become an environmental disaster. In most cases environmental disasters are caused by human error, accident, lack of foresight, corner cutting during industrial processes, greed, or by simple incompetence.

6.1. Case Study of Bhopal Plant Disaster, 1984

6.1.1. Introduction

The **Bhopal disaster**, also referred to as the **Bhopal gas tragedy**, was a gas leak incident in India, considered the world's worst industrial disaster. It occurred on the night of 2–3 December 1984 at the Union Carbide India Limited (UCIL) pesticide plant in Bhopal, Madhya Pradesh (Figure 6.1 and 6.2). Over 500,000 people were exposed to methyl isocyanate (MIC) gas and other chemicals. The highly toxic substance made its way into and around the shanty towns located near the plant. The Bhopal case is an in-depth study of the industrial accident at the Union Carbide factory in India that immediately killed 2,000 people, injured another 200,000 to 300,000 more, and immediately raised questions about plant safety and corporate responsibility around the world.

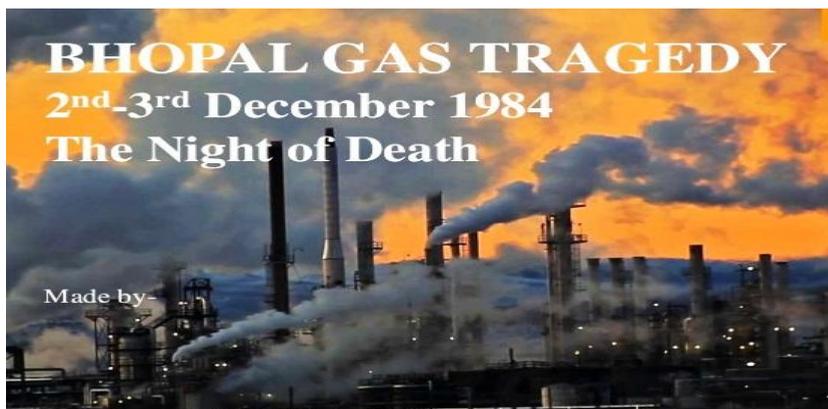
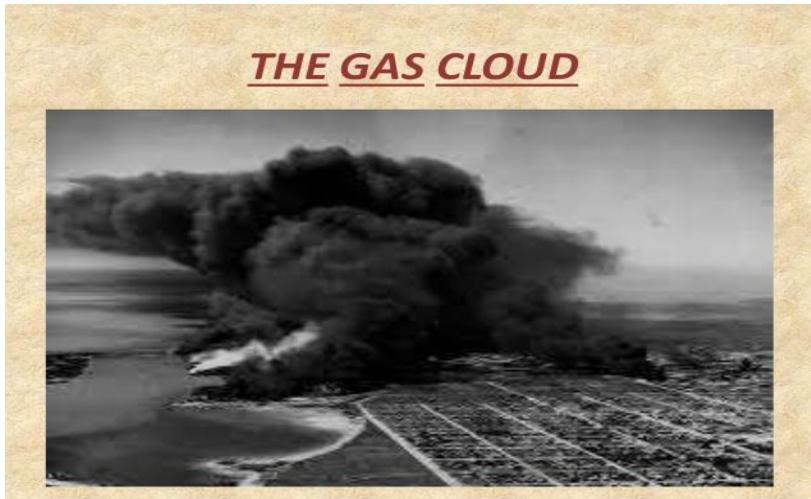


Figure 6.1. Bhopal gas tragedy, 1984. Source: Toxipedia

Estimates vary on the death toll. The official immediate death toll was 2,259. The government of Madhya Pradesh confirmed a total of 3,787 deaths related to the gas release. A government affidavit in 2006 stated that the leak caused 558,125 injuries, including 38,478 temporary partial injuries and approximately 3,900 severely and permanently disabling injuries. Others estimate that 8,000 died within two weeks, and another 8,000 or more have since died from gas-related diseases.



**Figure 6.2. The accidental release of 40 metric tons of methyl isocyanate from a Union Carbide pesticide plant in the heart of Bhopal.
Source: Toxipedia**

The cause of the disaster remains under debate. The Indian government and local activists argue that slack management and deferred maintenance created a situation where routine pipe maintenance caused a backflow of water into a MIC tank, triggering the disaster. Union Carbide Corporation (UCC) contends water entered the tank through an act of sabotage.

The owner of the factory, UCIL, was majority owned by UCC, with Indian Government-controlled banks and the Indian public holding a 49.1 percent stake. In 1989, UCC paid \$470m (\$907m in 2014 dollars) to settle litigation stemming from the disaster. In 1994, UCC sold its stake in UCIL to Eveready Industries India Limited (EIIL), which subsequently merged with McLeod Russel (India) Ltd. Eveready ended clean-up on the site in 1998, when it terminated its 99-year lease and turned over control of the site to the

state government of Madhya Pradesh. Dow Chemical Company purchased UCC in 2001, seventeen years after the disaster.

Civil and criminal cases were filed in the District Court of Bhopal, India, involving UCC and Warren Anderson, UCC CEO at the time of the disaster. In June 2010, seven ex-employees, including the former UCIL chairman, were convicted in Bhopal of causing death by negligence and sentenced to two years imprisonment and a fine of about \$2,000 each, the maximum punishment allowed by Indian law.

6.1.2. The initial effects of exposure

The initial effects of exposure were coughing, severe eye irritation and a feeling of suffocation, burning in the respiratory tract, blepharospasm, breathlessness, stomach pains and vomiting. People awakened by these symptoms fled away from the plant. Those who ran inhaled more than those who had a vehicle to ride. Owing to their height, children and other people of shorter stature inhaled higher concentrations. Thousands of people had died by the following morning (Figure 6.3).



Figure 6.3. Poison gas leaking from a Union Carbide pesticide factory in Bhopal spread throughout the city, killing thousands of people outright. Source: thinglink.com

Primary causes of deaths were choking, reflexogenic circulatory collapse and pulmonary oedema. Findings during autopsies revealed changes not only in the lungs but also cerebral oedema, tubular necrosis of the kidneys, fatty degeneration of the liver and necrotising enteritis. The still birth rate increased by up to 300% and neonatal mortality rate by around 200%.

Within a few days, trees in the vicinity became barren and bloated animal carcasses had to be disposed of. 170,000 people were treated at hospitals and temporary dispensaries; 2,000 buffalo, goats, and other animals were collected and buried. Supplies, including food, became scarce owing to suppliers' safety fears. Fishing was prohibited causing further supply shortages.

Lacking any safe alternative, on 16 December, tanks 611 and 619 were emptied of the remaining MIC by reactivating the plant and continuing the manufacture of pesticide. Despite safety precautions such as having water carrying helicopters continually overflying the plant, this led to a second mass evacuation from Bhopal. The Government of India passed the "Bhopal Gas Leak Disaster Act" that gave the government rights to represent all victims, whether or not in India. Complaints of lack of information or misinformation were widespread. An Indian government spokesman said, "Carbide is more interested in getting information from us than in helping our relief work".

Formal statements were issued that air, water, vegetation and foodstuffs were safe, but warned not to consume fish. The number of children exposed to the gases was at least 200,000. Within weeks, the State Government established a number of hospitals, clinics and mobile units in the gas-affected area to treat the victims.

6.1.3. Long-term health effects

Some data about the health effects are still not available. The Indian Council of Medical Research (ICMR) was forbidden to publish health effect data until 1994.

A total of 36 wards were marked by the authorities as being "gas affected," affecting a population of 520,000. Of these, 200,000 were below 15 years of age, and 3,000 were pregnant women. The official immediate death toll was 2,259, and in 1991, 3,928 deaths had been officially certified. Ingrid Eckerman estimated 8,000 died within two weeks.

The government of Madhya Pradesh confirmed a total of 3,787 deaths related to the gas release. Later, the affected area was expanded to include 700,000 citizens. A government affidavit in 2006 stated the leak caused 558,125 injuries including 38,478 temporary partial injuries and approximately 3,900 severely and permanently disabling injuries.

A cohort of 80,021 exposed people was registered, along with a control group, a cohort of 15,931 people from areas not exposed to MIC. Nearly every year since 1986, they have answered the same questionnaire. It shows over mortality and over morbidity in the exposed group. Bias and confounding factors cannot be excluded from the study. Because of migration and other factors, 75% of the cohort is lost, as the ones who moved out are not followed.

A number of clinical studies were performed. The quality varies, but the different reports support each other. Studied and reported long-term health effects are:

- Eyes: Chronic conjunctivitis, scars on cornea, corneal opacities, early cataracts
- Respiratory tracts: Obstructive and/or restrictive disease, pulmonary fibrosis, aggravation of TB and chronic bronchitis
- Neurological system: Impairment of memory, finer motor skills, numbness etc.
- Psychological problems: Post traumatic stress disorder (PTSD)
- Children's health: Peri- and neonatal death rates increased. Failure to grow, intellectual impairment, etc.

Missing or insufficient fields for research are female reproduction, chromosomal aberrations, cancer, immune deficiency, neurological sequelae, post traumatic stress disorder (PTSD) and children born after the disaster. Late cases that might never be highlighted are respiratory insufficiency, cardiac insufficiency (cor pulmonale), cancer and tuberculosis.



Figure 6.4. Children were born with mental and physical disabilities in Bhopal.
Source: bhopal.org

A 2014 report in *Mother Jones* quotes a "spokesperson for the Bhopal Medical Appeal, which runs free health clinics for survivors" as saying "An estimated 120,000 to 150,000 survivors still struggle with serious medical conditions including nerve damage, growth problems, gynecological disorders, respiratory issues, birth defects, and elevated rates of cancer and tuberculosis" (Figure 6.4).

6.1.4. Health care

The Government of India had focused primarily on increasing the hospital-based services for gas victims and therefore hospitals were built after the disaster. When UCC wanted to sell its shares in UCIL, it was directed by the Supreme Court to finance a 500-bed hospital for the medical care of the survivors. Thus, Bhopal Memorial Hospital and Research Centre (BMHRC) was inaugurated in 1998 and was obliged to give free care for survivors for eight years. BMHRC was a 350-bedded super speciality hospital where heart surgery and hemodialysis were done. There was a dearth of gynaecology, obstetrics and paediatrics. Eight mini-units (outreach health centres) were started and free health care for gas victims were to be offered till 2006. The management had also faced problems with strikes, and the quality of the health care being disputed. Sambhavna Trust is a charitable trust, registered in 1995, that gives modern as well as ayurvedic treatments to gas victims, free of charge.

6.1.5. Environmental rehabilitation

When the factory was closed in 1986, pipes, drums and tanks were sold. The MIC and the Sevin plants are still there, as are storages of different residues. The area around the plant was used as a dumping area for hazardous chemicals. In 1982 tubewells in the vicinity of the UCIL factory had to be abandoned and tests in 1989 performed by UCC's laboratory revealed that soil and water samples collected from near the factory and inside the plant were toxic to fish. Several other studies had also shown polluted soil and groundwater in the area. Reported polluting compounds include Inaphthol, naphthalene, Sevin, tarry residue, mercury, toxic organochlorines, volatile organochlorine compounds, chromium, copper, nickel, lead, hexachloroethane, hexachlorobutadiene, and the pesticide HCH.

In order to provide safe drinking water to the population around the UCIL factory, Government of Madhya Pradesh presented a scheme for improvement of water supply. In December 2008, the Madhya Pradesh High Court decided that the toxic waste should be incinerated at Ankleshwar in Gujarat, which was met by protests from activists all over India. On 8 June 2012, the Centre for incineration of toxic Bhopal waste agreed to pay US\$3.9 million to dispose of UCIL chemical plants waste in Germany. On 9 August 2012, Supreme court directed the Union and Madhya Pradesh Governments to take immediate steps for disposal of toxic waste lying around and inside the factory within six months.

A U.S. court rejected the lawsuit blaming UCC for causing soil and water pollution around the site of the plant and ruled that responsibility for remedial measures or related claims rested with the State Government and not with UCC. In 2005, the state government invited various Indian architects to enter their "concept for development of a memorial complex for Bhopal gas tragedy victims at the site of Union Carbide". In 2011, a conference was held on the site, with participants from European universities which was aimed for the same.

6.1.6. Ongoing contamination

Chemicals abandoned at the plant continue to leak and pollute the groundwater. Whether the chemicals pose a health hazard is disputed. Contamination at the site and surrounding area was not caused by the gas leakage. The area around the plant was used as a dumping ground for hazardous chemicals and by 1982 water wells in the vicinity of the UCIL factory had to be abandoned. UCC states that "after the incident, UCIL began clean-up work at the site under the direction of Indian central and state government authorities", which was continued after 1994 by the successor to UCIL. The successor, Eveready Industries India, Limited (EIIL), ended cleanup on the site in 1998, when it terminated its 99-year lease and turned over control of the site to the state government of Madhya Pradesh.

UCC's laboratory tests in 1989 revealed that soil and water samples collected from near the factory were toxic to fish. Twenty-one areas inside the plant were reported to be highly polluted. In 1991 the municipal authorities declared that water from over 100 wells was hazardous for health if used for drinking. In 1994 it was reported that 21% of the factory premises were seriously contaminated with chemicals.

Beginning in 1999, studies made by Greenpeace and others from soil, groundwater, well water and vegetables from the residential areas around UCIL and from the UCIL factory area show contamination with a range of toxic heavy metals and chemical compounds. Substances found, according to the reports, are naphthol, naphthalene, Sevin, tarry residues, alpha naphthol, mercury, organochlorines, chromium, copper, nickel, lead, hexachlorethane, hexachlorobutadiene, pesticide HCH (BHC), volatile organic compounds and halo-organics. Many of these contaminants were also found in breast milk of women living near the area. Soil tests were conducted by Greenpeace in 1999. One sample (IT9012) from "sediment collected from drain under former Sevin plant" showed mercury levels to be at "20,000 and 6 million times" higher than expected levels.

Organochlorine compounds at elevated levels were also present in groundwater collected from (sample IT9040) a 4.4 meter depth "bore-hole within the former UCIL site". This sample was obtained from a source posted with a warning sign which read "Water unfit for consumption". Chemicals that have been linked to various forms of cancer were also discovered, as well as trichloroethylene, known to impair fetal development, at 50 times above safety limits specified by the U.S. Environmental Protection Agency (EPA).

In 2002, an inquiry by Fact-Finding Mission on Bhopal found a number of toxins, including mercury, lead, 1, 3, 5 trichlorobenzene, dichloromethane and chloroform, in nursing women's breast milk. A 2004 BBC Radio 5 broadcast reported the site was contaminated with toxic chemicals including benzene hexachloride and mercury, held in open containers or loose on the ground. A drinking water sample from a well near the site had levels of contamination 500 times higher than the maximum limits recommended by the World Health Organization. In 2009, the Centre for Science and Environment, a Delhi-based pollution monitoring lab, released test results showing pesticide groundwater contamination up to three kilometres from the factory. Also in 2009, the BBC took a water sample from a frequently used hand pump, located just north of the plant. The sample, tested in UK, was found to contain 1,000 times the World Health Organization's recommended maximum amount of carbon tetrachloride, a carcinogenic toxin. In October 2011, the Institute of Environmental Management and Assessment published an article and video by two British environmental scientists, showing the current state of the plant, landfill and solar evaporation ponds and called for renewed international efforts to provide the necessary skills to clean up the site and contaminated groundwater.

6.1.7. Conclusion

Since 1984, individual activists have played a role in the aftermath of the tragedy. Cooperation with international NGOs including Pesticide Action Network UK and Greenpeace started soon after the tragedy. One of the earliest reports is the Trade Union report from ILO 1985. More than 15 national organisations have been engaged along with a number of international organisations.

In the immediate aftermath of the disaster, Union Carbide states on its website that it put \$2 million into the Indian prime minister's immediate disaster relief fund on 11 December 1984. The corporation established the Employees' Bhopal Relief Fund in February 1985, which raised more than \$5 million for immediate relief. According to Union Carbide, in August 1987, they made an additional \$4.6 million in humanitarian interim relief available.

Union Carbide stated that it also undertook several steps to provide continuing aid to the victims of the Bhopal disaster. The hospital was begun in October 1995 and was opened in 2001. The company provided a fund with

around \$90 million from sale of its UCIL stock. In 1991, the trust had amounted to approximately \$100 million. The hospital catered for the treatment of heart, lung and eye problems. UCC also provided a \$2.2 million grant to Arizona State University to establish a vocational-technical center in Bhopal, which was opened, but was later closed by the state government. They also donated \$5 million to the Indian Red Cross after the disaster. They also developed a Responsible Care system with other members of the chemical industry as a response to the Bhopal crisis, which was designed to help prevent such an event in the future.

6.2. Case Study of Chernobyl Disaster, 1986

6.2.1. Introduction

The **Chernobyl disaster**, also referred to as the **Chernobyl accident**, was a catastrophic nuclear accident (Figure 6.5). It occurred on 26 April 1986 in the No.4 light water graphite moderated reactor at the Chernobyl Nuclear Power Plant near Pripyat, in what was then part of the Ukrainian Soviet Socialist Republic of the Soviet Union (USSR). It is considered the worst nuclear power plant disaster in history. A nuclear meltdown in one of the reactors caused a fire that sent a plume of radioactive fallout that eventually spread all over Europe.

On April 26, 1986, a test was scheduled at the Chernobyl Nuclear Power Plant to test a method of keeping the reactors properly cooled in the event of a power grid failure. If the test had gone as planned, the risk to the plant was very small. When things did go wrong, though, the potential for disaster was miscalculated and the test was continued even as serious problems arose. Meltdown occurred at 1:23 AM, starting a fire that dispersed large quantities of radioactive materials into the atmosphere. The amount of radioactive material released was 400 times more than the amount the atomic bombing of Hiroshima released. The fallout would be detected in almost all parts of Europe.



Figure 6.5. The meltdown of Chernobyl created the largest uncontrolled release of radioactive materials into the environment of any civilian operation in history, 1986. Source: Toxipedia

The Chernobyl accident dominates the Energy accidents sub-category, of most disastrous nuclear power plant accident in history, both in terms of cost and casualties. It is one of only two nuclear energy accidents classified as a level 7 event (the maximum classification) on the International Nuclear Event Scale, the other being the Fukushima Daiichi nuclear disaster in Japan in 2011. The struggle to safeguard against scenarios which were, at many times falsely, perceived as having the potential for greater catastrophe and the later decontamination efforts of the surroundings, ultimately involved over 500,000 workers and cost an estimated 18 billion rubles. During the accident, blast effects caused 2 deaths within the facility and later 29 firemen and employees died in the days-to-months afterward from acute radiation syndrome, with the potential for long-term cancers still being investigated.

The remains of the No.4 reactor building were enclosed in a large sarcophagus (radiation shield) by December 1986, at a time when what was left of the reactor was entering the cold shut-down phase; the enclosure was built quickly as occupational safety for the crews of the other undamaged reactors at the power station, with No.3 continuing to produce electricity into 2000.

The accident motivated safety upgrades on all remaining Soviet-designed reactors in the RBMK (Chernobyl No.4) family, of which eleven continued to power electric grids as of 2013.

6.2.2. Environmental effects (National and international spread of radioactive substances)

Approximately 100,000 km² of land was significantly contaminated with fallout, with the worst hit regions being in Belarus, Ukraine and Russia. Slighter levels of contamination were detected over all of Europe except for the Iberian Peninsula.

The initial evidence that a major release of radioactive material was affecting other countries came not from Soviet sources, but from Sweden. On the morning of 28 April workers at the Forsmark Nuclear Power Plant (approximately 1,100 km (680 mi) from the Chernobyl site) were found to have radioactive particles on their clothes.

It was Sweden's search for the source of radioactivity, after they had determined there was no leak at the Swedish plant, that at noon on 28 April led to the first hint of a serious nuclear problem in the western Soviet Union. Hence the evacuation of Prip'yat on 27 April, 36 hours after the initial explosions, was silently completed before the disaster became known outside the Soviet Union. The rise in radiation levels had at that time already been measured in Finland, but a civil service strike delayed the response and publication.

Contamination from the Chernobyl accident was scattered irregularly depending on weather conditions, much of it deposited on mountainous regions such as the Alps, the Welsh mountains and the Scottish Highlands, where adiabatic cooling caused radioactive rainfall. The resulting patches of contamination were often highly localized, and water-flows across the ground contributed further to large variations in radioactivity over small areas. Sweden and Norway also received heavy fallout when the contaminated air collided with a cold front, bringing rain.

Rain was purposely seeded over 10,000 km² of the Belorussian SSR by the Soviet air force to remove radioactive particles from clouds heading toward highly populated areas. Heavy, black-coloured rain fell on the city of Gomel. Reports from Soviet and Western scientists indicate that

Belarus received about 60% of the contamination that fell on the former Soviet Union. However, the 2006 TORCH report stated that half of the volatile particles had landed outside Ukraine, Belarus, and Russia. A large area in Russia south of Bryansk was also contaminated, as were parts of northwestern Ukraine. Studies in surrounding countries indicate that over one million people could have been affected by radiation.

In Western Europe, precautionary measures taken in response to the radiation included seemingly arbitrary regulations banning the importation of certain foods but not others. In France some officials stated that the Chernobyl accident had no adverse effects. Official figures in southern Bavaria in Germany indicated that some wild plant species contained substantial levels of caesium, which were believed to have been passed onto them during their consumption by wild boars, a significant number of which already contained radioactive particles above the allowed level.



Figure 6.6. Piglet with dipygus on exhibit at the Ukrainian National Chernobyl Museum. Source: Wikipedia

Mutations in both humans and other animals increased following the disaster. On farms in Narodychi Raion of Ukraine, for instance, in the first four years of the disaster nearly 350 animals were born with gross deformities such as missing or extra limbs, missing eyes, heads or ribs, or deformed skulls; in comparison, only three abnormal births had been registered in the five years prior (Figure 6.6). Despite these claims, the World Health Organization states, "children conceived before or after their father's exposure showed no statistically significant differences in mutation frequencies".

6.2.3. Residual radioactivity in the environment rivers, lakes and reservoirs

Like many other releases of radioactivity into the environment, the Chernobyl release was controlled by the physical and chemical properties of the radioactive elements in the core. Particularly dangerous are the highly radioactive fission products, those with high nuclear decay rates that accumulate in the food chain, such as some of the isotopes of iodine, caesium and strontium. Iodine-131 and caesium-137 are responsible for most of the radiation exposure received by the general population.

The Chernobyl nuclear power plant is located next to the Pripyat River, which feeds into the Dnieper reservoir system, one of the largest surface water systems in Europe, which at the time supplied water to Kiev's 2.4 million residents, and was still in spring flood when the accident occurred. The radioactive contamination of aquatic systems therefore became a major problem in the immediate aftermath of the accident. In the most affected areas of Ukraine, levels of radioactivity (particularly from radionuclides I, Cs and Sr) in drinking water caused concern during the weeks and months after the accident, though officially it was stated that all contaminants had settled to the bottom "in an insoluble phase" and would not dissolve for 800–1000 years. Guidelines for levels of radioiodine in drinking water were temporarily raised to 3,700 Bq/L, allowing most water to be reported as safe, and a year after the accident it was announced that even the water of the Chernobyl plant's cooling pond was within acceptable norms. Despite this, two months after the disaster the Kiev water supply was abruptly switched from the Dnieper to the Desna River. Meanwhile, massive silt traps were constructed, along with an enormous 30m-deep underground barrier to prevent groundwater from the destroyed reactor entering the Pripyat River.

Bio-accumulation of radioactivity in fish resulted in concentrations (both in western Europe and in the former Soviet Union) that in many cases were significantly above guideline maximum levels for consumption. Guideline maximum levels for radiocaesium in fish vary from country to country but are approximately 1000 Bq/kg in the European Union. In the Kiev Reservoir in Ukraine, concentrations in fish were several thousand Bq/kg during the years after the accident.

6.2.4. Groundwater contamination

Groundwater was not badly affected by the Chernobyl accident since radionuclides with short half-lives decayed away long before they could affect groundwater supplies, and longer-lived radionuclides such as radiocaesium and radiostrontium were absorbed to surface soils before they could transfer to groundwater. However, significant transfers of radionuclides to groundwater have occurred from waste disposal sites in the 30 km (19 mi) exclusion zone around Chernobyl. Although there is a potential for transfer of radionuclides from these disposal sites off-site (i.e. out of the 30 km (19 mi) exclusion zone), the IAEA Chernobyl Report argues that this is not significant in comparison to current levels of washout of surface-deposited radioactivity.

6.2.5. Flora and fauna

After the disaster, four square kilometres of pine forest directly downwind of the reactor turned reddish-brown and died, earning the name of the "Red Forest". Some animals in the worst-hit areas also died or stopped reproducing. Most domestic animals were removed from the exclusion zone, but horses left on an island in the Pripyat River 6 km (4 mi) from the power plant died when their thyroid glands were destroyed by radiation doses of 150–200 Sv. Some cattle on the same island died and those that survived were stunted because of thyroid damage. The next generation appeared to be normal.

6.2.6. Human impact

In the aftermath of the accident, 237 people suffered from acute radiation sickness (ARS), of whom 31 died within the first three months. Most of the victims were fire and rescue workers trying to bring the accident under control, who were not fully aware of how dangerous the exposure to radiation in the smoke was.

In 2005 the Chernobyl Forum, composed of the IAEA, other UN organizations and the governments of Belarus, Russia and Ukraine, published a report on the radiological environmental and health consequences of the Chernobyl accident. On the death toll of the accident, the report states that 28 emergency workers ("liquidators") died from acute radiation syndrome including beta burns and 15 patients died from thyroid cancer in the following years, and it roughly estimated that cancer deaths caused by Chernobyl may reach a total of about 4,000 among the 5 million persons

residing in the contaminated areas, the report projected cancer mortality "increases of less than one per cent" (~0.3%) on a time span of 80 years, cautioning that this estimate was "speculative" since at this time only a few cancer deaths are linked to the Chernobyl disaster. The report says it is impossible to reliably predict the number of fatal cancers arising from the incident as small differences in assumptions can result in large differences in the estimated health costs. The report says it represents the consensus view of the eight UN organizations.

Of all 66,000 Belarusian emergency workers, by the mid-1990s only 150 (roughly 0.2%) were reported by their government as having died. In contrast, 5,722 casualties were reported among Ukrainian clean-up workers up to the year 1995, by the National Committee for Radiation Protection of the Ukrainian Population.

The four most harmful radionuclides spread from Chernobyl were iodine-131, caesium-134, caesium-137 and strontium-90, with half-lives of 8.02 days, 2.07 years, 30.2 years and 28.8 years respectively. The iodine was initially viewed with less alarm than the other isotopes, because of its short half-life, but it is highly volatile, and now appears to have travelled furthest and caused the most severe health problems in the short term. Strontium, on the other hand, is the least volatile of the four, and of main concern in the areas near Chernobyl itself. Iodine tends to become concentrated in thyroid and milk glands, leading, among other things, to increased incidence of thyroid cancers. Caesium tends to accumulate in vital organs such as the heart, while strontium accumulates in bones, and may thus be a risk to bone-marrow and lymphocytes. Radiation is most damaging to cells that are actively dividing. In adult mammals cell division is slow, except in hair follicles, skin, bone marrow and the gastrointestinal tract, which is why vomiting and hair loss are common symptoms of acute radiation sickness.

6.2.7. Thyroid cancer

Thyroid cancer incidence in children and adolescents from Belarus were reported after the Chernobyl accident. The 2005 Chernobyl Forum report revealed thyroid cancer among children to be one of the main health impacts from the Chernobyl accident. In that publication more than 4000 cases were reported, and that there was no evidence of an increase in solid cancers or leukemia. It said that there was an increase in psychological problems among

the affected population. Dr Michael Repacholi, manager of WHO's Radiation Program, reported that the 4000 cases of thyroid cancer resulted in nine deaths.

According to United Nation Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), up to the year 2005, an excess of over 6,000 cases of thyroid cancer have been reported. That is, over the estimated pre-accident baseline thyroid cancer rate, more than 6,000 casual cases of thyroid cancer have been reported in children and adolescents exposed at the time of the accident, a number that is expected to increase. They concluded that there was no other evidence of major health impacts from the radiation exposure.

Well-differentiated thyroid cancers are generally treatable, and when treated the five-year survival rate of thyroid cancer is 96%, and 92% after 30 years. UNSCEAR had reported 15 deaths from thyroid cancer in 2011. The International Atomic Energy Agency (IAEA) also states that there has been no increase in the rate of birth defects or abnormalities, or solid cancers (such as lung cancer) corroborating UNSCEAR's assessments. UNSCEAR does raise the possibility of long-term genetic defects, pointing to a doubling of radiation-induced mini satellite mutations among children born in 1994. However, the risk of thyroid cancer associated with the Chernobyl accident is still high according to published studies.

The German affiliate of the International Physicians for the Prevention of Nuclear War (IPPNW) argued that more than 10,000 people are today affected by thyroid cancer and 50,000 cases are expected in the future.

6.2.8. Other health disorders

Fred Mettler, a radiation expert at the University of New Mexico, puts the number of worldwide cancer deaths outside the highly contaminated zone at "perhaps" 5000, for a total of 9000 Chernobyl-associated fatal cancers, saying "the number is small (representing a few percent) relative to the normal spontaneous risk of cancer, but the numbers are large in absolute terms". The same report outlined studies based in data found in the Russian Registry from 1991 to 1998 that suggested that "of 61,000 Russian workers exposed to an average dose of 107 mSv about 5% of all fatalities that occurred may have been due to radiation exposure."

The report went into depth about the risks to mental health of exaggerated fears about the effects of radiation. According to the IAEA the "designation of the affected population as "victims" rather than "survivors" has led them to perceive themselves as helpless, weak and lacking control over their future". The IAEA says that this may have led to behaviour that has caused further health effects. Fred Mettler commented that 20 years later: "The population remains largely unsure of what the effects of radiation actually are and retain a sense of foreboding. A number of adolescents and young adults who have been exposed to modest or small amounts of radiation feel that they are somehow fatally flawed and there is no downside to using illicit drugs or having unprotected sex. To reverse such attitudes and behaviours will likely take years although some youth groups have begun programs that have promise. " In addition, disadvantaged children around Chernobyl suffer from health problems that are attributable not only to the Chernobyl accident, but also to the poor state of post-Soviet health systems.

The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), part of the Chernobyl Forum, have produced their own assessments of the radiation effects. UNSCEAR was set up as a collaboration between various United Nation bodies, including the World Health Organization, after the atomic bomb attacks on Hiroshima and Nagasaki, to assess the long-term effects of radiation on human health.

6.2.9. Chernobyl human mutations

At the time of the Chernobyl radiation disaster, Reactor 4 had 180 metric tons of uranium dioxide, out of which anywhere from 6 to 30% was released into the air and the surrounding environment. There was nothing to hold it in, no protection from escape when the fire erupted. The fuel that was in the reactor vaporized and flew into the surrounding air and land. It spread quickly through the atmosphere. The radiation that leaked after the explosion still harmed people and Chernobyl animals as well as plants that were in the area.

The radiation that leaked after the explosion still harmed people and Chernobyl animals as well as plants that were in the area. There were many

patients who were treated for diseases such as thyroid cancer, leukemia and also respiratory illnesses.

During the year 1986, the year of the Chernobyl Power Plant Disaster, the number of babies born with birth defects significantly increased by a rate of 200%. The number of those that were reported increased more than this and there were probably more that may not have been reported. Because their defects were because of genetic mutations, they are likely to pass it on to other generations. This leads to more birth defects and possibly more Chernobyl mutations from radiation (Figure 6.7 and 6.8).

For the mothers in the early stages of pregnancy, the infants suffered the effects of radiation poisoning. Some were simply born with cosmetic deformities. Others had organs grow in the wrong spot or potentially not at all. The mothers may have felt fine and appeared to be healthy, but that doesn't mean they weren't at risk for passing on new genetic mutations. Chernobyl child mutations include the fact that some children were born with heart defects caused by genetic mutation from the radiation.



Figure 6.7. Birth defects and Chernobyl mutations from radiation, Chernobyl Power Plant Disaster.
Source: Wikipedia



**Figure 6.8. Deformed child nicknamed 'the Chernobyl Child' in a special school for abandoned children in Belarus.
Photograph: Igor Kostin/Corbis (1988)**

6.2.10. Deaths due to radiation exposure

The number of potential deaths arising from the Chernobyl disaster is heavily debated. The WHO's prediction of 4,000 future cancer deaths in surrounding countries is based on the Linear no-threshold model (LNT), which assumes that the damage inflicted by radiation at low doses is directly proportional to the dose. Radiation epidemiologist Roy Shore contends that estimating health effects in a population from the LNT model "is not wise because of the uncertainties".

According to the Union of Concerned Scientists the number of excess cancer deaths worldwide (including all contaminated areas) is approximately 27,000 based on the same LNT.

Another study critical of the Chernobyl Forum report was commissioned by Greenpeace, which asserted that the most recently published figures indicate that in Belarus, Russia and Ukraine the accident could have resulted in 10,000–200,000 additional deaths in the period between 1990 and 2004. The Scientific Secretary of the Chernobyl Forum criticized the report's reliance on non-peer-reviewed locally produced studies. Although most of the study's sources were from peer-reviewed journals, including many Western medical journals, the higher mortality estimates were from non-peer-reviewed

sources, while Gregory Härtl (spokesman for the WHO) suggested that the conclusions were motivated by ideology.

Chernobyl: Consequences of the Catastrophe for People and the Environment is an English translation of the 2007 Russian Publication. It was published in 2009 by the New York Academy of Sciences in their *Annals of the New York Academy of Sciences*. It presents an analysis of scientific literature and concludes that medical records between 1986, the year of the accident, and 2004 reflect 985,000 premature deaths as a result of the radioactivity released. Though, it was impossible to precisely determine what dose the affected people received, knowing the fact that the received doses varied strongly from one individual to the other in the population above which the radioactive cloud travelled, and also knowing the fact that one cannot tell for sure if a cancer in an individual from the former USSR is produced by radiation from Chernobyl accident or by other social or behavioural factors, such as smoking or alcohol drinking.

6.2.11. Abortion requests

Following the accident, journalists mistrusted many medical professionals (such as the spokesman from the UK National Radiological Protection Board), and in turn encouraged the public to mistrust them. Throughout the European continent, in nations where abortion is legal, many requests for induced abortions, of otherwise normal pregnancies, were obtained out of fears of radiation from Chernobyl, including an excess number of abortions in Denmark in the months following the accident. In Greece, following the accident many obstetricians were unable to resist requests from worried pregnant mothers over fears of radiation. Although it was determined that the effective dose to Greeks would not exceed 1 mSv (100 mrem), a dose much lower than that which could induce embryonic abnormalities or other non-stochastic effects, there was an observed 2,500 excess of otherwise wanted pregnancies being terminated, probably out of fear in the mother of radiation risk. A "slightly" above the expected number of requested induced abortions occurred in Italy.

Worldwide, an estimated excess of about 150,000 elective abortions may have been performed on otherwise healthy pregnancies out of unfounded fears of radiation from Chernobyl, according to Dr Robert Baker and ultimately a 1987 article published by Linda E. Ketchum in the *Journal of*

Nuclear Medicine which mentions but does not reference an IAEA source on the matter.

The available statistical data excludes the Soviet/Ukraine/Belarus abortion rates, as they are presently unavailable. From the available data, an increase in the number of abortions in what were healthy developing human offspring in Denmark occurred in the months following the accident, at a rate of about 400 cases. In Greece, there was an observed 2,500 excess of otherwise wanted pregnancies being terminated. In Italy, a "slightly" above the expected number of induced abortions occurred, approximately 100.

6.2.12. Radioactive materials and waste management

As of 2006, some fuel remained in the reactors at units 1 through 3, most of it in each unit's spent fuel pool, as well as some material in a small spent fuel interim storage facility pond (ISF-1).

In 1999 a contract was signed for construction of a radioactive waste management facility to store 25,000 used fuel assemblies from units 1–3 and other operational wastes, as well as material from decommissioning units 1–3 (which will be the first RBMK units decommissioned anywhere). The contract included a processing facility able to cut the RBMK fuel assemblies and to put the material in canisters, which were to be filled with inert gas and welded shut.

The canisters were to be transported to dry storage vaults, where the fuel containers would be enclosed for up to 100 years. This facility, treating 2,500 fuel assemblies per year, would be the first of its kind for RBMK fuel. However, after a significant part of the storage structures had been built, technical deficiencies in the concept emerged, and the contract was terminated in 2007. The interim spent fuel storage facility (ISF-2) will now be completed by others by mid-2013.

Another contract has been let for a liquid radioactive waste treatment plant, to handle some 35,000 cubic meters of low- and intermediate-level liquid wastes at the site. This will need to be solidified and eventually buried along with solid wastes on site.

6.2.13. Conclusion

The Chernobyl Shelter Fund was established in 1997 at the Denver 23rd G8 summit to finance the Shelter Implementation Plan (SIP). The plan calls for transforming the site into an ecologically safe condition by means of stabilization of the sarcophagus followed by construction of a New Safe Confinement (NSC). While the original cost estimate for the SIP was US\$768 million, the 2006 estimate was \$1.2 billion. The SIP is being managed by a consortium of Bechtel, Battelle, and Électricité de France, and conceptual design for the NSC consists of a movable arch, constructed away from the shelter to avoid high radiation, to be slid over the sarcophagus. The NSC is expected to be completed in 2015, and will be the largest movable structure ever built.

The United Nations Development Programme has launched in 2003 a specific project called the Chernobyl Recovery and Development Programme (CRDP) for the recovery of the affected areas. The programme was initiated in February 2002 based on the recommendations in the report on Human Consequences of the Chernobyl Nuclear Accident. The main goal of the CRDP's activities is supporting the Government of Ukraine in mitigating long-term social, economic, and ecological consequences of the Chernobyl catastrophe. CRDP works in the four most Chernobyl-affected areas in Ukraine: Kyivska, Zhytomyrska, Chernihivska and Rivnenska.

The International Project on the Health Effects of the Chernobyl Accident (IPEHCA) was created and received US \$20 million, mainly from Japan, in hopes of discovering the main cause of health problems due to radiation. These funds were divided between Ukraine, Belarus, and Russia, the three main affected countries, for further investigation of health effects. As there was significant corruption in former Soviet countries, most of the foreign aid was given to Russia, and no positive outcome from this money has been demonstrated.

Chernobyl Children International (CCI) is a United Nations accredited, non-profit, international development, medical, and humanitarian organization that works with children, families and communities that continue to be affected by the Chernobyl nuclear disaster. The organization's founder and chief executive is Adi Roche, the Irish humanitarian and peace campaigner. The CCI was founded in 1991 in response to an appeal

from Ukrainian and Belarusian doctors for aid. Roche then began organizing 'rest and recuperation' holidays for a few Chernobyl children. Recruiting Irish families who would welcome and care for them, CCI expanded into the United States in 2001.

Over its lifetime, the organization has grown in strength and numbers. It works closely with the Belarusian government, the United Nations, and many thousands of volunteers worldwide to deliver a broad range of supports to the children and the wider community. It also acts as an advocate for the rights of those affected by the Chernobyl explosion, and engages in research and outreach activities to encourage the rest of the world to remember the victims and understand the long-term impact on their lives.

6.3. Case Study of the Seveso Disaster, 1976

6.3.1. Introduction

The **Seveso disaster** was an industrial accident that occurred around 12:37 pm on July 10, 1976, in a small chemical manufacturing plant approximately 20 kilometres (12 mi) north of Milan in the Lombardy region of Italy (Figure 6.9). It resulted in the highest known exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in residential populations, which gave rise to numerous scientific studies and standardized industrial safety regulations. The EU industrial safety regulations are known as the Seveso II Directive.



Figure 6.9. Industrial accident occurred at Pesticide plant in the town of Seveso, 1976. Source: Toxipedia

The Seveso disaster was named because Seveso, with a population of 17,000 in 1976, was the community most affected. Other affected neighbouring communities were Meda (19,000), Desio (33,000), Cesano Maderno (34,000) and to a lesser extent Barlassina (6,000) and Bovisio-Masciago (11,000). The industrial plant, located in Meda, was owned by the company ICMESA (Industrie Chimiche Meda Società Azionaria), a subsidiary of Givaudan, which in turn was a subsidiary of Hoffmann-La Roche (Roche Group). The factory building had been built many years earlier and the local population did not perceive it as a potential source of danger. Moreover, although several exposures of populations to dioxins had occurred before, mostly in industrial accidents, they were of a more limited scale.

6.3.2. Immediate health effects

The affected area was split into zones A, B and R in decreasing order of surface soil concentrations of TCDD. Zone A was further split into 7 sub-zones. The local population was advised not to touch or eat locally grown fruits or vegetables.

At first, the effect of the acrid cloud of dioxin on the inhabitants of Seveso was to create nausea, dizziness, headaches, and burn-like sores on the skin. Later in the day, birds, chickens, and domestic animals such as cats and rabbits began to sicken and die. One man reported that when he tried to pick up his dying cat, its tail fell off. Some eighty-one domestic animals died in the contamination or were slaughtered as a health precaution.

The long-term medical effects of the dioxin contamination are uncertain. One hundred and eighty-seven confirmed cases of the skin condition chloracne (a rash of acne-like blackheads, cysts, and pustules) were confirmed, but most of these cleared up after treatment. Thirty-five percent of the people in the northern part of the contaminated zone (i.e., the part closest to the ICMESA plant) were found to have enlarged livers, which were regarded as important, because dioxin is especially harmful to the liver. One health concern was dioxin's well-known effect of deforming the fetuses of animals. A herd of forty cows was being bred near the ICMESA plant, and there were ten spontaneous abortions among the thirteen cows that became pregnant. Of the three cows that carried their pregnancies to term, only one had a calf that lived very long after it was born. The fear of birth defects

caused thirty-four women to obtain therapeutic abortions, with the permission of the Catholic Church. One estimate said that another 120 women in the area may have had abortions without the permission of the church. The fear of birth defects has proven justified. Babies born with birth defects in the Seveso area in 1975, the year before the contamination, numbered three. In 1978, they totaled fifty-three.

On July 25, 1976, over two weeks after the dioxin contamination, the Seveso regional authorities finally ordered the evacuation of 739 residents from the 253 acres (102 hectares) immediately adjacent to the ICMESA plant. Their evacuation took place from July 26 to August 2. Unfortunately, the evacuees took with them their cars, trucks, furniture, and bicycles that were certain to have been contaminated with dioxin. A fence nine feet (2.7 meters) high and four miles (6.4 kilometers) long was eventually built around the contaminated 253 acres. Another area of 665 acres (269 hectares) with a population of five thousand people was sealed off from nonresidents, but the residents were allowed to stay and to come and go as they pleased.

The process of decontamination was only partially effective, and dioxin spread around the neighboring regions. After the evacuation, teams of decontamination workers began to remove the carcasses of dead animals, as well as trees, brush, other vegetation, and eight inches (20 centimeters) of the topsoil in many areas of the 253 acres of land adjacent to the ICMESA plant. But some decontamination measures only spread the dioxin around. As topsoil was bulldozed in the contaminated area, clouds of dust were raised and dioxin was blown away to yet other adjacent areas. As people tracked in and out of the contaminated area to remove their belongings, they spread the dioxin. Heavy and continuous rains in October of 1976 flooded the most polluted part of Seveso, washing dioxin and contaminated soil away. In Milan, thirteen miles (21 kilometers) south of Seveso, dioxin traces have been found in the mud. In the summer of 1977, over one hundred schools in the areas around Seveso were found to be contaminated with low levels of dioxin.

The owners of the ICMESA factory, the Hoffmann-La Roche pharmaceutical company, set aside over US\$134 million to pay for the dioxin contamination. Some evacuees were paid to have their houses completely renovated. Others received the equivalent of two years' salary. Because the

legal system in Italy moves very slowly, it was almost seven years; that is, April of 1983, before a criminal case against five former employees of the ICMESA plant was begun. In an odd twist, one supervisor at the plant was murdered by terrorists before he could be prosecuted. Other legal cases against Hoffmann-La Roche were also pending at that time.

The accident at Seveso had repercussions throughout the chemical industry. After the dioxin contamination, plans to construct several chemical plants in Italy were halted and a plant that manufactured trichlorophenol in Coalite, England, was shut down. In addition, because the main cause of the Seveso dioxin contamination was worker error, authorities proposed that companies educate workers about mandatory procedures and the consequences of failing to follow them. One expert also advised that future trichlorophenol manufacturing plants should have total containment systems that would not vent any expanding gases to the outside environment.

A number of pollution regulations were developed in Europe after the Seveso accident. Companies have been required to disclose any dangerous processes used at their factories, and regulations have been developed to limit the transport of toxic wastes across European national boundaries. These latter regulations have made it very difficult for Hoffmann-La Roche to dispose of the dioxin collected during its cleanup activities. The company was again embarrassed by its handling of this problem when it was discovered that forty-one barrels of waste material contaminated with dioxin from Seveso had been located in an unused slaughterhouse close to the middle of Paris, France. The company had hired an Italian subsidiary of a German engineering company to arrange for disposal, and it, in turn, subcontracted with a French individual who drove from Seveso to Paris in 1983 with the barrels. After the disclosure of this cross-country violation, the waste was later incinerated at a disposal plant in Switzerland.

The authorities in the Seveso area mishandled the accident. First, they were slow to react to the accident and took their time letting people know that there had been a problem. Without this delay, the residents of the Seveso area might have evacuated in time to escape harm. Furthermore, the cleanup process progressed slowly and sloppily. The contaminated zone was neither protected nor policed during this period, and many of the vacated houses were

vandalized. After frequent delays and much conflicting information, evacuees became homesick and started pressuring the authorities to let them return to their land. The authorities partially relented in the fall of 1977, and some of the evacuees were allowed to return to their homes in the cleaned-up areas of the contaminated zone. The area immediately surrounding the factory was designated Zone A, and no one was allowed to return, but were settled later in Zones B and R. In 1989, a team of researchers published a study describing increased rates of cardiovascular deaths among Seveso residents. In 1993, that same team published another study stating that people living in contaminated areas around Seveso have an elevated risk for several types of cancer.

The disaster at Seveso, Italy, produced numerous studies by the scientific community. It also resulted in many industrial safety regulations directed toward the chemical industry. In the European Union, the industrial safety regulations instituted on December 9, 1996, are called the Council Directive 96/82/EC, or commonly called the Seveso II Directive. The law controls the hazards involving major accidents of dangerous substances.

Within days a total of 3,300 animals, mostly poultry and rabbits, were found dead. Emergency slaughtering commenced to prevent TCDD from entering the food chain, and by 1978 over 80,000 animals had been slaughtered. 15 children were quickly hospitalised with skin inflammation. By the end of August, Zone A had been completely evacuated and fenced, 1,600 people of all ages had been examined and 447 were found to suffer from skin lesions or chloracne. An advice center was set up for pregnant women of which only 26 opted for an abortion, which was legal in special cases, after consultation. Another 460 women brought on their pregnancies without problems, their children not showing any sign of malformation or pathologies. Herwig von Zwehl (Technical Director of ICMESA) and Paolo Paoletti (director of production at ICMESA) were arrested. Two government commissions were established to develop a plan for quarantining and decontaminating the area, for which the Italian government allotted 40 billion lire (US \$47.8 million). This amount would be tripled two years later.

6.3.3. Long-term health effects

A 1991 study 14 years after the accident sought to assess the effects to the thousands of persons that had been exposed to dioxin. The most evident adverse health effect ascertained was chloracne (193 cases). Other early effects noted were peripheral neuropathy and liver enzyme induction. The ascertainment of other, possibly severe sequelae of dioxin exposure (e.g., birth defects) was hampered by inadequate information; however, generally, no increased risks were evident.

A study published in 1998 concluded that chloracne (nearly 200 cases with a definite exposure dependence) was the only effect established with certainty. Early health investigations including liver function, immune function, neurologic impairment, and reproductive effects yielded inconclusive results.

An excess mortality from cardiovascular and respiratory diseases was uncovered, and excess of diabetes cases was also found. Results of cancer incidence and mortality follow-up showed an increased occurrence of cancer of the gastrointestinal sites and of the lymphatic and hematopoietic tissue. Results cannot be viewed as final or comprehensive, however, because of various limitations: the lack of individual exposure data, short latency period, and small population size for certain cancer types.

A 2001 study confirmed in victims of the disaster, that dioxin is carcinogenic to humans and corroborate its association with cardiovascular- and endocrine-related effects. In 2009, an update including 5 more years (up to 1996) found an increase in "lymphatic and hematopoietic tissue neoplasms" and increased breast cancer.

The male children of mothers who were, during pregnancy of those children, exposed to high levels of toxic dioxins due to the Seveso disaster, have been found to have lower-than-average sperm counts. This result of the underlying Seveso study has been noted to provide the most pronounced evidence for prenatal exposure to an environmental chemical causing low sperm counts.

6.3.4. Conclusion

The safety operations handled by the company's directors and local government were well coordinated, but the ICMESA refused to admit immediately the incident. At least a week passed before it was publicly stated that dioxin had been emitted and another week passed before evacuation

began (the government had to control which area was mostly polluted and, after that, to organise everything for the evacuated people). Even worse, the factory's safety measures weren't created to preserve also the external environment. As a result, the local population was caught unaware when the accident happened and unprepared to cope with the danger of an invisible poison.

In the context of such heightened tensions, Seveso became a microcosm where all the existing conflicts within society (political, institutional, religious, industrial) were reflected. However, within a relatively short time such conflicts abated and the recovery of the community proceeded. For, in Seveso, the responsible party was known from the outset and soon offered reparation. Moreover, the eventual disappearance of the offending factory itself and the physical exportation of the toxic substances and polluted soil enabled the community to feel cleansed. The resolution of the emotional after-effects of the trauma, so necessary for the recovery of a community, was facilitated by these favourable circumstances."

Industrial safety regulations were passed in the European Community in 1982 called the Seveso Directive which imposed much harsher industrial regulations. The Seveso Directive was updated in 1996, amended lastly in 2008 and is currently referred to as the Seveso II Directive (or COMAH Regulations in the United Kingdom).

Treatment of the soil in the affected areas was almost perfect; the area now has a dioxin level below the normal level. The whole site has been turned into a public park, Seveso Oak Forest park. This is a really important place for the inhabitants of Seveso, and it's a protected area, where it's forbidden to build anything (roads, buildings...). There are two artificial hills in the park; today, under these hills there is all what remains of the toxic area (destroyed houses, tons of poisoned dirt, animal corpses...), protected in a cement sarcophagus. Some inspections under them declared the sarcophagus has to resist for almost 300 years before the toxic substances will vanish.

Several studies have been completed on the health of the population of surrounding communities. While it has been established that people from Seveso exposed to TCDD are more susceptible to certain rare cancers, when all types of cancers are grouped into one category, no statistically significant excess has yet been observed. This indicates that more research is needed to determine the true long-term health effects on the affected population.

6.4. Case Study of Love Canal Toxic Waste Disaster, 1978

6.4.1. Introduction

One of the most famous and important examples of groundwater pollution in the U.S. was the Love Canal tragedy in Niagara Falls, New York. It is important because the pollution disaster at Love Canal, along with similar pollution calamities at that time (Times Beach, Missouri and Valley of Drums, Kentucky), helped to create **Superfund**, a federal program instituted in 1980 and designed to identify and clean up the worst of the hazardous chemical waste sites in the U.S.

Love Canal is a neighborhood in Niagara Falls named after a large ditch (approximately 15 m wide, 3–12 m deep, and 1600 m long) that was dug in the 1890s for hydroelectric power. The ditch was abandoned before it actually generated any power and went mostly unused for decades, except for swimming by local residents. In the 1920s Niagara Falls began dumping urban waste into Love Canal, and in the 1940s the U.S. Army dumped waste from World War II there, including waste from the frantic effort to build a nuclear bomb. Hooker Chemical purchased the land in 1942 and lined it with clay. Then, the company put into Love Canal an estimated 21,000 tons of hazardous chemical waste, including the carcinogens benzene, dioxin, and PCBs in large metal barrels and covered them with more clay (Figure 6.10). In 1953, Hooker sold the land to the Niagara Falls school board for \$1, and included a clause in the sales contract that both described the land use (filled with chemical waste) and absolved them from any future damage claims from the buried waste. The school board promptly built a public school on the site and sold the surrounding land for a housing project that built 200 or so homes along the canal banks and another 1,000 in the neighborhood. During construction, the canal's clay cap and walls were breached, damaging some of the metal barrels.



Figure 6.10. 21,000 tons of hazardous chemical waste put into Love canal. Important examples of groundwater pollution in the U.S. was the Love Canal tragedy in Niagara Falls, New York. Source: US Environmental Protection Agency

Eventually, the chemical waste seeped into people's basements, and the metal barrels worked their way to the surface. Trees and gardens began to die; bicycle tires and the rubber soles of children's shoes disintegrated in noxious puddles. From the 1950s to the late 1970s, residents repeatedly complained of strange odors and substances that surfaced in their yards. Local residents allegedly experienced major health problems including high rates of miscarriages, birth defects, and chromosome damage, but studies by the New York State Health Department disputed that.

Finally, in 1978 President Carter declared a state of emergency at Love Canal, making it the first human-caused environmental problem to be designated that way. The Love Canal incident became a symbol of improperly stored chemical waste. Clean up of Love Canal, which was funded by Superfund and completely finished in 2004, involved removing contaminated soil, installing drainage pipes to capture contaminated groundwater for treatment, and covering it with clay and plastic. In 1995, Occidental Chemical (the modern name for Hooker Chemical) paid \$102 million to Superfund (A federal program created in 1980 and designed to identify and clean up the worst of the hazardous chemical waste sites in the U.S.) for cleanup and \$27 million to Federal Emergency Management Association for the relocation of more than 1,000 families. New York State paid \$98 million to EPA and the

US government paid \$8 million for pollution by the Army. The total clean up cost was estimated to be \$275 million. The only good thing about the Love Canal tragedy is that it helped to create Superfund, which has analyzed tens of thousands of hazardous waste sites in the U.S. and cleaned up hundreds of the worst ones. Nevertheless, over 1,000 major hazardous waste sites with a significant risk to human health or the environment are still in the process of being cleaned.

6.4.2. Contaminants

Numerous contaminants dumped in the landfill included chlorinated hydrocarbon residues, processed sludge, fly ash, and other materials, including residential municipal garbage. Data showed unacceptable levels of toxic vapors associated with more than 80 compounds were emanating from the basements of numerous homes in the first ring directly adjacent to the Love Canal. Ten of the most prevalent and most toxic compounds - including benzene, a known human carcinogen - were selected for evaluation purposes and as indicators of the presence of other chemical constituents.

Laboratory analyses of soil and sediment samples from the Love Canal indicate the presence of more than 200 distinct organic chemical compounds; approximately 100 of these have been identified to date. Numerous other chemicals seeped through the ground. Some of the chemicals and toxic materials found included Benzene, chloroform, toluene, Dioxin, and various kinds of PCB (Table 6.1).

6.4.3. Health effects

At first, scientific studies did not conclusively prove the chemicals were responsible for the residents' illnesses yet scientists were divided on the issue, even though eleven known or suspected carcinogens had been identified, one of the most prevalent being benzene. Also present was dioxin (polychlorinated dibenzodioxins) in the water, a very hazardous substance. Dioxin pollution is usually measured in parts per trillion; at Love Canal, water samples showed dioxin levels of 53 parts per billion. Geologists were recruited to determine whether underground swales were responsible for carrying the chemicals to the surrounding residential areas. Once there, chemicals could leach into basements and evaporate into household air.

Table 6.1. Contaminants dumped in the landfill at the Love Canal Site (1979). (Draft Report on Hazardous Waste Disposal in Erie and Niagara Counties, New York, March 1979)

Type of Waste	Physical State	Total Estimated Quantity-Tons	Container
Misc. acid chlorides other than benzoyl - includes acetyl, caprylyl, butyryl, nitro benzoyls	liquid and solid	400	drum
Thionyl chloride and misc. sulfur/chlorine compounds	liquid and solid	500	drum
Misc. chlorination - includes waxes, oils, naphthenes, aniline	liquid and solid	1,000	drum
Dodecyl (Lauryl, Lorol) mercaptans (DDM), chlorides and misc. organic sulfur compounds	liquid and solid	2,400	drum
Trichlorophenol (TCP)	liquid and solid	200	drum
Benzoyl chlorides and benzo- trichlorides	liquid and solid	800	drum
Metal chlorides	solid	400	drum
Liquid disulfides (LDS/LDSN/BDS) and chlorotoluenes	liquid	700	drum

Hexachlorocyclohexane (Lindane/BHC)	solid	6,900	drum and nonmetallic containers
Chlorobenzenes	liquid and solid	2,000	drum and nonmetallic containers
Benzylchlorides - includes benzyl chloride, benzyl alcohol, benzyl thiocyanate	solid	2,400	drum
Sodiumsulfide/sulfhydrates	solid	2,000	drum

The ultimate goal of the Health Department's long-range epidemiologic investigation is to obtain a detailed health profile of all persons who presently or ever lived near the Love Canal landfill to determine whether these individuals are at higher risk for acute and/or chronic health disorders.

Miscarriages and birth defects are considered prime indicators of human toxicity since recent studies in developmental pharmacology establish that the prenatal period is characterized by a unique susceptibility to certain chemical agents. In addition, several known or suspected teratogens (producers of physical defects in fetuses) have been identified among the chemicals dumped in the Love Canal area

Liver function, as determined through blood analysis, was chosen as a factor for immediate investigation because current experimental studies suggest that many of the chemical agents identified at the site may play a role in development of cancer or direct injury to the liver. Analyses of the 2,800 blood samples taken to date have been completed and all individuals have been notified of test results via their private physicians. No conclusions relative to residence on the Canal can be drawn at this time with regard to the significance of minor abnormalities detected. Efforts will be made to confirm and more fully investigate abnormal test results.

Since mercury is an established teratogen and is readily identifiable in blood samples, blood mercury determinations were conducted on some area residents during the early investigative stage. Results of all mercury tests performed were within normal limits. The initial epidemiologic investigation was based on historical information and blood test results from the ninety-seven families in the first ring of homes bordering directly on the Love Canal site. The families comprised 230 adults (18 years of age or older) and 134 children. General health information was obtained from 97 percent of the adults and 92 percent of the children.

All reported birth defects were confirmed through medical records, and the past medical and drug histories of the mothers were evaluated for possible confounding influences. Reported miscarriages also were confirmed through private physicians' and hospital records. Following is a list of some of the more important chemicals identified at the Love Canal site and the human biologic hazards associated with them (Table 6.2).

Table 6.2. List of some chemicals identified at the Love Canal site and the effects on human. (U.S. Environmental Protection Agency, 1978)

Compound	Acute Effects	Chronic Effects
benzene	Narcosis Skin irritant	Acute leukemia Aplastic anemia Pancytopenia Chronic lymphatic leukemia Lymphomas (probable)
toluene	Narcosis (more powerful than benzene)	Anemia (possible) Leukopenia (possible)
benzoic acid	Skin irritant	
lindane	Convulsions High white cell counts	
trichloroethylene	Central nervous depression Skin irritant Liver damage	Paralysis of fingers Respiratory and cardiac arrest Visual defects Deafness

dibromoethane	Skin irritant	
benzaldehydes	Allergen	
methylene chloride	Anesthesia (increased carboxy hemoglobin)	Respiratory distress Death
carbon tetrachloride	Narcosis Hepatitis Renal damage	Liver tumors (possible)
chloroform	Central nervous narcosis Skin irritant Respiratory irritant Gastrointestinal symptoms	

Table 6.3. Congenital Malformations among children from the Love Canal (U.S. Environmental Protection Agency, 1978)

Type of Malformation	Sex	Date of Birth (month/year)	Location (North,South Canal)	Medication, Radiation during pregnancy
Cleft palate, deformed ears and teeth, hearing defect, mental retardation, heart defect	F	11/68	99th Street (South)	No
Abnormalities of renal pelvis and reflux ureters	F	4/75	99th Street (South)	No
Mental retardation (autistic)	F	1/66	97th Street (South)	No
Congenital deafness	M	2/66	99th Street (South)	No
Club foot	M	1/58	99th Street (North)	No

Table 6.3 provides information on the birth defects of five children born on the Love Canal. As was true for miscarriages, there appears to be a concentration of malformations on 99th Street.

Although further investigation obviously will be required, data analyzed to date seems to suggest that the risk for miscarriages and birth defects might be localized in 99th Street, particularly in the southern section. Researchers are now examining the possibility that this phenomenon may be related to the higher concentration of benzene (a known inhibitor of cell division) found in the southern Canal section.

Based on preliminary epidemiologic investigations, the Commissioner of Health recommended immediate relocation of all pregnant women and all children under two years of age from the Love Canal area. He also ordered delayed opening of the 99th Street elementary school which is situated in the central Love Canal section.

All reported birth defects were confirmed through medical records, and the past medical and drug histories of the mothers were evaluated for possible confounding influences. Reported miscarriages also were confirmed through private physicians' and hospital records.

Air samples are taken by the Division of Laboratories and Research of the State Health Department in July 1978 from the basements of 88 houses peripheral to those built adjacent to the landfill site to monitor the compounds (Table 6.4). Seven of the chemicals identified in the air samples taken by the Division of Laboratories and Research are carcinogenic in animals and one, benzene, is a known human carcinogen.

In 1979, the EPA announced the result of blood tests which showed high white blood cell counts, a precursor to leukemia, and chromosome damage in Love Canal residents. 33% of the residents had undergone chromosomal damage. In a typical population, chromosomal damage affects 1% of people. Other studies were unable to find harm. The United States National Research Council (NRC) surveyed Love Canal health studies in 1991. The NRC noted the major exposure of concern was the groundwater rather than drinking water; the groundwater "seeped into basements" and then led to exposure through air and soil noted several studies reported higher levels of low-birth weight babies and birth defects among the exposed

residents with some evidence the effect subsided after the exposure was eliminated.

Table 6.4. Ten compounds found in air samples taken from basements of 88 houses peripheral to those built adjacent to the Love Canal landfill site. (U.S. Environmental Protection Agency, 1978)

Compounds	No. Of Times Found In Houses	Percent of Total Houses Sampled	Highest Value Observed
Chloroform	23	26	24 ug/m ³
Benzene	20	23	270 ug/m ³
Trichloroethene	74	84	73 ug/m ³
Toluene	54	61	570 ug/m ³
Tetrachloroethene	82	93	1140 ug/m ³
Chlorobenzene	6	7	240 ug/m ³
Chlorotoluene	32	36	6700 ug/m ³
m+p xylene	35	40	140 ug/m ³
o-xylene	17	19	73 ug/m ³
Trichlorobenzene	11	13	74 ug/m ³

The National Research Council also noted a study which found exposed children were found to have an "excess of seizures, learning problems, hyperactivity, eye irritation, skin rashes, abdominal pain, and incontinence" and stunted growth. Voles in the area were found to have significantly increased mortality compared to controls (mean life expectancy in exposed animals "23.6 and 29.2 days, respectively, compared to 48.8 days" for control animals).

According to the United States Environmental Protection Agency (EPA) in 1979, residents exhibited a "disturbingly high rate of miscarriages ... Love Canal can now be added to a growing list of environmental disasters involving toxics, ranging from industrial workers stricken by nervous disorders and cancers to the discovery of toxic materials in the milk of nursing mothers." In one case, two out of four children in a single Love Canal family had birth defects; one girl was born deaf with a cleft palate, an extra row of teeth, and slight retardation, and a boy was born with an eye defect.

6.4.4. Evacuations

The federal government relocated more than 800 families and reimbursed them for the loss of their homes. The state government and federal government used \$15 million to purchase 400 homes closest to Love Canal and demolished several rings of houses.

In year 1978, the State Departments of Health and Environmental Conservation launched sampling and analysis of the homes adjacent to the Love Canal. The analysis had identified 82 different chemical compounds at the landfill, of one is known as human carcinogen and 11 are known or presumed animal carcinogens. The Niagara Country Health Commissioner had been summoned to eliminate the visible chemicals and restrict access of the site.

Extensive analyses showed that the chemicals seeped into the basement and contaminated the groundwater. The resident of Love Canal started to concern about the health issue and thereby Lois Gibbs, the head of the Love Canal Homeowners' Association made complaints about the surfaced chemicals substances in their yards. However, it was in vain as the local politicians were not assisting the residents. The dumped site was declared as an unprecedented state emergency on August, 1978. The residents were urged to evacuate the neighbourhood for good sake. United State President Jimmy Carter called for allocation of federal funds to remedy Love Canal site. The toxic area has been reburied with a thick plastic linear clay and dirt. The area is restricted from the public today while the issue has become a catalyst for future environmental laws creation.

6.4.5. Relocation of residents

Well before completion of the Department of Health's preliminary assessment of the scope of the health hazard posed by the Love Canal leachate, the Governor's Office began making preparations to mobilize the expertise and resources of key State agencies, including the Departments of Transportation, Health, Environmental Conservation, Housing, Social Services, Banking, Insurance, Office of Disaster Preparedness and Division of Equalization and Assessment. An initial step was a market survey by Department of Transportation real estate experts to determine availability of temporary and permanent replacement housing and to estimate the cost of relocating Love Canal residents and purchasing their homes.

The day after Commissioner Whalen's August 2 declaration that a medical emergency exists, interviewers from the regional offices of the Department of Transportation and the Department of Social Services opened a relocation assistance office at the 99th Street School - center of the stricken area.

Priority was given to securing temporary housing for families with children under two years of age and pregnant women. Some 41 top priority families were identified in the first two "rings" of homes - the 235 properties nearest the former canal bed.

Following Governor Carey's visit to the area on August 7, teams of interviewers began visiting homes to expedite the process of gathering the personal information needed to match families with available housing. By August 10, the scope of the relocation effort reached its present dimension with the decision to offer to relocate and purchase the homes of all 235 families in the first two rings.

Appraisal of properties which the State will offer to purchase, was begun August 15 by a team of Department of Transportation real estate appraisers, with purchase negotiations expected to begin within two weeks. The Urban Development Corporation will become the owner of the properties. At this date, the relocation effort is well advanced with some 136 families having accepted alternative housing. Of these, about 85 have already moved out of their canal area homes.

6.4.6. Conclusion

In 1978 President Carter declared a state of emergency at Love Canal, making it the first human-caused environmental problem to be designated that way. The Love Canal incident became a symbol of improperly stored chemical waste. Clean up of Love Canal, which was funded by Superfund and completely finished in 2004, involved removing contaminated soil, installing drainage pipes to capture contaminated groundwater for treatment, and covering it with clay and plastic. In 1995, Occidental Chemical (the modern name for Hooker Chemical) paid \$102 million to Superfund (A federal program created in 1980 and designed to identify and clean up the worst of the hazardous chemical waste sites in the U.S.) for cleanup and \$27 million to Federal Emergency Management Association for the relocation of more than 1,000 families. New York State paid \$98 million to EPA and the US

government paid \$8 million for pollution by the Army. The total clean up cost was estimated to be \$275 million. The only good thing about the Love Canal tragedy is that it helped to create Superfund, which has analyzed tens of thousands of hazardous waste sites in the U.S. and cleaned up hundreds of the worst ones.

6.5. Case Study of Methylmercury Poisoning in Japan (Minamata disease), 1956

6.5.1. Introduction

Minamata disease was first discovered in Minamata city in Kumamoto prefecture, Japan, in 1956. It was caused by the release of methylmercury in the industrial wastewater from the Chisso Corporation's chemical factory, which continued from 1932 to 1968 (Figure 6.11).



Figure 6.11. The release of methylmercury in the industrial wastewater from the Chisso Corporation's chemical factory, which continued from 1932 to 1968. The Chisso factory and its wastewater routes. Source: Wikipedia

This highly toxic chemical bioaccumulated in shellfish and fish in Minamata Bay and the Shiranui Sea, which, when eaten by the local populace, resulted in mercury poisoning. While cat, dog, pig, and human deaths continued for 36 years, the government and company did little to prevent the pollution. The animal effects were severe enough in cats that they came to be named as having "dancing cat fever". Minamata disease, sometimes referred to as Chisso-Minamata disease, is a neurological syndrome caused by severe mercury poisoning. Symptoms include ataxia, numbness in the hands and feet, general muscle weakness, loss of peripheral vision, and damage to hearing and speech. In extreme cases, insanity, paralysis, coma, and death follow within weeks of the onset of symptoms. A congenital form of the disease can also affect fetuses in the womb.

As of March 2001, 2,265 victims had been officially recognised as having Minamata disease (1,784 of whom had died) and over 10,000 had received financial compensation from Chisso. By 2004, Chisso Corporation had paid \$86 million in compensation, and in the same year was ordered to clean up its contamination. On March 29, 2010, a settlement was reached to compensate as-yet uncertified victims.

A second outbreak of Minamata disease occurred in Niigata Prefecture in 1965. The original Minamata disease and Niigata Minamata disease are considered two of the four big pollution diseases of Japan.

6.5.2. Causes of Minamata Disease

Researchers from Kumamoto University also began to focus on the cause of the strange disease. They found that the victims, often members of the same family, were clustered in fishing hamlets along the shore of Minamata Bay. The staple food of victims was invariably fish and shellfish from Minamata Bay. The cats in the local area, which tended to eat scraps from the family table, had died with symptoms similar to those now discovered in humans. This led the researchers to believe that the outbreak was caused by some kind of food poisoning, with contaminated fish and shellfish being the prime suspects (Figure 6.12).

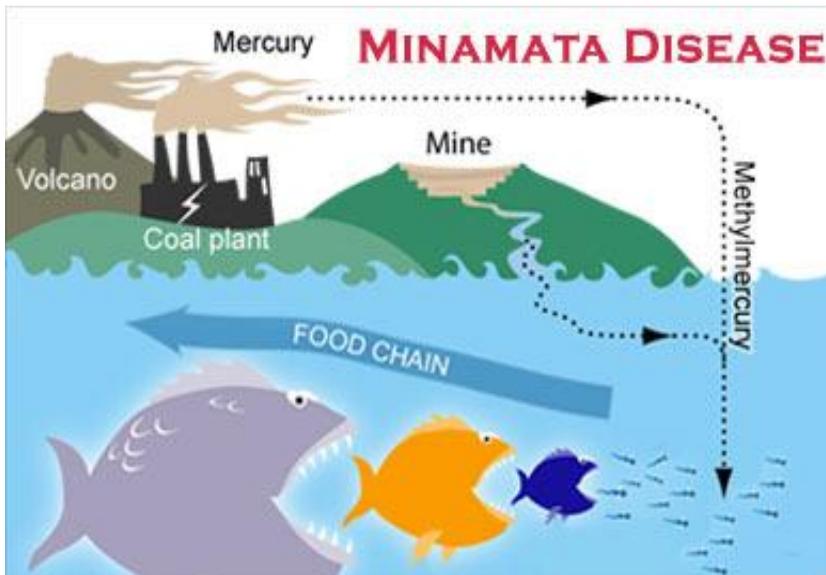


Figure 6.12. Minamata disease is caused through the absorption of methyl mercury into the human body through various environmental sources, primarily through seafood. Source: Wikipedia

On November 4, the research group announced its initial findings: "Minamata disease is rather considered to be poisoning by a heavy metal, presumably it enters the human body mainly through fish and shellfish."

As soon as the investigation identified a heavy metal as the causal substance, the wastewater from the Chisso plant was immediately suspected as the origin. The company's own tests revealed that its wastewater contained many heavy metals in concentrations sufficiently high to bring about serious environmental degradation, including lead, mercury, manganese, arsenic, thallium, and copper, plus the chalcogen selenium. Identifying which particular poison was responsible for the disease proved to be extremely difficult and time-consuming. During 1957 and 1958, many different theories were proposed by different researchers. At first, manganese was thought to be the causal substance due to the high concentrations found in fish and the organs of the deceased. Thallium, selenium, and a multiple contaminant theory were also proposed, but in March 1958, visiting British neurologist Douglas McAlpine suggested that Minamata symptoms resembled those of

organic mercury poisoning, so the focus of the investigation centered on mercury.

In February 1959, the mercury distribution in Minamata Bay was investigated. The results shocked the researchers involved. Large quantities of mercury were detected in fish, shellfish, and sludge from the bay. The highest concentrations centred around the Chisso factory wastewater canal in Hyakken Harbour and decreased going out to sea, clearly identifying the plant as the source of contamination. Pollution was so heavy at the mouth of the wastewater canal, a figure of 2 kg of mercury per ton of sediment was measured: a level that would be economically viable to mine. Indeed, Chisso did later set up a subsidiary to reclaim and sell the mercury recovered from the sludge.

Hair samples were taken from the victims of the disease and also from the Minamata population in general. In patients, the maximum mercury level recorded was 705 parts per million (ppm), indicating very heavy exposure and in nonsymptomatic Minamata residents, the level was 191 ppm. This compared to an average level of 4 ppm for people living outside the Minamata area.

On November 12, 1959, the Ministry of Health and Welfare's Minamata Food Poisoning Subcommittee published its results:

"Minamata disease is a poisoning disease that affects mainly the central nervous system and is caused by the consumption of large quantities of fish and shellfish living in Minamata Bay and its surroundings, the major causative agent being some sort of organic mercury compound."

During the investigation by researchers at Kumamoto University, the causal substance had been identified as a heavy metal and it was widely presumed that the Chisso plant was the source of the contamination. Chisso was coming under closer scrutiny and to deflect criticism, the wastewater output route was changed. Chisso knew of the environmental damage caused by its wastewater and was well aware that it was the prime suspect in the Minamata disease investigation. Despite this, from September 1958, instead of

discharging its waste into Hyakken Harbour (the focus of investigation and source of original contamination), it discharged wastewater directly into Minamata River. The immediate effect was the death of fish at the mouth of the river, and from that point on, new Minamata disease victims began to appear in other fishing villages up and down the coast of the Shiranui Sea, as the pollution spread over an even greater area.

The most common source is through contaminated seafood, as was the case for people residing in Minamata. A company by the name of Chisso, which was one of the biggest Japanese companies at the time, released its waste into the Minamata Bay. Methyl mercury was just one of the pollutants in the waste that was dumped and this particular pollutant accumulated in the food chain. It was absorbed and ingested by plankton, which is the primary food source for shellfish and fish in the water. Larger fish that ate such contaminated fish also absorbed the mercury and the poisoning continued to spread further in the food chain. Birds that fed on fish were also contaminated and so were other predators that preyed on these birds. In Minamata Bay, the pollutants were not dispersed over a wide area, but remained concentrated within the bay and this made the risk even greater as residents got their protein intake from seafood.

Mercury poisoning is also a huge health risk for pregnant women, as Minamata disease is known to affect the unborn child as well. Methyl-mercury can enter the fetus via the placenta and it has an adverse effect on brain development. In the case of residents in Minamata Bay area, it was observed that methyl-mercury deposits are concentrated in neural tissues. This pattern also serves as an explanation for the effect of neurotoxicity, especially in new-born babies as fetal transfer of the toxin cannot be prevented.

6.5.3. Symptoms and Signs of Minamata Disease

- Severe uncontrollable tremors
- Loss of motor control
- Sensory loss affecting both auditory and visual senses
- Partial paralysis
- Loss of muscle control during voluntary movements, known as ataxia
- Numbness in the extremities like the hands and feet
- Speech impairment may also be observed

While these are typical symptoms of mercury poisoning, the symptoms and combinations of symptoms can differ in severity. In children and infants who have inherited the condition, the symptoms can include the following:

- Infants will display symptoms that are similar to those afflicted with cerebral palsy, which is not surprising as it results in impaired neurological development and causes seizures.
- Growth and developmental problems with both physical and mental health
- Microcephaly, a neurodevelopmental disorder, in children
- Mental retardation
- Blindness and deafness in children

6.5.4. Diagnosis of Minamata Disease

Mercury poisoning is most often diagnosed with a laboratory measuring mercury levels in a hair sample. This is obviously non-invasive and is a very straightforward procedure. If you live in an area where there have been cases of mercury poisoning or if there is a sudden rise in similar cases with symptoms resembling those of mercury poisoning, health care providers would most likely request testing. Whether a person in Japan has Minamata disease is not just decided by your doctor but by the national government and local governors because of the legal requirements with regard to compensation and pollution related health incidents. If the symptoms are hard to identify, especially in newer cases with mild symptoms, and health care providers suspect Minamata Disease, they can request electro-ophthalmography (EOG) and optokinetic nystagmus pattern (OKP) to obtain referential data.

6.5.5. Treatment for Minamata Disease

Treatment may vary depending on severity of the condition and the symptoms present. However, there are certain standard approaches that are followed:

- Identifying and isolating the source of exposure so as to prevent any further exposure. This is obviously the first step and is in many ways a prerequisite to treatment.

- The most important aspect of treatment is the removal of mercury from the body using chelating agents. Chelating agents prevent heavy metals like mercury from binding with body tissue, by reacting with and binding with it themselves. This obviously comes with its own set of side-effects, which is why experts recommend agents with the lowest levels of toxicity. One risk from the use of chelating agents is that mercury may be redistributed and can even get to the brain.
- Loss of muscle function and paralysis are extremely common in patients and although severity may vary, physical rehabilitation is almost always necessary or useful as it helps patients regain some amount of control over mobility.
- In some cases, patients may suffer from convulsions in which case doctors will prescribe anticonvulsant drugs.
- Mercury is extremely harmful to the body as it also increases levels of reactive oxygen, which can be countered through the use of antioxidants.

6.5.6. Congenital Minamata Disease

Local doctors and medical officials had noticed for a long time an abnormally high frequency of cerebral palsy and other infantile disorders in the Minamata area. In 1961, a number of medical professionals including Masazumi Harada (later to receive an honour from the United Nations for his body of work on Minamata disease) set about re-examining children diagnosed with cerebral palsy. The symptoms of the children closely mirrored those of adult Minamata disease patients, but many of their mothers did not exhibit symptoms. The fact that these children had been born after the initial outbreak and had never been fed contaminated fish also led their mothers to believe they were not victims. At the time the medical establishment believed the placenta would protect the foetus from toxins in the bloodstream, which is indeed the case with most chemicals. What was not known at the time was that exactly the opposite is the case with methylmercury: the placenta removes it from the mother's bloodstream and concentrates the chemical in the foetus.

After several years of study and the autopsies of two children, the doctors announced that these children were suffering from an as yet unrecognised congenital form of Minamata disease. The certification committee convened on 29 November 1962 and agreed that the two dead

children and the 16 children still alive should be certified as patients, and therefore liable for "sympathy" payments from Chisso, in line with the 1959 agreement.

6.5.7. Outbreak of Niigata Minamata Disease

Minamata disease broke out again in 1965, this time along the banks of the Agano River in Niigata Prefecture. The polluting factory (owned by Showa Denko) employed a chemical process using a mercury catalyst very similar to that used by Chisso in Minamata. As in Minamata, from the autumn of 1964 to the spring of 1965, cats living along the banks of the Agano River had been seen to go mad and die. Before long, patients appeared with identical symptoms to patients living on the Shiranui Sea, and the outbreak was made public on 12 June 1965. Researchers from the Kumamoto University Research Group and Hajime Hosokawa (who had retired from Chisso in 1962) used their experience from Minamata and applied it to the Niigata outbreak. In September 1966, a report was issued proving Showa Denko's pollution to be the cause of this second Minamata disease.

Unlike the patients in Minamata, the victims of Showa Denko's pollution lived a considerable distance from the factory and had no particular link to the company. As a result, the local community was much more supportive of patients' groups and a lawsuit was filed against the company in March 1968, only three years after discovery.

The events in Niigata catalysed a change in response to the original Minamata incident. The scientific research carried out in Niigata forced a re-examination of that done in Minamata and the decision of Niigata patients to sue the polluting company allowed the same response to be considered in Minamata. Masazumi Harada has said that, "It may sound strange, but if this second Minamata disease had not broken out, the medical and social progress achieved by now in Kumamoto... would have been impossible."

Around this time, two other pollution-related diseases were also grabbing headlines in Japan. Victims of Yokkaichi asthma and Itai-itai disease were forming citizens' groups and filed lawsuits against the polluting companies in September 1967 and March 1968, respectively. As a group, these diseases came to be known as the four big pollution diseases of Japan.

6.5.8. Epidemiology

As of March 2001, 2,265 victims have been officially certified (1,784 of whom have died) and over 10,000 people have received financial compensation from Chisso, although they are not recognised as official victims. The issue of quantifying the impact of Minamata disease is complicated, as a full epidemiological study has never been conducted and patients were recognised only if they voluntarily applied to a certification council to seek financial compensation. Many victims of Minamata disease faced discrimination and ostracism from the local community if they came out into the open about their symptoms. Some people feared the disease to be contagious, and many local people were fiercely loyal to Chisso, depending on the company for their livelihoods. In this atmosphere, sufferers were reluctant to come forward and seek certification. Despite these factors, over 17,000 people have applied to the council for certification. Also, in recognising an applicant as a Minamata disease sufferer, the certification council qualified that patient to receive financial compensation from Chisso. For that reason, the council has always been under immense pressure to reject claimants and minimise the financial burden placed on Chisso. Rather than being a council of medical recognition, the decisions of the council were always affected by the economic and political factors surrounding Minamata and the Chisso Corporation. Furthermore, compensation of the victims led to continued strife in the community, including unfounded accusations that some of the people who sought compensation did not actually suffer from the disease. More properly, the impact should be called a criminal 'poisoning', not a clinical 'disease'. These forms of obfuscation are commonly experienced by 'environmental victims' in many countries.

6.5.9. Conclusion

Minamata disease remains an important issue in contemporary Japanese society. Lawsuits against Chisso and the prefectural and national governments are still continuing and many regard the government responses to date as inadequate. The company's "historical overview" in its current website makes no mention of their role in the mass contamination of Minamata and the dreadful aftermath. Their 2004 Annual Report however reports an equivalent of about US\$50 million (5,820 million yen) in "Minamata Disease Compensation Liabilities". From 2000 to 2003, the company also reported total compensation liabilities of over US\$170 million.

Their 2000 accounts also show that the Japanese and Kumamoto prefectural governments waived an enormous US\$560 million in related liabilities. Their FY2004 and FY2005 reports refer to Minamata disease as "mad hatter's disease", a term coined from the mercury poisoning experienced by hat-makers of the last few centuries.

A memorial service was held at the Minamata Disease Municipal Museum on 1 May 2006 to mark 50 years since the official discovery of the disease. Despite bad weather, the service was attended by over 600 people, including Chisso chairman Shunkichi Goto and Environment Minister Yuriko Koike.

On Monday, March 29, 2010, a group of 2,123 uncertified victims reached a settlement with the government of Japan, the Kumamoto Prefectural government, and Chisso Corporation to receive individual lump-sum payments of 2.1 million yen and monthly medical allowances.

Most congenital patients are now in their forties and fifties and their health is deteriorating. Their parents, who are often their only source of care, are into their seventies or eighties or already deceased. Often these patients find themselves tied to their own homes and the care of their family, effectively isolated from the local community. Some welfare facilities for patients do exist. One notable example is **Hot House**, a vocational training centre for congenital patients as well as other disabled people in the Minamata area. Hot House members are also involved in raising awareness of Minamata disease, often attending conferences and seminars as well as making regular visits to elementary schools throughout Kumamoto Prefecture.

6.6. Case Study of Iraq Poison Grain Disaster, 1971

6.6.1. Introduction

The **1971 Iraq poison grain disaster** was a mass methylmercury poisoning incident that began in late 1971. Grain treated with a methylmercury fungicide and never intended for human consumption was imported into Iraq as seed grain from Mexico and the United States. Due to a number of factors, including foreign-language labelling and late distribution within the growing cycle, this toxic grain was consumed as food by Iraqi residents in rural areas (Figure 6.13). People suffered from paresthesia

(numbness of skin), ataxia (lack of coordination of muscle movements) and vision loss, symptoms similar to those seen when Minamata disease affected Japan. The recorded death toll was 459 people, but figures at least ten times greater have been suggested. The 1971 poisoning was the largest mercury poisoning disaster when it occurred, with cases peaking in January and February 1972 and stopping by the end of March.

Reports after the disaster recommended tighter regulation, better labelling and handling of mercury-treated grain, and wider involvement of the World Health Organization in monitoring and preventing poisoning incidents. Investigation confirmed the particular danger posed to fetuses and young children.



Figure 6.13. A sack of "pink grain". Grain treated with a methylmercury fungicide, 1971. Source: Wikipedia

6.6.2. Context

The properties of mercury make it an effective fungicide. However, in Europe and America, its health risks (even when consumed in small quantities) were known. Methylmercury had been banned in Sweden in 1966, the first country to do so, and the United Kingdom followed in 1971. Previous mercury-poisoning incidents had occurred in Iraq in 1956 and 1960. In 1956, there had been around 200 cases, and 70 deaths; in 1960 there had been 1,000

cases and 200 deaths, in both cases due to ethylmercury compounds. Among the recommendations made after the 1960 incident had been to colour any toxic grain for easy identification. Before the 1971 incident, around 200–300 cases of methylmercury poisoning had been reported worldwide. Drought had reduced harvests in 1969, affecting 500,000 people, and in 1970. Saddam Hussein, as the government's no. 2 behind Ahmed Hassan al-Bakr, decided to import mercury-coated seed grain for the late 1971 planting season. Hussein himself may have worked in the Department of Agriculture in the aftermath of the 1960 incident.

6.6.3. Causes

Some 95,000 tonnes (93,000 long tons; 105,000 short tons) of grain (73,201 tonnes of wheat grain and 22,262 tonnes of barley), coloured a pink-orange hue, were shipped to Iraq from the United States and Mexico. The wheat arrived in Basra on SS *Trade Carrier* between 16 September and 15 October, barley between 22 October and 24 November 1971. Iraq's government chose Mexipak, a high-yield wheat seed developed in Mexico by Norman Borlaug. The seeds contained an average of 7.9 µg/g of mercury, with some samples containing up to nearly twice that. The decision to use mercury-coated grain has been reported as made by the Iraqi government, rather than the supplier, Cargill. The three Northern governorates of Ninawa, Kirkuk and Erbil together received more than half the shipments. Contributing factors to the epidemic included the fact that distribution started late, and much grain arrived after the October–November planting season.

Farmers holding grain ingested it instead, since their own planting had been completed. Distribution was hurried and open, with grain being distributed free of charge or with payment in kind. Some farmers sold their own grain lest this new grain devalue what they had. This left them dependent on tainted grain for the winter. Many Iraqis were either unaware of the significant health risk posed, or chose to ignore the warnings. Initially, farmers were to certify with a thumbprint or signature that they understood the grain was poison, but according to some sources, distributors did not ask for such an indication. Warnings on the sacks were in Spanish and English, not at all understood, or included the black-and-white skull and crossbones design, which meant nothing to Iraqis. The long latent period may have granted farmers a false sense of security, when animals fed the grain appeared to be

fine. The red dye washed off the grain; the mercury did not. Hence, washing may have given only the appearance of removing the poison.

Mercury was ingested through the consumption of homemade bread, meat and other animal products obtained from livestock given treated barley, vegetation grown from soil contaminated with mercury, game birds that had fed on the grain and fish caught in rivers, canals, and lakes into which treated grain had been dumped by the farmers. Ground seed dust inhalation was a contributing factor in farmers during sowing and grinding. Consumption of ground flour through homemade bread is thought to have been the major cause, since no cases were reported in urban areas, where government flour supplies were commercially regulated.

6.6.4. Symptoms, outbreak and treatment

The effect of mercury took some time – the latent period between ingestion and the first symptoms (typically paresthesia – numbness in the extremities) was between 16 and 38 days. Paresthesia was the predominant symptom in less serious cases. Worse cases included ataxia (typically loss of balance), blindness or reduced vision, and death resulting from central nervous system failure. Anywhere between 20 and 40 mg of mercury has been suggested as sufficient for paresthesia (between 0.5 and 0.8 mg/kg of body weight). On average, individuals affected consumed 20 kg or so of bread; the 73,000 tonnes provided would have been sufficient for over 3 million cases.

The hospital in Kirkuk received large numbers of patients with symptoms that doctors recognised from the 1960 outbreak. The first case of alkylmercury poisoning was admitted to hospital on 21 December. By 26 December, the hospital had issued a specific warning to the government. By January 1972, the government had started to strongly warn the populace about eating the grain, although dispatches did not mention the large numbers already ill. The Iraqi Army soon ordered disposal of the grain and eventually declared the death penalty for anyone found selling it. Farmers dumped their supplies wherever possible, and it soon got into the water supply (particularly the River Tigris), causing further problems. The government issued a news blackout and released little information about the outbreak.

The World Health Organization assisted the Iraqi government through the supply of drugs, analytical equipment and expertise. Many new

treatments were tried, since existing methods for heavy metal poisoning were not particularly effective. Dimercaprol was administered to several patients, but caused rapid deterioration of their condition. It was ruled out as a treatment for this sort of poisoning following the outbreak. Polythiol resins, penicillamine and dimercaprol sulfonate all helped, but are believed to have been largely insignificant in overall recovery and outcomes. Dialysis was tested on a few patients late in the treatment period, but they showed no clinical improvement. The result of all treatments was varied, with some patients' blood mercury level being dramatically reduced, but a negligible effect in others. All patients received periods of treatment interspersed with lay periods; continuous treatment was suggested in future cases. Later treatment was less effective in reducing blood toxicity.

6.6.5. Effects

6,530 patients were admitted to hospitals with poisoning, and 459 deaths reported. Cases reached a peak of hundreds per day in January, and had largely subsided by the beginning of March. The last admittance was on 27 March; admissions represented every age and gender stratum, although those under the age of ten represented a third of admitted cases. This number is "certainly an underestimate", because of the availability of hospital treatment, hospital overcrowding and lack of faith in treatment. In the most severely affected areas, prevalence was 28% and mortality was 21% of the cases (Figure 6.14). Some Iraqi doctors believe both the number of cases and fatalities are at least ten times too low, with perhaps 100,000 cases of brain damage. One suggested reason for the vast discrepancy between reported and estimated numbers of deaths is the Iraqi custom, common to large parts of the Middle East, for a person to die at home when possible. Home deaths would not have been recorded.

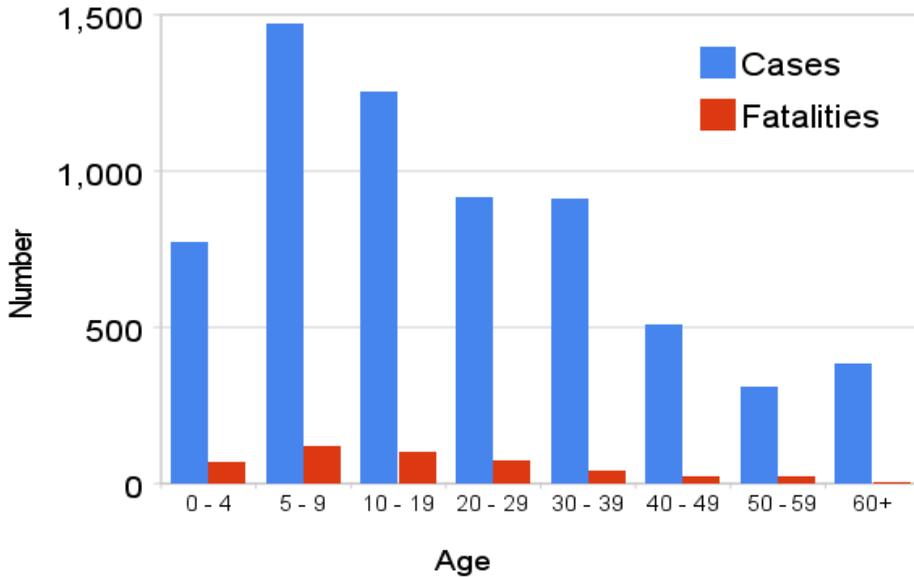


Figure 6.14. Incidence of cases and fatalities, by age group. Iraq Poison Grain Disaster, 1971. Source: Wikipedia

A large number of patients with minor symptoms recovered completely; those with more serious symptoms improved. This was in contrast to expected outcomes, largely based on analysis of Minamata disease in Japan. In boys with mercury levels below clinical poisoning, a reduction in school performance was noted, although this correlation could not be confirmed. In infants, the mercury poisoning caused central nervous system damage. Relatively low doses caused slower development in children, and abnormal reflexes. Different treatments for mercury poisoning have since been developed, and "quiet baby syndrome", characterised by a baby who never cries, is now a recognised symptom of methylmercury-induced brain damage. Ongoing recommendations of the food regulation authorities have focused on consumption by pregnant women and infant children, noting the particular susceptibility of fetuses and infants to methylmercury poisoning. Data from Iraq have confirmed that methylmercury can pass to a child *in utero*, and mercury levels were equal to or higher in the newborn child than in the mother.

6.6.6. Conclusion

In 1974, a joint Food and Agriculture Organization (FAO) and World Health Organisation (WHO) meeting made several recommendations to prevent a similar outbreak. These included stressing the importance of labelling bags in the local language and with locally understood warning symbols. The possibility of an additive creating a strong bitter taste was studied. The meeting urged governments to strictly regulate methyl- and ethylmercury use in their respective countries, including limiting use to where no other reasonable alternative was available. It also recommended the involvement of the FAO and WHO in assisting national governments in regulation and enforcement, and the setting up of national poison control centres.

Over 9–13 November, a Conference on Intoxication due to Alkylmercury-Treated Seed was held in Baghdad. It supported the recommendations of the FAO/WHO report and further suggested that local and national media should publicise outbreaks, including size and symptoms; it considered the distribution of this information crucial. It also laid out a general plan as to the collection of relevant information from the field and potential analysis for further investigation. It called on national governments to make use of WHO involvement whenever feasible, and absolved world governments in clear terms, saying that "No country should ever feel that any blame will attach to it for allowing an outbreak to occur".

Iraq now has the highest incidence of Parkinson's in the world. Parkinson's symptoms are very similar to mercury poisoning symptoms. Mercury that enters the brain has a half-life of 27.5 years and chelators are not able to remove it.

6.7. Case Study of Air pollution in London, 1952. (Great Smog of London)

6.7.1. Introduction

The **Great Smog of London**, or **Great Smog of 1952** sometimes called the **Big Smoke**, was a severe air-pollution event that affected the British capital of London in December 1952 (Figure 6.15 and 6.16). A period of cold weather, combined with an anticyclone and windless conditions,

collected airborne pollutants – mostly arising from the use of coal – to form a thick layer of smog over the city. It lasted from Friday, 5 December to Tuesday, 9 December 1952 and then dispersed quickly when the weather changed.



Figure 6.15. Great Smog of London, 1952. Source: Getty Images

It caused major disruption by reducing visibility and even penetrating indoor areas, far more severe than previous smog events experienced in the past, called "pea-soupers". Government medical reports in the following weeks, however, estimated that up until 8 December, 4,000 people had died as a direct result of the smog and 100,000 more were made ill by the smog's effects on the human respiratory tract.

London had suffered since the 1200s from poor air quality, which worsened in the 1600s, but the Great Smog is known to be the worst air-pollution event in the history of the United Kingdom, and the most significant in terms of its effect on environmental research, government regulation, and public awareness of the relationship between air quality and health.



Figure 6.16. The smoke belching from millions of London's chimney pots.
Source: Daily Mail

6.7.2. Sources of pollution

The cold weather preceding and during the Great Smog led Londoners to burn more coal than usual to keep warm. Post-war domestic coal tended to be of a relatively low-grade, sulphurous variety (economic necessity meant that better-quality "hard" coals tended to be exported), which increased the amount of sulphur dioxide in the smoke. There were also numerous coal-fired power stations in the Greater London area, including Fulham, Battersea, Bankside and Kingston upon Thames, all of which added to the pollution. According to the UK's Met Office, the following pollutants were emitted each day during the smoggy period: 1,000 tonnes of smoke particles, 140 tonnes of hydrochloric acid, 14 tonnes of fluorine compounds, and 370 tonnes of sulphur dioxide which may have been converted to 800 tonnes of sulphuric acid.

Research suggested that additional pollution-prevention systems fitted at Battersea may have worsened the air quality, reducing the output of soot at the

cost of increased sulphur dioxide, though this is not certain. Additionally, there was pollution and smoke from vehicle exhaust – particularly from steam locomotives and diesel-fuelled buses, which had replaced the recently abandoned electric tram system – and from other industrial and commercial sources. Prevailing winds had also blown heavily polluted air across the English Channel from industrial areas of Continental Europe.

6.7.3. Weather

On 4 December 1952, an anticyclone settled over a windless London, causing a temperature inversion with cold, stagnant air trapped under a layer (or "lid") of warm air. The resultant fog, mixed with chimney smoke, particulates such as those from vehicle exhausts, and other pollutants such as sulphur dioxide, formed a persistent smog, which blanketed the capital the following day. The presence of tarry particles of soot gave the smog its yellow-black colour, hence the nickname "pea-souper". The absence of significant wind prevented its dispersal and allowed an unprecedented accumulation of pollutants.

6.7.4. Effect on London

Although London was accustomed to heavy fogs, this one was denser and longer-lasting than any previous fog. Visibility was reduced to a few metres ("It's like you were blind") making driving difficult or impossible.

Public transport ceased, apart from the London Underground; and the ambulance service stopped functioning, forcing users to transport themselves to hospital. The smog even seeped indoors, resulting in the cancellation or abandonment of concerts and film screenings as visibility decreased in large enclosed spaces, and stages and screens became harder to see from the seats. Outdoor sports events were also affected.

In the inner London suburbs and away from town centres there was no disturbance by moving traffic to thin out the dense fog in the back streets. The result was that visibility could be down to a metre or so in the daytime. Walking out of doors became a matter of shuffling one's feet to feel for potential obstacles such as road kerbs. This was made even worse at night since each back street lamp at the time was fitted with an incandescent light-bulb, which gave no penetrating light onto the pavement for pedestrians to see

their feet, or even the lamp post. Fog-penetrating fluorescent lamps did not become widely available until later on in the 1950s. "Smog masks" were worn by those who were able to purchase them from chemists.

Near railway lines, on which "fog working" was implemented, loud explosions similar to the report of a shotgun were a common feature. The explosions were made by "detonators" – a form of large percussion cap placed on the track and activated by the wheels of trains. These devices were placed by certain signals to provide an audible warning to match the visual indication provided by the signal for the driver.

6.7.5. Health effects

There was no panic, as London was renowned for its fog. In the weeks that ensued, however, statistics compiled by medical services found that the fog had killed 4,000 people. Most of the victims were very young or elderly, or had pre-existing respiratory problems. In February 1953, Lieutenant-Colonel Lipton suggested in the House of Commons that the fog had caused 6,000 deaths and that 25,000 more people had claimed sickness benefits in London during that period (Figure 6.17).



**Figure 6.17. Patient suffering from respiratory tract infections.
Air pollution in London, 1952. Source: Getty Image**

Mortality remained elevated for months after the fog. A preliminary report, never finalised, blamed the ongoing deaths on an influenza epidemic. Emerging evidence revealed that only a fraction of the deaths could be from influenza. Most of the deaths were caused by respiratory tract infections, from hypoxia and as a result of mechanical obstruction of the air passages by pus arising from lung infections caused by the smog. The lung infections were mainly bronchopneumonia or acute purulent bronchitis superimposed upon chronic bronchitis. More recent research suggests that the number of fatalities was considerably greater than contemporary estimates, at about 12,000.

6.7.6. Environmental impact

The death toll formed an important impetus to modern environmentalism, and it caused a rethinking of air pollution, as the smog had demonstrated its lethal potential. New regulations were implemented, restricting the use of dirty fuels in industry and banning black smoke.

Environmental legislation since 1952, such as the City of London (Various Powers) Act 1954 and the Clean Air Acts of 1956 and 1968, led to a reduction in air pollution. Financial incentives were offered to householders to replace open coal fires with alternatives (such as installing gas fires), or for those who preferred, to burn coke instead which produces minimal smoke. Central heating (using gas, electricity, oil or permitted solid fuel) was rare in most dwellings at that time, not finding favour until the late 1960s onwards. Despite improvements, insufficient progress had been made to prevent one further smog event approximately ten years later, in early December 1962.

6.7.7. Conclusion

For five days in December 1952, a fog that contained pollutants enveloped all of London. By the time the dense fog cover lifted, more than 150,000 people had been hospitalized and at least 4,000 people had died. Researchers now estimate that the total death count was likely more than 12,000 people, as well as thousands of animals. Despite its lethal nature, the exact cause and nature of the killer fog has largely remained a mystery. Recently, a team of researchers has determined the likely reasons for its formation

Atmospheric scientists at Texas A&M University investigating the haze of polluted air in Beijing realized their research led to a possible cause for the London event in 1952. "By examining conditions in China and experimenting in a lab, the scientists suggest that a combination of weather patterns and chemistry could have caused London fog to turn into a haze of concentrated sulfuric acid."

Even though research findings point in this direction, the two events are not identical. In China, the combination of nitrogen dioxide and sulfur dioxide, both produced by burning coal, with a humid atmosphere, created sulfates while building up acidic conditions that, left unchanged, would have stalled the reaction. However, ammonia from agricultural activity neutralized the acid allowing sulfate production to continue.

It is theorized that in 1952 in London, the nitrogen dioxide and sulfur dioxide combined with fog rather than humidity; larger droplets of water diluted the acid products, allowing more sulfate production as sulfuric acid. Sunrise burned off the fog, leaving concentrated acid droplets which killed citizens.

The 1952 killer fog led to the creation of the Clean Air Act, which the British Parliament passed in 1956. Researchers still consider it the worst air pollution event in European history.

7. Environmental Toxins and Human Cancers

7.1. Introduction

Cancer is caused by changes to certain genes that alter the way our cells function. Some of these genetic changes occur naturally when DNA is replicated during the process of cell division. But others are the result of environmental exposures that damage DNA. One research study concludes that as many as 35% of all cancers are driven by Environmental toxins. A British Government White Paper concluded that across 4000 common compounds used in-home, two thirds were toxic, and one third were probably carcinogenic.

Many well-known scientists, public health officials and physicians have been sounding alarms about the links between environmental toxins and human cancer for years now. The incidence of certain cancers, particularly thyroid cancer and leukemia, may very well rise in the Japanese population most heavily exposed to radiation from the Fukushima accident. Environmental toxins pose potentially grave threats to our health, and accidents only compound these threats both locally and for people all over the world who breathe air, eat food, and drink water. People can avoid some cancer-causing exposures, such as tobacco smoke and the sun's rays. But others are harder to avoid, especially if they are in the air we breathe, the water we drink, the food we eat, or the materials we use to do our jobs.

Scientists are studying which exposures may cause or contribute to the development of cancer. Understanding which exposures are harmful, and where they are found, may help people to avoid them. The chemicals in our water, air, and food, the materials in our home, and non-ionizing radiation present cancer risks. But that doesn't mean that we are defenseless. Researchers have identified several mechanisms by which most cancer-producing toxins disrupt our body's defense systems. Compelling evidence reveals how we can defend against these carcinogenic mechanisms.

7.2. Sources of Cancer-Inducing and Cancer-Promoting Toxins

While it is impossible to avoid all cancer-causing environmental toxins, it is important to be aware of some of the most prominent sources.

Researchers have compiled a list of common toxins broken down by their environmental sources. As you'll see from this list, these can be found in sources we interact with on a daily basis, including our food, water, plastic, cell phones, and even sunlight (Table 7.1).

- **Aflatoxins** are toxic chemicals produced by *Aspergillus* fungi growing on grains and peanuts, particularly those stored improperly. Chronic exposure induces cancer by multiple mechanisms.
- **Polycyclic aromatic hydrocarbons** are chemical structures composed of carbon, hydrogen, and occasionally other atoms. They are products of fossil fuel combustion, particularly petrochemicals, and are a major source of cancer-causing chemicals in polluted air.
- **Bisphenol A (BPA)** is one of the highest-volume toxic chemicals found worldwide. It is used in making all kinds of plastics and resins, including water bottles and food containers.
- **Heavy metals** (including cadmium, arsenic, nickel, lead, and mercury) are naturally occurring components of the earth's crust. Human exposure results from mining, smelting, and petroleum manufacturing, all of which release heavy metals into the air, water, and soil.
- **Pesticides and herbicides**, especially those containing organic chemicals bonded to chlorine or bromine, are found in agricultural settings, where they make their way into the food chain. Sadly, even after the highly toxic dichlorodiphenyltrichloroethane (DDT) was banned, risks still abound, both because of persistent DDT in the environment and because newer compounds intended to replace DDT (such as methoxychlor) are turning out to have their own cancer-inducing properties.
- **Dioxins** and dioxin-like chemicals such as polychlorinated biphenyls (PCBs) are commonly found in foods of animal origin (meat, dairy, and fish, depending on the country of origin).
- **Heterocyclic amines** are chemicals that form when meat is cooked at high temperatures (e.g., grilled or broiled).
- **Ultraviolet radiation** is a natural component of sunlight, but serves as a powerful source of many of the changes that lead to cancer.
- **Electromagnetic field radiation**, especially the kind produced by cellular phones and their transmitting stations, are only now emerging as potential environmental threats. Such radiation is associated with DNA damage, potentially leading to cancer.

This is by no means an exhaustive listing of cancer-related environmental toxins. Toxins are ubiquitous, particularly in our highly industrialized society. They are, therefore, nearly impossible to avoid, but as we have read, we know that we are not helpless. We can arm ourselves with knowledge about natural products capable of offsetting much of the increased cancer risk posed by environmental toxins.

**Table 7.1. Toxins that induce or promote cancers
(International Agency for Research on Cancer, 2011)**

Basic Mechanism	Toxins
Increased DNA damage	<ul style="list-style-type: none"> • Aflatoxin A1 • Air pollutants (polycyclic aromatic hydrocarbons, tobacco smoke) • Arsenic • Bisphenol A (BPA) • Cadmium chloride • Microwave radiation • Mobile phone radiation • Nickel salts • Pesticides • Ultraviolet light
Activation by liver enzymes	<ul style="list-style-type: none"> • Aflatoxin B1 • Dioxin • Heterocyclic amines (from cooking meat) Nicotine • Polycyclic aromatic hydrocarbons (e.g., benzo[a]pyrene)
Suppress immune surveillance	<ul style="list-style-type: none"> • Dioxin • Mercury • Mycotoxins (e.g., aflatoxins, fumonisins, and deoxynivalenol) • Perfluorinated hydrocarbons • Tobacco smoke • Ultraviolet light

Endocrine disruption	<ul style="list-style-type: none"> • BPA • Cyprodinil (a fungicide related to polycyclic aromatic hydrocarbons) • Dioxin • Heavy metals (arsenic, cadmium, lead, mercury) • Methoxychlor • Phthalates • PCBs) • Polycyclic aromatic hydrocarbons • Triclosan
Loss of apoptosis	<ul style="list-style-type: none"> • Aflatoxins • Heavy metals • Pesticides/herbicides • UV light

7.3. How Toxins Produce Cancer and Powerful Cancer Prevention from Nutrients

The heavily industrialized nature of our modern world constantly exposes us to toxic, cancer-inducing, and cancer-promoting influences. Chemical toxin as well as various sources of radiation can initiate cancer through DNA damage and promote tumor development through mechanisms involving liver enzyme systems, suppressed immunity, disruption of your hormones, and hijacking the cells' normal death-inducing programs. You can't realistically evade all of the factors that can cause cancer in your life, but you can leverage modern scientific knowledge to your benefit. Many nutrients are available with known cancer-fighting benefits, including those that directly counteract the major processes by which toxic compounds and radiation promote malignancies.

Despite the vast number and diversity of cancer types, there are a relatively small number of events that typically occur in the progression from healthy cell to malignancy. Toxic environmental chemicals, electromagnetic

fields, and ionizing radiation may initiate and/or promote malignancy, operating along a number of mechanisms. Some of these mechanisms include:

- DNA damage,
- Liver detoxification impairment,
- Immune impairment,
- Endocrine disruptors, and
- Loss of apoptosis.

7.3.1. DNA Damage

The first way toxins may lead to cancer is by breaking DNA strands. Damage to DNA is a major initiating factor in cellular transformation to cancer. DNA damage can be caused by toxins that break DNA strands (such as pro-oxidant chemicals or ionizing radiation). Such damage can induce mutations in the DNA that trigger cancer. That is why nutrients that prevent DNA damage, or ones that promote its **repair**, are so potent in protecting against cancer. Given the role of oxidative stress in causing such damage, **nutrients that reduce DNA damage** (i.e. **vitamins C and E** and the trace mineral **selenium**), are often considered as a first line of defense.

Surprisingly, **probiotics**, which are normally associated with improved gastrointestinal function, have been found to be effective at reducing DNA damage specifically in the colon. This may help to prevent **colon cancer**, the third cause of cancer-related deaths in the US. And, while sunlight exposure can boost vitamin D levels, such exposure also raises DNA skin damage, but **vitamin D supplementation** can protect against DNA damage throughout the body. Nearly a dozen nutrients have been found to prevent DNA damage, resulting in a positive impact on cancers of the prostate, colon, breast, skin, liver, and more.

Powerful Cancer Prevention from nutrients that reduce DNA damage. It is easy to become fearful about the vast numbers of toxic chemicals and other influences all around us, lurking to produce catastrophic cancers. Fortunately, there are solutions in the form of specific nutrients with powerful cancer-preventing effects. Compelling scientific studies show that specific nutrients counteract major processes by which toxic compounds and radiation promote malignancies. Table 7.2 lists a few of the nutrients known for their DNA-protective effect.

**Table 7.2. Nutrients that reduce DNA damage
(International Agency for Research on Cancer, 2011)**

Nutrient	Cancers Affected
Carotenoids (lutein, astaxanthin, lycopene)	Prostate, colon
Coenzyme Q10	Colon, head-and-neck
Fish oil (omega-3 fats)	Colon, skin
Genistein (from soy)	Leukemia
Plant polyphenols	Multiple
Probiotics (<i>Lacto-bacillus rhamnosus</i>)	Colon
Quercetin	Head-and-neck
Selenium	Colon, breast
Vitamin C	Breast, colon, skin
Vitamin D	Colon, skin
Vitamin E	Liver, breast

7.3.2. Liver Detoxification Systems

The second way environmental toxins cause cancer is through their detrimental impact on liver detoxification systems. **Liver detoxification systems** play a major role in managing ingested toxins because blood from the digestive tract goes to the liver before being pumped around to the remainder of the body.

The liver has two major detoxification pathways: Phase I and Phase II. In **Phase I** enzymes convert toxic chemicals into compounds that may be more toxic than the parent compound. Unfortunately, if the toxic load is too heavy, it can cause overactivity of Phase I enzymes, which can have the reverse effect of converting relatively harmless substances into potential DNA-damaging carcinogens. Making matters worse, the worst offenders of overactive Phase I enzymes are substances some people encounter on a daily basis, including alcohol, saturated fats, and exhaust fumes, among others.

In **Phase II** detoxification, the liver adds another substance to the toxic chemical in order to make it more water soluble. This allows your body to excrete the toxin through bile or urine, helping remove the potentially carcinogenic substance from the body.

For these reasons, cancer-preventive nutrients that influence liver metabolism are generally those that *regulate* toxin-enhancing Phase I reactions, promote toxin-neutralizing Phase II reactions, or, in many cases, do both. Nutrients that regulate these liver detoxification systems come largely from dietary plants and their extracts.

Several nutrients have this dual action on liver enzymes, including curcumin, folic acid, and garlic, among others. Research suggests this may have a positive impact on preventing some of the most common and deadly cancers. List of nutrients that prevent cancer by regulating the liver's detoxification enzymes are shown in Table 7.3.

Table 7.3. Nutrients that prevent cancer by regulating the liver's detoxification enzymes. (International Agency for Research on Cancer, 2011)

Nutrient	Impact on Liver Enzymes	Cancers Affected
Chlorophyllin	Inhibit Phase I; boost Phase II	Liver, colon, prostate
Curcumin	Inhibit Phase I; boost Phase II	Breast, colon, prostate, pancreas
Folic acid	Inhibit Phase I; boost Phase II	Breast, pancreas
Garlic	Inhibit Phase I; boost Phase II	Breast, liver, prostate
Genistein	Boost Phase II	Colon
Isothiocyanates (sulforaphane, PEITC) from cruciferous vegetables	Inhibit Phase I; boost Phase II	Liver, colon, breast, prostate
Plant flavonoids (i.e. chrysin, genistein, quercetin)	Inhibit Phase I; boost Phase II	Multiple
Silymarin (milk thistle)	Boost Phase II	Liver

7.3.3. Immune Surveillance

The third way environmental toxins can cause cancer is through their impact on immune surveillance. *Immune surveillance* refers to the immune system's continual search for cells bearing signs that they have become cancerous. A number of environmental toxins can suppress immune surveillance, raising the risk that a malignant cell will slip under the radar, form a tumor, and successfully spread to other parts of the body.

Nutrients that enhance immune surveillance are only now being recognized as powerful contributors to the body's lifelong fight against cancer. These nutrients boost those components of the immune system that are responsible for recognizing the unique tumor "markers" displayed on the surface of malignant cells, and then destroying those cells.

**Table 7.4. Nutrients that boost immune surveillance
(International Agency for Research on Cancer, 2011)**

Nutrient	Immune Mechanism	Cancers Affected
Enzymatically modified rice bran	Increases natural killer cell activity	Leukemia, Multiple Myeloma, Liver
<i>Cistanche</i> extracts	Increases naïve T-cells, increased expression of transforming growth factor beta, decreases inflammation	Colon
Grape seed proanthocyanidins	Induction of immunoregulatory cytokines; stimulation of tumor-destroying T cells	Skin
Green tea polyphenols (EGCG)	Activation of tumor-killing T-lymphocytes and natural killer cells; induction of immuno-regulatory cytokines	Colon, skin, lung, prostate, breast

Probiotics (lactic acid bacteria)	Decrease inflammation; increase immunoregulatory cytokines; increased interferon-gamma production	Colon, skin
Reishi mushroom (<i>Ganoderma lucidum</i>)	Enhanced proliferation of tumor-killing T-lymphocytes, antibody-producing B-lymphocytes, and natural killer cells	Liver, lymphoma, lung
Resveratrol	Sensitizes tumor cells to killing by cytokine-induced killer cells; enhances cytokine-induced killer cell activity	Leukemia
Silymarin	Induction of immunoregulatory cytokines; stimulation of tumor-destroying T cells	Skin
Vitamin D	Reduced inflammation	Colon

Nutrients that enhance immune surveillance may stimulate growth and proliferation of **tumor-detecting lymphocytes**, promote a vigorous attack on tumor cells by so-called “**natural killer cells**,” and/or stimulate **antibody production**, which aids in immobilization and destruction of malignant cells. Table 7.4 lists nutrients capable of activating one or more components of the immune system in order to destroy developing cancers.

7.3.4. Endocrine Disruptors

The fourth way environmental toxins can cause cancer is through their impact on endocrine disruptors. **Endocrine disruptors** are chemicals that interact with sex hormones and/or their receptors to promote cancer development.

Not surprisingly, **nutrients that inhibit endocrine disruptors** show promise in preventing hormone-dependent cancers such as those of the breast, uterus, and prostate. Although scientists don't yet fully know how these

nutrients work to inhibit endocrine disruptors, it may involve enhanced excretion or reduced absorption of toxins from the intestinal tract.

Table 7.5. Nutrients capable of inhibiting endocrine-disrupting Pollutants. (International Agency for Research on Cancer, 2011)

Nutrient	Toxin Inhibited	Cancers Affected
<i>Chlorella pyrenoidosa</i> (More efficiently obtained as chlorophyllin)	Dioxin (a polychlorinated biphenyl [PCB]), perfluorinated compounds (PFCs)	Breast, prostate
Folic acid	Bisphenol A (BPA), phthalates	Breast, prostate
Genistein	BPA, phthalates	Breast, prostate
Probiotics (<i>Lactobacillus</i> , <i>Bifidobacterium</i>)	BPA	Breast, prostate
Vitamin C	Heavy metals (lead, copper, iron)	Liver, lung, prostate
Vitamin E	Heavy metals (lead, copper, iron)	Liver, lung, prostate

Table 7.5 lists nutrients capable of inhibiting endocrine-disrupting pollutants. In addition, there are also a number of **plant flavonoids** (i.e. chrysin, genistein, quercetin) that are effective against endocrine disrupting toxins. They appear to reduce the activity of estrogen-producing enzymes such as aromatase, thereby reducing overall sex hormone predominance and starving hormone-dependent tumors of their vital growth factors.

7.3.5. Loss of Apoptosis

Another way environmental toxins are associated with cancer is through inducing a loss of apoptosis, or programmed cell death. **Loss of apoptosis** refers to the “immortality” typical of cancer cells. Normal body cells are programmed to die off when appropriate. Cancer cells have lost this

ability (often as a result of DNA damage), which allows them to reproduce essentially without limit. A number of chemical toxins, particularly **aflatoxin**, a potent inducer of liver damage, can switch off the gene responsible for producing apoptosis, which results in cancer promotion.

Nutrients that restore cells' natural ability to die by apoptosis represent the final category in our listing of nutrients that help fight against cancers caused by environmental toxins. These nutrients typically act by modifying various signaling pathways. This means that they can activate genes that become suppressed when cells become cancerous, including genes that normally support the graceful death of a cell that is no longer useful or poses a threat.

By restoring the natural self-destruction program initiated by apoptosis genes, these nutrients put a sharp roadblock in the way of a developing tumor. This allows other anticancer mechanisms such as immune surveillance to clear the remainder of the battlefield.

Nutrients known to promote apoptosis include coffee extract, quercetin, pine bark extract, and selenium. Research shows they have a positive impact on bladder, colon, and ovarian cancers, among others. Detailed list of nutrients that promote or restore apoptosis capabilities in malignant cells are shown in Table 7.6.

**Table 7.6. Nutrients that promote or restore apoptosis
(International Agency for Research on Cancer, 2011)**

Nutrient	Cancers Affected
Chlorophyllin	Bladder
Coffee extract	Colon
Curcumin	Leukemia, colon
<i>Emblica officinalis</i> (amla; Indian gooseberry)	Ovary
Green tea extract (EGCG)	Leukemia, lymphoma, head-and-neck
Lycopene	Prostate
Phenyl isothiocyanate (PEITC) from cruciferous vegetables	Bladder, lung

Pine bark extract (Enzogenol)	Leukemia
Prebiotics (fermentable fiber, which produces butyrate, induces apoptosis)	Colon
Probiotics (<i>Lactobacillus salivarius</i>)	Oral
Propolis	Colon
Quercetin	Ovary
Red clover isoflavones	Prostate
Rosemary (carnosol)	Prostate, colon, skin, breast, kidney, liver
Sarsaparilla (<i>Smilax glabra</i>)	Multiple
Selenium	Colon, lung, prostate
Soy isoflavones (genistein, daidzein)	Prostate

7.4. Conclusion

We are awash in a sea of toxins and invisible radiation that constantly promotes malignant transformation of our cells, leading to persistently high rates of cancer. Despite the seemingly immeasurable amount of environmental toxins, there are five mechanisms through which they typically work to promote cancer. This allows us to identify nutrients that have cancer-fighting properties that work specifically against these mechanisms. Just as the chemicals that cause cancer do so by multiple mechanisms, natural products offer multiple, overlapping, and complementary approaches to cancer prevention.

By becoming familiar with the major cancer-inducing and cancer-promoting toxic influences in your world, you can then develop a supplement regimen that covers all five mechanisms by which we know that toxins and radiation induce cancerous changes. By choosing carefully from among the nutrients listed in this article, you can establish a solid cancer-fighting base in your own body—one that works with your natural defenses to defeat cancers before they get established.

Environmental toxins can cause serious health effects when exposure is allowed to accumulate. Problems usually result from prolonged or excessive

exposure. While it is impossible to completely eliminate exposure, a few simple steps will go a long way towards protecting you and your family:

1. Filtering home tap water and not storing water in plastic bottle.
2. Not using plastic plates to heat food in a microwave oven.
3. Reduce use of canned foods and eat mostly fresh or frozen foods.
4. Use baby bottles that are BPA free (or better yet use glass bottles) and look for toys labeled BPA free.
5. Use PVC-free containers. Buy plastic wrap and bags made from polyethylene and use glass containers. If you do use plastic containers, do not heat or microwave them.
6. Choose phthalate-free toys. Many large toymakers have pledged to stop using phthalates, but be sure to look for toys made from polypropylene or polyethylene.
7. Purchase phthalate-free beauty products. Avoid nail polish, perfumes, colognes, and other scented products that list phthalates as an ingredient.
8. Eating food grown without pesticides or chemical fertilizers.
9. Avoiding processed, charred and well-done meats.
10. Reducing cell phone usage.
11. Reducing exposure to radiation from medical sources by discussing with healthcare providers whether medical tests or procedures (such as CT-scans) that use radiation are really necessary.
12. Get your home air and water checked for radon.

There is no need to freak out over occasional exposure to environmental toxins. Just look for simple ways to reduce your everyday exposure. Make changes slowly, one at a time, in a manageable way, and you will decrease your risk with minimal stress.

8. Treating Cancer with Foods

8.1. Introduction

Cancers are primarily an environmental disease with **90–95% of cases** attributed to **environmental factors** and **5–10% due to genetics**. *Environmental*, as used by cancer researchers, means any cause that is not inherited genetically, not merely pollution. Common environmental factors that contribute to cancer death include **tobacco (25–30%)**, **diet and obesity (30–35%)**, **infections (15–20%)**, **radiation (both ionizing and non-ionizing, up to 10%)**, stress, lack of physical activity, and environmental pollutants.

There are more than 200 types of cancer. **About 35 percent of cancers are related to nutritional factors.** **Diet can influence some cancers.** **Cancers of the stomach, bowel, lung, prostate and uterus** are more likely to develop if your diet is high in fat and low in fruit, vegetables and fibre. Studies have linked consumption of red or processed meat to an increased risk of **breast cancer, colon cancer, and pancreatic cancer**, a phenomenon which could be due to the presence of carcinogens in foods cooked at high temperatures

The foods we eat can affect our risk of developing certain types of cancer. **High-energy and high-fat diets** can lead to obesity and are generally thought to increase the risk of some cancers. **Plant-based diets** high in fresh fruits, vegetables, legumes and whole grain foods may help to prevent cancer.

Diet is just one of the lifestyle factors that influence the risk of developing cancer. **Smoking, obesity, alcohol, sun exposure** and physical activity levels are also important. It is nearly impossible to prove what caused a cancer in any individual, because most cancers have multiple possible causes. For example, if a person who uses tobacco heavily develops lung cancer, then it was probably caused by the tobacco use, but since everyone has a small chance of developing lung cancer as a result of air pollution or radiation, then there is a small chance that the cancer developed because of air pollution or radiation.

Diet, physical inactivity, and obesity are related to approximately 30–35% of cancer deaths. In the United States excess body weight is associated with the development of many types of cancer and is a factor in 14–20% of all cancer deaths. Physical inactivity is believed to contribute to cancer risk not only through its effect on body weight but also through negative effects on **immune system** and **endocrine system**. More than half of the effect from diet is due to **overnutrition** rather than from eating too few healthful foods.

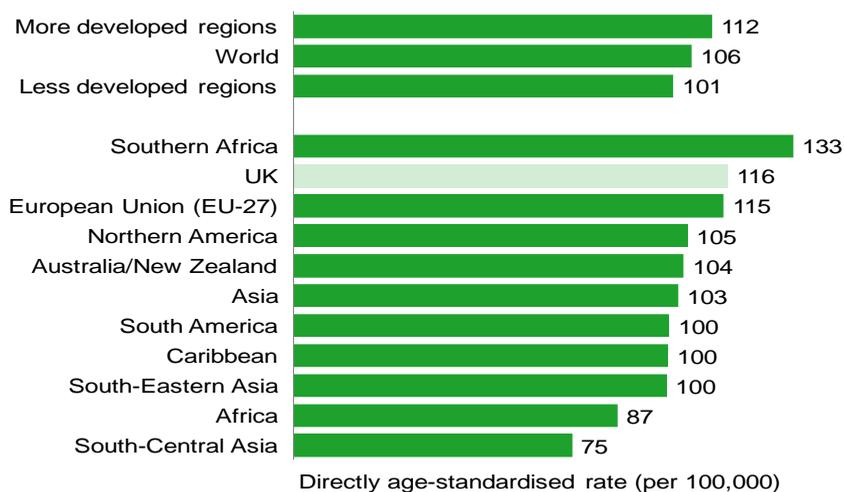
Diets that are very low in vegetables, fruits and whole grains, and high in **processed red meats** are linked with a number of cancers. A **high-salt diet** is linked to **gastric cancer**, **aflatoxin**, a frequent food contaminate, with **liver cancer**, and **betel nut** chewing with **oral cancer**. This may partly explain differences in cancer incidence in different countries. For example, **gastric cancer** is more **common in Japan** due to its high-salt diet and **colon cancer** is more **common in the United States** due to its high-fat diet.

Thus dietary recommendations for cancer prevention typically include a **vegan diet**: "mainly vegetables, fruit, whole grains, nuts & legumes", while completely omitting an intake of red meat, milk-products, animal fat and refined sugar.

8.2. Worldwide Cancer Mortality

An estimated 7.6 million people died from cancer worldwide in 2008. Because of the size of the populations, almost two-thirds of these deaths occur in the developing countries, and around 2% occur in the UK (Figure. 8.1).

Compared with incidence, the variations in cancer mortality across the world are much smaller, with less than a two-fold difference in rates between the regions. The highest mortality rates are seen in Southern Africa and the more developed regions of the world, such as the UK and the European Union, and the lowest rates are seen in the less developed regions, such as South-Central Asia and Africa. The UK mortality rate is slightly higher than the average in the more developed regions of the world, and more than 15% higher than the average in the less developed regions.



**Figure 8.1. Estimated worldwide cancer mortality 2008
(with comparison to UK 2008)**
Source: Cancer Research UK, 2012

8.2.1. Mortality in Males

An estimated 4.2 million men died from cancer worldwide in 2008. **Lung cancer** is by far the biggest killer, accounting for almost one in four (23%) cancer deaths in men (Figure 8.2).

Liver and stomach cancers are also big cancer killers in men worldwide (11% each). The prognosis for liver cancer is generally poor and much of the variation in incidence and mortality across the world can be explained by the distribution of Hepatitis B and C infection. **Stomach cancer** incidence and mortality has declined in many developed nations due to improvements in food preservation and storage, and falls in the prevalence of *Helicobacter pylori* infection.

In the UK, **lung cancer** also accounts for around one in four (24%) cancer deaths in men. **Prostate cancer** is responsible for relatively more deaths in the UK men than worldwide (12% vs. 6%), whereas deaths from **stomach** (4%) and **liver** (2%) cancers are relatively less common

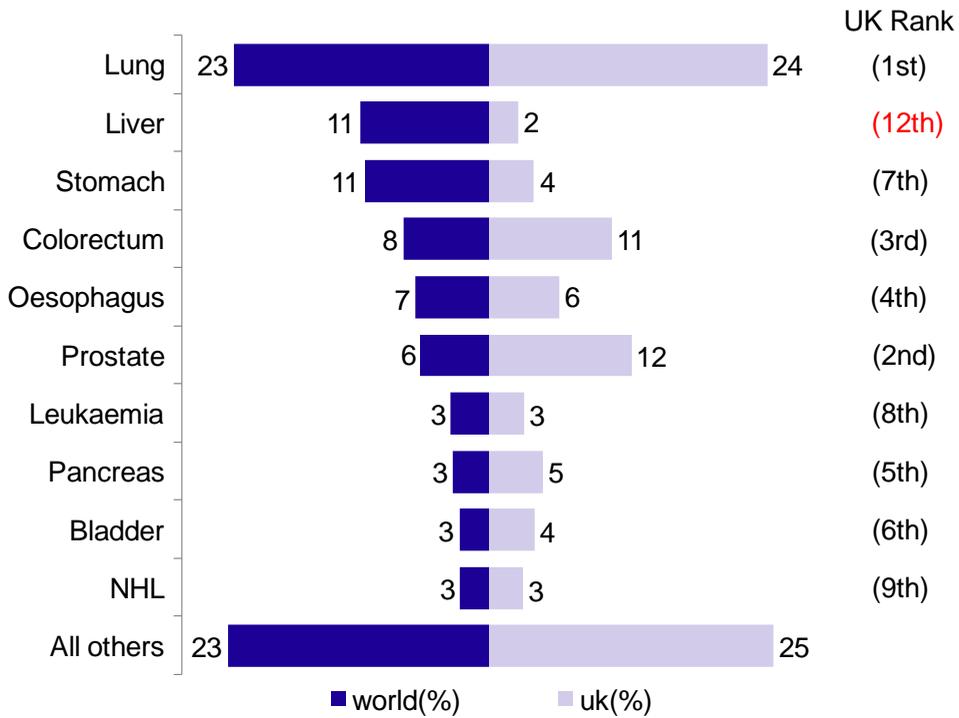


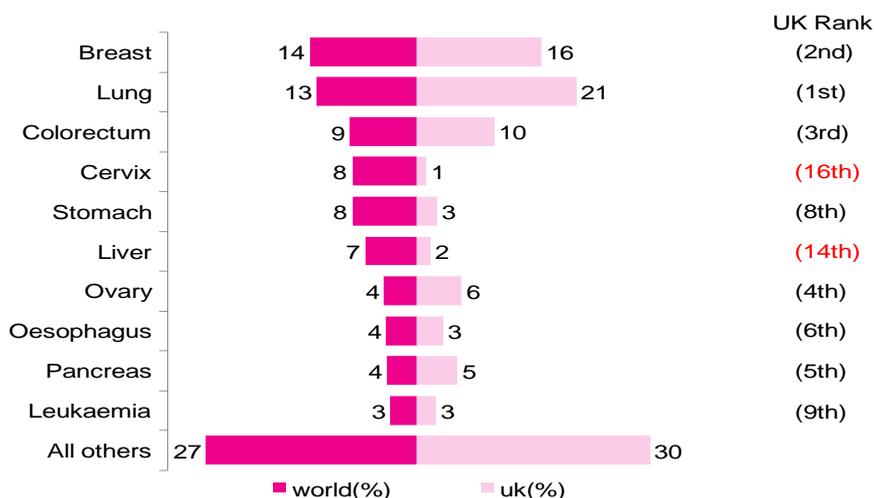
Figure 8.2. The ten most common cancer deaths in males worldwide 2008 (with comparison to UK 2008).

Source: Cancer Research UK, 2012

8.2.2. Mortality in Females

An estimated 3.4 million women died from cancer worldwide in 2008. **Breast and lung cancers** are the most common killers, accounting for 14% and 13% of deaths, respectively. **Colorectal** (9%), **cervical** (8%), **stomach** (8%) and **liver** (7%) cancers are also big cancer killers in women worldwide (Figure 8.3).

In the UK, **lung cancer** accounts for around one in five (21%) cancer deaths in women. **Breast cancer** is the next biggest killer (16%). Deaths from **cervical** (1%), **stomach** (3%) and **liver** (2%) cancers are relatively less common in UK women compared to many countries worldwide.



**Figure 8.3. The ten most common cancer deaths in females worldwide 2008 (with comparison to UK 2008).
Source: Cancer Research UK, 2012**

Common cancer types diagnosed with the greatest frequency are: Breast Cancer, Colon and Rectal Cancer, Cervical cancer, Lung Cancer, Stomach cancer, Uterus cancer, Liver cancer, Ovary cancer, Thyroid cancer, Kidney (Renal Cell) Cancer, Leukemia, Pancreatic Cancer and Prostate Cancer.

According to WHO Report (2008) cancer is a leading cause of death worldwide and accounted for 7.6 million deaths (around 13% of all deaths) in 2008. The main types of cancer are:

- lung (1.37 million deaths)
- stomach (736 000 deaths)
- liver (695 000 deaths)
- colorectal (608 000 deaths)
- breast (458 000 deaths)
- cervical cancer (275 000 deaths)

About 70% of all cancer deaths occurred in low- and middle-income countries and 30% of cancers could be prevented. A new report by the World Health Organization's International Agency for Cancer Research (IARC), (2011) suggests that the incidence of cancer worldwide will grow by 75% by the year 2030, nearly doubling in some of the developing countries. Deaths

from cancer worldwide are projected to continue to rise to over 13.1 million in 2030.

8.2.3. World Health Rankings with respect to Cancer Mortality

According to World Health Rankings with respect to cancer mortality (WHO, 2011), out of 192 countries Myanmar stood rank number 66. Myanmar cancer world ranks in type are shown in Table 8.1.

Table 8.1. Myanmar Cancer Ranks by Type per 100,000 Population. WHO data (2011)

No	Type	Rate	World Rank	No	Type	Rate	World Rank
1	Lung Cancers	18.31	72	10	Leukemia	4.31	67
2	Breast Cancer	13.47	126	11	Ovary Cancer	2.45	75
3	Liver Cancer	11.55	33	12	Bladder Cancer	2.09	99
4	Stomach Cancer	10.23	52	13	Prostate Cancer	1.86	170
5	Cervical Cancer	8.82	55	14	Pancreas Cancer	1.43	146
6	Colon-Rectum Cancers	8.41	81	15	Other Neoplasms	1.31	162
7	Oesophagus Cancer	7.82	29	16	Uterine Cancer	0.89	107
8	Oral Cancer	7.26	16	17	Skin Cancers	0.70	135
9	Lymphomas	5.49	88				

8.3. The top cancer-causing foods

While a high-energy, low-fibre diet may increase a person's risk of developing cancer, some individual foods have also been singled out as potentially causing cancer (carcinogenic). These include:

8.3.1. Red and processed meat.

Bowel and stomach cancer are more common in people who eat lots of red and processed meat. Red meat includes all fresh, minced and frozen beef, pork, lamb or veal. Processed meats have been preserved in some way other than freezing and include bacon, ham, salami, sausages, spam, corned

beef, black pudding, and tinned meat. There is now convincing scientific evidence that eating processed meat increases **bowel cancer** risk.

The World Cancer Research Fund (WCRF) has recently recommended people to avoid eating processed meat. Processed meats include any meat that has been preserved by curing, salting or smoking, or by adding chemical preservatives. These include hot dogs, ham, bacon and some sausages and burgers. Bacon and other cured or pickled meats contain a substance called nitrate, which has the potential to cause cancer.

8.3.2. Cured, pickled or salty foods

Bacon and other cured or pickled meats contain a substance called nitrate, which has the potential to cause cancer. To be on the safe side, it is best to limit the amount of cured meats in the diet because they are generally high in fat and salt. Foods that are high in salt or preserved using salt can increase your risk of **cancers of the stomach and nasopharynx** and should be consumed in limited amounts.

8.3.3. Burnt or barbecued foods

A group of carcinogenic substances called polycyclic aromatic hydrocarbons (PAHs) can be produced if foods are overheated or burnt. Charred or smoked foods may increase risk of cancer of the **bowel or gullet (oesophagus)**. However, when cooking, it's best to use relatively low temperature methods wherever possible and limit your intake of char-grilled meats and foods. Low temperature cooking methods include steaming, boiling, poaching, stewing, casseroles, braising, baking, stir-frying, microwaving or roasting. **Frying and baking meat at high temperatures** can also create chemicals called heterocyclic amines. These may increase risk of cancer of the **bowel or gullet (oesophagus)**.

8.3.4. Fat

Fat are a necessary part of our diet but high-fat diets can increase our risk of cancer, heart disease and other conditions. Vegetable foods are richer in monounsaturated or polyunsaturated fats, while meat is higher in saturated fats. There is evidence that eating too much saturated fat may increase your risk of **breast cancer**. Try not to eat too many fatty foods. In particular, try to

cut down on saturated fats as contained in fatty meat, biscuits, crisps, cheese and butter. Choose lean cuts of meat and semi-skimmed or skimmed milk. Try to avoid frying food in lots of oil - try steaming, braising or lightly grilling instead. A high-fat diet may lead to obesity, which is a risk factor for several cancers including **cancer of the colon, breast, kidney, oesophagus, gallbladder and endometrium**. A **high-fat diet** that comprises mostly animal fat sources (such as dairy products, fatty meats and takeaway foods) may increase the risk of **Prostate cancer**.

8.3.5. Peanuts

Some laboratory animals can develop cancer after eating peanuts that are contaminated with toxin-producing moulds. A good example of this is a toxin called aflatoxin that comes from a mould. It grows on stored food in hot and humid countries, especially on peanuts. Aflatoxin is known to help cause **liver cancer** so **anything that stops the mould from getting into the nuts helps to prevent cancer**.

8.3.6. French fries

Fries are made with hydrogenated oil and fried at high temperatures. They also contain cancer-causing acrylamides which occur during the frying process. Some chains even add sugar to their fry recipe to make them even more irresistible. Not only do they clog your arteries with saturated fat and trans fat, they also contain acrylamides. They should be called "**cancer fries**," not French fries.

8.3.7. Alcohol

Consuming alcohol increases the risk of cancers of the **mouth, pharynx, larynx, oesophagus, breast and liver**. Alcohol can increase risk of a number of cancers. A review in 2011 by Cancer Research UK suggests that around 4 out of 100 cancers (4%) are linked to alcohol. It increases **the risk of mouth cancer, liver cancer, breast cancer, bowel cancer, and throat cancer**, which includes **pharyngeal cancer, laryngeal cancer and cancer of the food pipe (oesophagus)**. 3.6% of all cancer cases and 3.5% of cancer deaths worldwide are attributable to consumption of alcohol. **Breast cancer** in women is linked with alcohol intake. Alcohol also increases the risk of cancers of the **mouth, esophagus, pharynx and larynx, colorectal cancer,**

liver cancer, stomach and ovaries. The **International Agency for Research on Cancer** (Centre International de Recherchesur le Cancer) of the **World Health Organization** has classified alcohol as a **Group 1 carcinogen**.

8.3.8. Genetically-modified organisms (GMOs)

It goes without saying that GMOs have no legitimate place in any cancer-free diet, especially now that both GMOs and the chemicals used to grow them have been shown to **cause rapid tumor growth**. But GMOs are everywhere, including in most food derivatives made from conventional corn, soybeans, and canola. However, you can avoid them by sticking with certified organic, certified non-GMO verified, and locally-grown foods that are produced naturally without biotechnology.

8.3.9. Microwave popcorn

They might be convenient, but those bags of microwave popcorn are lined with chemicals that are linked to causing **not only infertility but also liver, testicular, and pancreatic cancers**. The U.S. Environmental Protection Agency (EPA) recognizes the perfluorooctanoic acid (PFOA) in microwave popcorn bag linings as "likely" carcinogenic, and several independent studies have linked the chemical to causing tumors. Similarly, the diacetyl chemical used in the popcorn itself is linked to causing both **lung damage and cancer**.

8.3.10. Soda pop

Like processed meats, soda pop has been shown to cause cancer as well. Loaded with sugar, food chemicals, and colorings, soda pop acidifies the body and literally feeds cancer cells. Common soda pop chemicals like caramel color and its derivative 4-methylimidazole (**4-MI**) **have also specifically been linked to causing cancer**.

8.3.11. Conventional apples, grapes, and other 'dirty' fruits

Many people think they are eating healthy when they buy apples, grapes, or strawberries from the store. But unless these fruits are organic or verified to be pesticide-free, they could be a major cancer risk. The Environmental Working Group (EWG) found that up to 98 percent of all

conventional produce, and particularly the type found on its "dirty" fruits list, is contaminated with cancer-causing pesticides.

8.4. The Anti-Cancer Diets

Whether you have a history of cancer in your family, or are currently battling the disease, lifestyle factors, including your diet, can make a huge difference in helping you fight off cancer. Some foods actually increase your risk of cancer, while others support your body and strengthen your immune system. By making smart food choices, you can protect your health, feel better, and boost your ability fight off cancer and other disease.

8.4.1. Cancer prevention diet-1: Focus on plant-based foods

The best diet for preventing or fighting cancer is a predominantly plant-based diet that includes a variety of vegetables, fruits, and whole grains. A plant-based diet means eating mostly foods that come from plants: vegetables, fruits, nuts, grains, and beans.

There are many ways to add plant-based foods to your diet. A nice visual reminder is to aim for a plate of food that is filled at least two-thirds with whole grains, vegetables, beans, or fruit. Dairy products, fish, and meat should take up no more than a third of the plate. Keep in mind that you don't need to go completely vegetarian. Just as important, try to minimize or reduce the amount of processed foods you eat. Eat an apple instead of drinking a glass of apple juice, for example.

8.4.2. Cancer prevention diet -2: Bulk up on fiber

Another benefit of eating plant-based foods is that it will also increase your fiber intake. Fiber is found in fruits, vegetables, and whole grains. In general, the more natural and unprocessed the food, the higher it is in fiber. There is no fiber in meat, dairy, sugar, or "white" foods like white bread, white rice, and pastries

Fiber plays a key role in keeping your digestive system clean and healthy. It helps keep food moving through your digestive tract, and it also moves cancer-causing compounds out before they can create harm.

8.4.2.1. Simple ways to add more fiber to your diet:

- Use brown rice instead of white rice
- Substitute whole-grain bread for white bread
- Choose a bran muffin over a croissant or pastry
- Snack on popcorn instead of potato chips
- Eat fresh fruit such as a pear, a banana, or an apple (with the skin)
- Have a baked potato, including the skin, instead of mashed potatoes
- Enjoy fresh carrots and celery, instead of chips and a sour cream dip
- Use beans instead of ground meat in chili and casseroles

8.4.2.2. High-fiber, cancer fighting foods

Whole grain	whole-wheat pasta, raisin bran, barley, oatmeal, oat bran muffins, popcorn, brown rice, whole-grain or whole-wheat bread
Fruit	raspberries, apples, pears, strawberries, bananas, blackberries, blueberries, mango, apricots, citrus fruits, dried fruit, prunes, raisins
Legumes	lentils, black beans, split peas, lima beans, baked beans, kidney beans, pinto, chick peas, navy beans, black-eyed peas
Whole grain	whole-wheat pasta, raisin bran, barley, oatmeal, oat bran muffins, popcorn, brown rice, whole-grain or whole-wheat bread
Fruit	raspberries, apples, pears, strawberries, bananas, blackberries, blueberries, mango, apricots, citrus fruits, dried fruit, prunes, raisins
Vegetables	broccoli, spinach, dark green leafy vegetables, peas, artichokes, corn, carrots, tomatoes, brussels sprouts, potatoes

8.4.3. Cancer prevention diet - 3: Cut down on meat

Research shows that vegetarians are about fifty percent less likely to develop cancer than those who eat meat. High-fat diets have been linked to higher rates of cancer. And saturated fat is particularly dangerous. Depending on how it is prepared, meat can develop carcinogenic compounds.

8.4.3.1. Making better meat and protein choices

There is no need to cut out meat completely and become a vegetarian. But most people consume far more meat than is healthy. So it is needed to cut down your cancer risk substantially by reducing the amount of animal-based products and by choosing healthier meats.

- Keep meat to a minimum.** Try to keep the total amount of meat in diet to no more than fifteen percent of your total calories. Ten percent is even better.
- Eat red meat only occasionally.** Red meat is high in saturated fat, so eat it sparingly.
- Reduce the portion size of meat in each meal.** The portion should be able to fit in the palm of your hand.
- Use meat as a flavoring or a side, not the entrée.** You can use a little bit of meat to add flavor or texture to your food, rather than using it as the main element.
- Add beans** and other plant-based protein sources to your meats.
- Choose leaner meats**, such as fish, chicken, or turkey. If possible, buy organic.
- Avoid processed meats** such as hotdogs, sausage, deli meats, and salami.

8.4.4. Cancer prevention diet - 4: Choose your fats wisely

A major benefit of cutting down on the amount of meat you eat is that you will automatically cut out a lot of unhealthy fat. Eating a diet high in fat increases your risk for many types of cancer. But cutting out fat entirely isn't the answer, either. In fact, some types of fat may actually protect against cancer. The trick is to choose your fats wisely and eat them in moderation.

- Fats that increase cancer risk** – The two most damaging fats are saturated fats and trans fats. Saturated fats are found mainly in animal products such as red meat, whole milk dairy products, and eggs. Trans fats, also called partially hydrogenated oils, are created by adding hydrogen to liquid vegetable oils to make them more solid and less likely to spoil—which is very good for food manufacturers, and very bad for you.
- Fats that decrease cancer risk** – The best fats are unsaturated fats, which come from plant sources and are liquid at room temperature. Primary sources include olive oil, canola oil, nuts, and avocados. Also focus on omega-3 fatty acids, which fight inflammation and support brain and heart health. Good sources include salmon, tuna, and flaxseeds.

8.4.4.1. Tips for choosing cancer-fighting fats and avoiding the bad

- Reduce your consumption of red meat, whole milk, butter, and eggs**, as these are the primary source of saturated fats.
- Cook with olive oil instead of regular vegetable oil.** Canola oil is another good choice, especially for baking.
- Trim the fat off of meat** when you do eat it, and avoid eating the skin of the chicken.
- **Choose nonfat dairy products** and eggs that have been fortified with omega-3 fatty acids.
- Add nuts and seeds** to cereal, salads, soups, or other dishes. Good choices include walnuts, almonds, pumpkin seeds, hazelnuts, pecans, and sesame seeds.
- Limit fast food, fried foods, and packaged foods**, which tend to be high in trans fats. This includes foods like potato chips, cookies, crackers, French fries, and doughnuts.
- Eat fish once or twice a week.** Good choices include wild salmon, sardines, herring, and black cod. But be conscious of mercury, a contaminant found in many types of fish.

8.4.5. Cancer prevention diet - 5: Choose cancer-fighting foods

Your immune system keeps you healthy by fighting off unwanted invaders in your system, including cancer cells. There are many things you can eat to maximize the strength of your immune system, as well as many

cancer-fighting foods. But keep in mind that there is no single miracle food or ingredient that will protect you against cancer. Eating a colorful variety gives you the best protection.

- Boost your antioxidants.** Antioxidants are powerful vitamins that protect against cancer and help the cells in your body function optimally. Fruits and vegetables are the best sources of antioxidants such as beta-carotene, vitamin C, vitamin E and selenium.
- Eat a wide range of brightly colored fruits and vegetables.** Colorful fruits and vegetables are rich in phytochemicals, a potent disease-fighting and immune-boosting nutrient. The greater the variety of colors that you include, the more you will benefit, since different colors are rich in different phytochemicals.
- Flavor with immune-boosting spices and foods.** Garlic, ginger, and curry powder not only add flavor, but they add a cancer-fighting punch of valuable nutrients. Other good choices include turmeric, basil, rosemary, and coriander. Use them in soups, salads, casseroles, or any other dish.
- Drink plenty of water.** Water is essential to all bodily processes. It stimulates the immune system, removes waste and toxins, and transports nutrients to all of your organs.

8.4.6. Cancer prevention diet - 6: Prepare your food in healthy ways

Choosing healthy food is not the only important factor. It also matters how you prepare and store your food. The way you cook your food can either help or hurt your anti-cancer efforts.

8.4.6.1. Preserving the cancer-fighting benefits of vegetables

Here are a few tips that will help you get the most benefits from eating all those great cancer-fighting vegetables:

- Eat at least some raw fruits and vegetables.** These have the highest amounts of vitamins and minerals, although cooking some vegetables can make the vitamins more available for our body to use.

- **When cooking vegetables, steam until just tender using a small amount of water.** This preserves more of the vitamins. Overcooking vegetables removes many of the vitamins and minerals. If you do boil vegetables, use the cooking water in a soup or another dish to ensure you're getting all the vitamins.
- **Wash all fruits and vegetables.** Use a vegetable brush for washing. Washing does not eliminate all pesticide residue, but will reduce it. Choose organic produce if possible, grown without the use of pesticides.

8.4.6.2. Cooking and carcinogens

Carcinogens are cancer-causing substances found in food. Carcinogens can form during the cooking or preserving process—mostly in relation to meat—and as foods start to spoil. Examples of foods that have carcinogens are cured, dried, and preserved meats (e.g. bacon, sausage, beef jerky); burned or charred meats; smoked foods; and foods that have become moldy. Here are some ways reduce your exposure to carcinogens:

- **Do not cook oils on high heat.** Low-heat cooking or baking (less than 240 degrees) prevents oils or fats from turning carcinogenic. Instead of deep-frying, pan-frying, and sautéing, opt for healthier methods such as baking, boiling, steaming, or broiling.
- **Go easy on the barbecue.** Burning or charring meats creates carcinogenic substances. If you do choose to barbecue, don't overcook the meat and be sure to cook at the proper temperature (not too hot).
- **Store oils in a cool dark place in airtight containers,** as they quickly become rancid when exposed to heat, light, and air.
- **Choose fresh meats** instead of cured, dried, preserved, or smoked meats.
- **Avoid foods that look or smell moldy,** as they likely contain aflatoxin, a strong carcinogen. Aflatoxin is most commonly found on moldy peanuts. Nuts will stay fresh longer if kept in the refrigerator or freezer.

- **Be careful what you put in the microwave.** Use waxed paper rather than plastic wrap to cover your food in the microwave. And always use microwave-safe containers.

8.5. Best Foods for Cancer Prevention

About 35 percent of cancers are related to nutritional factors. To help prevent cancer, eat a wide variety of foods rich in nutrients that protect your body's cells from damage

8.5.1 Grapefruit

Vitamin C— an antioxidant found in many fruits and vegetables such as **grapefruit**, oranges, bell peppers, and broccoli — helps to prevent the formation of cancer-causing nitrogen compounds. Diets high in vitamin C have been linked to a **reduced risk of cancers of the stomach, colon, esophagus, bladder, breast, and cervix.**

8.5.2. Peanuts and peanut butter

Some research shows that eating a vitamin E-rich diet **reduces the risk of stomach, colon, lung, liver, and other cancers.** Adding vitamin E-rich foods like **peanuts, peanut butter**, almonds, almond butter, and sunflower seeds to your diet will help keep your cells' defenses strong.

8.5.3. Berries

Of all the fruits and vegetables studied, **berries** rank among the most likely to reduce cancer risk. Raspberries, blueberries, and cranberries in particular have shown very promising potential to help prevent cancer. An antioxidant called pterostilbene, found in high quantities in blueberries, has cancer-fighting properties. Laboratory animals fed with black raspberries had a 60 percent reduction in **tumors of the esophagus and an 80 percent reduction in colon tumors.**

8.5.4. Sweet potatoes

Beta-carotene is a powerful antioxidant. Studies have shown that people who eat a diet high in beta-carotene — found primarily in orange

vegetables and leafy greens — have a reduced risk of cancer, particularly of the **lung, colon, and stomach**. Among premenopausal women, one study found that eating a lot of vegetables that include beta-carotene, folate, vitamin C, and fiber-like **sweet potatoes** — reduced the risk of breast cancer by about half.

8.5.5. Wild salmon

Low vitamin D levels have been linked to several cancers, including colon and breast. Scientists theorize that vitamin D may help block the development of blood vessels that feed growing tumors and help stop the proliferation of cancerous and precancerous cells. It is recommend for eating plenty of vitamin D-rich foods, such as **wild salmon**, and choosing vitamin D-fortified dairy products, like milk and yogurt.

8.5.6. Ground flaxseed

Omega-3 fatty acids may help prevent cancer by inhibiting cancer cell proliferation and disrupting steps that are critical to tumor growth. **Omega-3 fatty acids** also help reduce inflammation, which means they could theoretically reduce the possibility of cellular mutations. But even if omega-3s don't directly reduce the risk of cancer, they certainly help keep our bodies strong and healthy. In addition to fatty fish and shellfish, mixing ground **flaxseed** into yogurt and smoothies is an excellent way to include more omega-3s in your diet.

8.5.7. Turmeric

Turmeric is the yellow-colored spice found in curry powder. Curcumin, the active ingredient in turmeric, functions as both an anti-inflammatory and an antioxidant, and it may help prevent cancer by interfering with aspects of cellular signaling. In laboratory animals, curcumin has been shown to help prevent **cancer of the breast, colon, stomach, liver, and lung**.

8.5.8. Tea

Tea contains compounds called catechins, compounds that scientists theorize may help stop the growth of cancer cells and prevent cellular

mutations that contribute to cancer development. In Japan, where **tea** is the preferred beverage, green tea consumption has been linked to **reduced risk of stomach cancer** among women. In China, **green tea** drinkers were found to have a **lower risk of developing rectal and pancreatic cancers** compared with non-tea drinkers. **Regular tea drinkers have also been shown to be at reduced risk for colon, breast, ovarian, prostate, and lung cancers.**

8.5.9. Cruciferous vegetables

All plant foods — grains, fruits, and vegetables — contain small amounts of phytonutrients: naturally occurring chemical compounds that are just as important as vitamins and minerals are for maintaining health. There are thousands of known phytonutrients, many of which have demonstrated the potential to protect us against cancer. Cruciferous vegetables like **broccoli, cauliflower, and cabbage** contain phytonutrients known as glucosinolates, which may help inhibit the metabolism of some carcinogens and stimulate the body's production of detoxification enzymes.

8.5.10. Pomegranates

Pomegranates are chock full of ellagic acid. In laboratory and animal studies, ellagic acid has been shown to inhibit cancer cell growth and deactivate cancer-causing compounds. To take advantage of these health properties, incorporate pomegranate seeds into smoothies or use them to top off a bowl of yogurt or cereal. Other foods rich in ellagic acid include **raspberries, blackberries, strawberries, walnuts, pecans, cranberries, and grapes** (red, black, purple).

8.6. Conclusion

Diet is just one of the lifestyle factors that influence the risk of developing cancer. Foods to limit in your diet or eat less of include: Fatty processed red meats, highly processed foods that are low in fibre and heavily salted and pickled foods. Eat unprocessed foods and base your diet largely on plants. Consume foods that have omega-3 fats and other essential fatty acids. Eat lots of fruits and vegetables; many common ones have known cancer-fighting properties. Get regular vigorous exercise, since tumors cannot thrive in highly oxygenated environments. Keep your blood sugar stable to avoid being an all-you-can-eat buffet for cancer cells.

Eat foods high in natural vitamin C, a nutrient that deters the conversion of nitrite into nitrosamine and promotes healthy immune function. Make sure you get adequate amounts of cancer-fighting vitamin D through exposure to sunlight — about 10 to 15 minutes each day if you have fair skin, or ten times as long if you have dark skin pigmentation. **Buy organic foods which are** grown without added pesticides or hormones. Avoid smoking and don't use conventional fragrance, cosmetics and personal care products - virtually all of them contain cancer-causing chemicals.

A potential cancer-causing compound called acrylamide forms as a result of the chemical changes that occur in foods when they're baked, fried, or roasted. Many foods with the greatest amounts of acrylamide are french fries and potato chips. French fries, potato chips, and baked sweets should be consumed in limited amounts.

Drinking excessive amounts of alcohol regularly increases your risk factor for many types of cancer. Studies suggest that men who consume 2 alcoholic drinks per day and women who have 1 alcoholic drink per day significantly increase their risk factors for certain types of cancer.

To help prevent cancer, eat a wide variety of foods such as: berries, grape fruit, cruciferous vegetables, turmeric and garlic. Garlic contains sulfur compounds that may stimulate the immune system's natural defenses against cancer, and may have the potential to reduce tumor growth. Studies suggest that garlic can reduce the incidence of stomach cancer.

Preventing cancer is actually quite straightforward. Even the World Health Organization says that 70 percent of all cancers can be prevented with simple changes in diet and lifestyle. The truth is that most people give themselves cancer through the foods, drinks and products they choose to consume.

9. Thyroid diseases and Environmental Toxins

9.1. Introduction

Thyroid disease is a medical condition that affects the function of the thyroid gland (the endocrine organ found at the front of the neck that produces thyroid hormones). The symptoms of thyroid disease vary depending on the type. There are four general types: (1) **hypothyroidism** (low function) caused by not having enough thyroid hormones, (2) **hyperthyroidism** (high function) caused by having too much thyroid hormones, (3) **structural abnormalities**, most commonly an enlargement of the thyroid gland, **tumors** which can be benign or cancerous, and (4) **abnormal thyroid function tests** without any clinical symptoms. Common hypothyroid symptoms include fatigue, low energy, weight gain, inability to tolerate the cold, slow heart rate, dry skin and constipation. Common hyperthyroid symptoms include irritability, weight loss, fast heartbeat, heat intolerance, diarrhea, and enlargement of the thyroid. In both hypothyroidism and hyperthyroidism, there may be swelling of a part of the neck, which is also known as **goiter**.

Hypothyroidism affects 3-10% percent of adults, with a higher incidence in women and the elderly. An estimated one-third of the world's population currently lives in areas of low dietary iodine levels, making iodine-deficiency the most common cause of hypothyroidism and **endemic goiter**. In regions of severe iodine deficiency, the prevalence of goiter is as high as 80%. In areas where iodine-deficiency is not found, the most common type of hypothyroidism is an autoimmune subtype called **Hashimoto's thyroiditis**, with a prevalence of 1-2%. As for hyperthyroidism, **Graves' disease**, another autoimmune condition, is the most common type with a prevalence of 0.5% in males and 3% in females. Although thyroid nodules are common, thyroid cancer is rare. Thyroid cancer accounts for less than 1% of all cancer in the UK, though it is the most common endocrine tumor and makes up greater than 90% of all cancers of the **endocrine glands**.

9.2. Thyroid gland

9.2.1. Anatomy of the Thyroid gland

The thyroid gland is located in the lower part of the neck, below the Adam's apple, wrapped around the trachea (windpipe). It has the shape of a butterfly: two wings (lobes) attached to one another by a middle part called the isthmus. Typically four parathyroid glands are found, two each at the back of thyroid lobe (Figure.9.1).

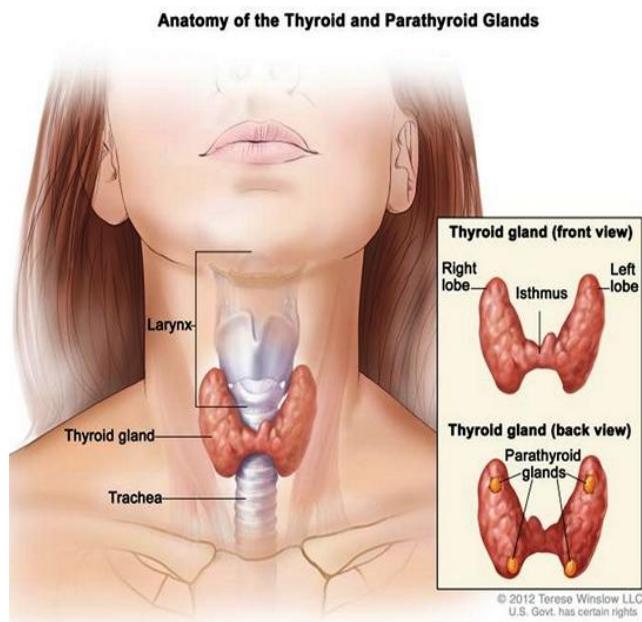


Figure 9.1. Anatomy of Thyroid and Parathyroid glands.

Source: Womens Health.gov

9.2.2. Functions of the Thyroid Gland

The thyroid uses iodine, a mineral found in some foods and in iodized salt, to make its hormones. The two most important thyroid hormones are thyroxine (T4) and triiodothyronine (T3). Thyroid stimulating hormone (TSH), which is produced by the pituitary gland, acts to stimulate hormone production by the thyroid gland. The thyroid gland also makes the hormone

calcitonin, which is involved in calcium metabolism and stimulating bone cells to add calcium to bone. Thyroid gland makes and stores hormones that help regulate the heart rate, blood pressure, body temperature, and the rate at which food is converted into energy. Thyroid hormones are essential for the function of every cell in the body.

It is important that T3 and T4 levels are neither too high nor too low. Two glands in the brain—the hypothalamus and the pituitary communicate to maintain T3 and T4 balance. The hypothalamus produces TSH Releasing Hormone (TRH) that signals the pituitary to tell the thyroid gland to produce more or less of T3 and T4 by either increasing or decreasing the release of a hormone called thyroid stimulating hormone (TSH). When T3 and T4 levels are low in the blood, the pituitary gland releases more TSH to tell the thyroid gland to produce more thyroid hormones. If T3 and T4 levels are high, the pituitary gland releases less TSH to the thyroid gland to slow production of these hormones.

T3 and T4 travel in your bloodstream to reach almost every cell in the body. T3 and T4 regulate your heart rate and how fast your intestines process food. So if T3 and T4 levels are low, your heart rate may be slower than normal, and you may have constipation/weight gain. If T3 and T4 levels are high, you may have a rapid heart rate and diarrhea/weight loss.

9.3. Thyroid Diseases

Thyroid diseases sometimes result from inappropriate TSH levels, or may be caused by problems in the thyroid gland itself.

9.3.1. Goiters

A thyroid goiter is a dramatic enlargement of the thyroid gland (Figure 9.2). Goiters are often removed because of cosmetic reasons or, more commonly, because they compress other vital structures of the neck including the trachea and the esophagus making breathing and swallowing difficult. Sometimes goiters will actually grow into the chest where they can cause trouble as well. Several x-rays will help explain all types of thyroid goiter problems.

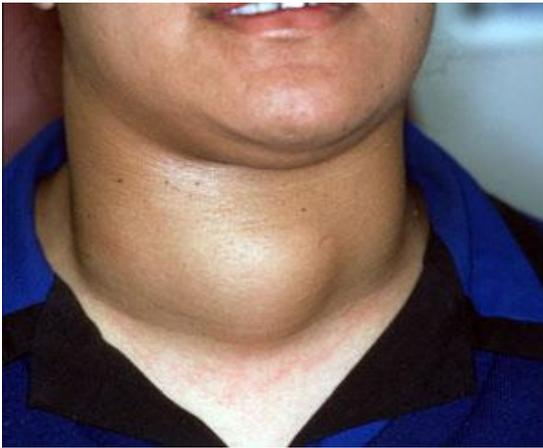


Figure 9.2. Goiter (Enlarged Thyroid Gland). Source: ehealthhut.com

9.3.1.1. Hashimoto's disease. A goiter can also result from an underactive thyroid (hypothyroidism). Hashimoto's disease damages your thyroid so that it produces too little hormone. Sensing a low hormone level, your pituitary gland produces more TSH to stimulate the thyroid, which then causes the gland to enlarge. Surgical removal and certain medications (e.g., amiodarone, lithium) can also cause hypothyroidism (Figure. 9.3).

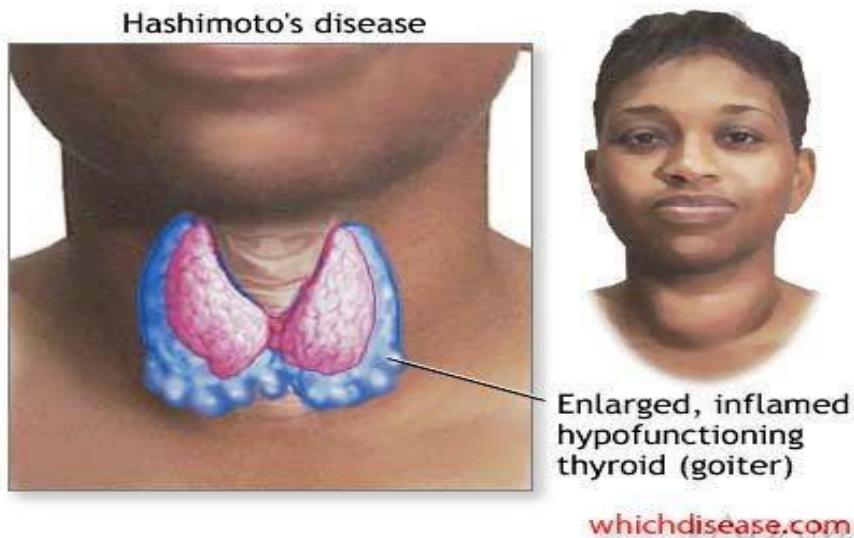


Figure 9.3. Hashimoto's disease. Source: whichdisease.com

9.3.1.2. Graves' disease. A goiter can sometimes occur when your thyroid gland produces too much thyroid hormone (hyperthyroidism). In Graves' disease, antibodies produced by your immune system mistakenly attack your thyroid gland, causing it to produce excess thyroxine. This overstimulation causes the thyroid to swell.

9.3.1.3. Multinodular goiter. In this condition, several solid or fluid-filled lumps called nodules develop in both sides of your thyroid, resulting in overall enlargement of the gland. Less than 5 percent of thyroid nodules are cancerous (Figure. 9.4).

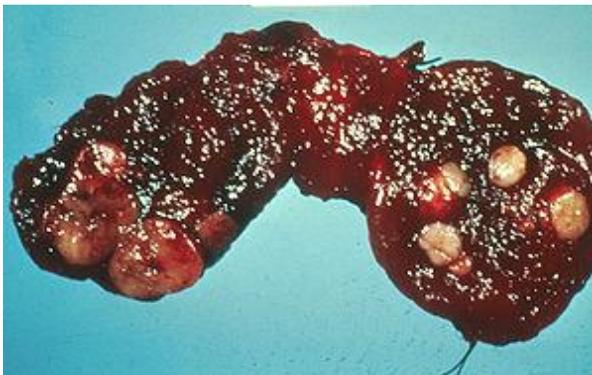


Figure 9.4. Multinodular goiter. Source: thyroidlife.blogspot.com

9.3.1.4. Solitary thyroid nodule. In this case, a single nodule develops in one part of your thyroid gland. There are several characteristics of nodules of the thyroid which make them suspicious for malignancy. Although as many as 50% of the population will have a nodule somewhere in their thyroid, the overwhelming majority of these are benign. Occasionally, thyroid nodules can take on characteristics of malignancy and require either a needle biopsy or surgical excision (Figure. 9.5).

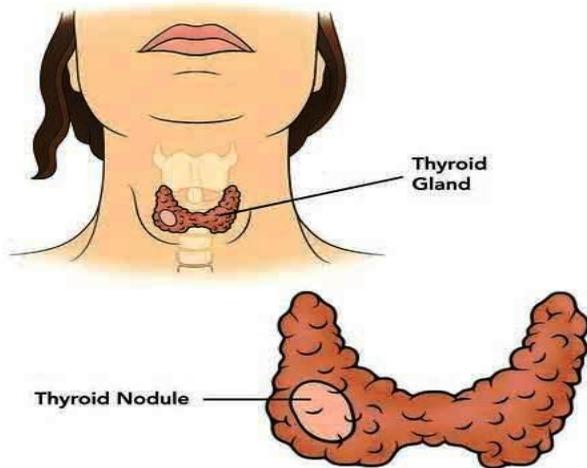


Figure 9.5. Solitary thyroid nodule. Source: thyroidlife.blogspot.com

9.3.2. Thyroiditis

Another possible cause of hyperthyroidism is a condition called **thyroiditis**. This condition occurs when the thyroid gland becomes inflamed. Depending on the type of thyroiditis, this may lead to temporary hyperthyroidism that might be followed by hypothyroidism.

9.3.3. Thyroid cancers

Thyroid cancer is a disease in which malignant (cancer) cells form in the tissues of the thyroid gland (Figure. 9.6). Most thyroid nodules are not cancer. There are four types of thyroid cancers: **papillary, follicular, anaplastic, and medullary cancer**. Medullary thyroid cancer is sometimes caused by a change in a gene that is passed from parent to child.

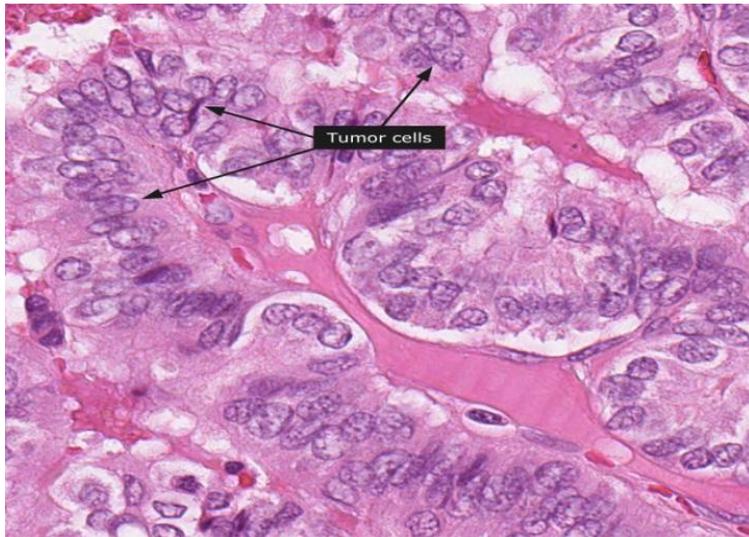


Figure 9.6. Cancer (Tumor) cells in Thyroid gland.
Source: thyroidcancer.com

9.4. Causes of Thyroid Diseases

9.4.1. Toxins that affect the thyroid hormones synthesis, metabolism and action

There are many toxins that affect the thyroid hormones synthesis, metabolism and action. **Perchlorates** that are used in rocket fuel additives are also found in well water, which happens to be commonly used in agriculture as well as for drinking. Perchlorates have been shown to cause thyroid disorders by altering the production of thyroid hormones. Perchlorates can also cause hypothyroidism and aggravate an already low thyroid function. Furthermore, when potential mothers drink water that has been contaminated with perchlorates, the byproducts can be passed down to the infant through breast milk. In addition to drinking water, perchlorates can be found in foods such as beef, lettuce, milk and berries, so consuming organic, hormone and additive free food is very important.

The list of toxins doesn't stop there; **thiocyanates** found in cigarettes, **dioxins** found in **pesticides** and **polychlorinated biphenyls (PCB's)** can all alter the thyroid gland function. **Bisphenol A** found in consumer goods such

as water bottles and sports equipment also affects the thyroid gland by affecting the receptor function.

Harmful elements, such as **fluoride**, **chloride** and **bromide** exposure, can cause inhibition of iodine transport and block the conversion of the T4 to T3 – the active thyroid hormone. **Heavy metal toxicity** from **lead**, **aluminum** and **mercury** can all trigger antibodies that trigger auto-immune thyroid conditions, such as the Grave's disease and Hashimoto's disease.

9.4.2. Thyroid diseases sometimes caused by problems in the thyroid gland itself.

Thyroid diseases sometimes result from inappropriate TSH levels, or may be caused by problems in the thyroid gland itself. **The most common cause of hypothyroidism is Hashimoto's thyroiditis**, an autoimmune condition where the body makes antibodies that destroy parts of the thyroid gland. Surgical removal and certain medications (e.g., amiodarone, lithium) can also cause hypothyroidism.

Other causes of hypothyroidism include pituitary problems, hypothalamus problems, and iodine deficiency (rare in North America, but affects nearly 2 billion people worldwide). Some babies are born with hypothyroidism - this is called **congenital hypothyroidism**.

There are different causes of hyperthyroidism. Graves' disease is the most common cause of hyperthyroidism. This condition occurs when the immune system produces an antibody that stimulates the entire thyroid gland; this leads to overactivity and higher levels of thyroid hormones.

Another form of hyperthyroidism is called **toxic nodular goiter or toxic thyroid adenoma**. Adenomas, abnormal nodules of tissue in the thyroid, constantly produce thyroid hormones even when they are not needed.

Secondary hyperthyroidism is caused when the pituitary gland makes too much TSH, leading to constant stimulation of the thyroid gland. A pituitary tumour may cause TSH levels to rise. More rarely, the pituitary gland becomes insensitive to thyroid hormones, no longer responding to high levels.

Another possible cause of hyperthyroidism is a condition called *thyroiditis*. This condition occurs when the thyroid gland becomes inflamed. Depending on the type of thyroiditis, this may lead to temporary hyperthyroidism that might be followed by hypothyroidism.

There are four types of thyroid cancers: papillary, follicular, anaplastic, and medullary cancer. These are associated with radiation treatment to the head, neck, or chest. Radiation treatment for benign (non-cancerous) conditions is no longer carried out in these areas, but was more common in the past. In other cases, a genetic mutation might be associated with thyroid cancer, either alone or in conjunction with other types of cancers (e.g., multiple endocrine neoplasia, BRAF gene mutations). Less commonly, other cancers might metastasize to the thyroid (e.g., lymphoma, breast cancer).

9.4.3. Radiation released from nuclear bomb and accidents

An association of autoimmune thyroid diseases with radiation exposure from nuclear fallout, from the atomic bomb detonated in Japan and the Chernobyl disaster, have been reported. Radiation is clearly associated with thyroid hypofunction, thyroid nodules, and thyroid cancer. These effects are generally associated with greater radiation exposure and can be tracked with position of the exposed individual at time of the accident, or, in the case of the Chernobyl exposure, the pattern of the wind currents dispersing radiation. Other exposures, such as Hanford in Washington State, the site of radioisotope production for the atomic bomb, have not been associated with measurable thyroid effects in those exposed compared to controls.

The initial study of atomic bomb survivors in Japan showed an increase in the incidence of thyroid autoimmunity, but a more recent study, with longer follow-up, did not. A study of children exposed to radiation from Chernobyl showed an increase in thyroid size, higher serum TSH levels, and a greater incidence of thyroid autoantibody positivity, although iodine intake in the area was low. A more recent study of those exposed to radiation from the Chernobyl accident showed an increase in thyroid autoantibodies, but not an increase in the incidence of hypothyroidism. It is possible that there is not an increase in autoimmune thyroid disease in response to radiation exposure.

Another possibility is that the increase in autoimmune thyroid disease occurs during a specific window of time after exposure, in contrast to the

increase incidence of thyroid hypofunction, nodules, and cancer that persists after exposure for as long as it has been studied. This is consistent with studies in Japan and Chernobyl showing an increase in autoimmune thyroid disease in the initial follow-up studies after exposure, but not in later studies with longer follow-up. Workers at nuclear power plants did not have an increase in thyroid cancer or autoimmune thyroid disease, but did have a higher serum TSH than controls. A long-term follow-up of children exposed to nuclear testing in Nevada, from 1951–1962, showed an increased risk of autoimmune thyroid disease for those in the highest dose exposure group.

9.5. Symptoms and Complications

Hypothyroidism results in low levels of T4 and T3 in the blood. Not having enough T4 and T3 in the blood causes your metabolism to slow down.

Common symptoms include:

- coarse and dry hair
- confusion or forgetfulness (often mistaken for dementia in seniors)
- constipation
- depression
- dry, scaly skin
- fatigue or a feeling of sluggishness
- hair loss
- increased menstrual flow (women)
- intolerance to cold temperatures
- irritability
- muscle cramps
- slower heart rate
- weakness
- weight gain

If hypothyroidism isn't treated, the symptoms will progress. Rarely, a severe form of hypothyroidism, called *myxedema*, can develop. Symptoms of myxedema include:

- low body temperature
- dulled mental processes

- congestive heart failure, a condition where the heart cannot pump enough blood to meet the body's needs

Myxedema coma occurs in people with severe hypothyroidism who have been exposed to additional physical stresses such as infections, cold temperatures, trauma, or the use of sedatives. Symptoms include loss of consciousness, seizures, and slowed breathing.

Hyperthyroidism results in high levels of T4 and T3 circulating in the blood. These hormones speed up your metabolism. Some of the most common symptoms include:

- increased heart rate with abnormal rhythm or pounding (palpitations)
- high blood pressure
- increased body temperature (feeling unusually warm)
- increased sweating
- clamminess
- feeling agitated or nervous
- tremors in the hands
- feeling of restlessness even though the person is tired or weak
- increased appetite accompanied by weight loss
- interrupted sleep
- frequent bowel movements, sometimes with diarrhea
- puffiness around the eyes, increased tears, sensitivity to light, or an intense stare
- bone loss (osteoporosis)
- stopped menstrual cycles

Graves' disease, in addition to the common symptoms of hyperthyroidism, may cause a bulge in the neck (goiter) at the location of the enlarged thyroid gland. It also might cause the eyes to bulge out, which may result in double vision. Sometimes, the skin over the shins becomes raised.

If hyperthyroidism is left untreated or is not treated properly, a life-threatening complication called *thyroid storm* (extreme overactivity of the thyroid gland) can occur. Symptoms include:

- confusion
- coma

- fever
- high blood pressure
- irregular heartbeat, which can be fatal
- jaundice associated with liver enlargement
- mood swings
- muscle wasting
- restlessness
- shock
- weakness

Thyroid storm, considered a medical emergency, can also be triggered by trauma, infection, surgery, uncontrolled diabetes, pregnancy or labour, or taking too much thyroid medication.

9.6. Making the Diagnosis

Thyroid disease suspected by clinical history and physical exam is confirmed by laboratory tests. Laboratory tests usually measure levels of TSH and thyroid hormones. Serology tests can measure the levels of antibodies associated with hypothyroidism and hyperthyroidism. If your doctor suspects thyroid cancer, a biopsy can be used to sample the thyroid tissue and test for cancer.

Another method called a *functional stimulation test* can be used to distinguish whether the pituitary and thyroid glands are the source of medical symptoms. Ultrasounds and nuclear thyroid scans allow for visual and functional examination of the thyroid gland or of nodules.

9.7. Treatment

The usual treatment for hypothyroidism is thyroid hormone replacement therapy. With this treatment, synthetic thyroid hormone (e.g., levothyroxine¹) is taken by mouth to replace the missing thyroid hormone. Treatment is usually life-long.

Most people who take thyroid replacement therapy do not experience side effects. However, if too much thyroid hormone is taken, symptoms can

include shakiness, heart palpitations, and difficulty sleeping. Women who are pregnant may require an increase in their thyroid replacement by up to 50%. It takes about 4 to 6 weeks for the effect of an initial dose or change in dose to be reflected in laboratory tests.

Hyperthyroidism can be treated with iodine (including radioactive iodine), anti-thyroid medications or surgery.

Radioactive iodine can destroy parts of the thyroid gland. This may be enough to get hyperthyroidism under control. In at least 80% of cases, one dose of radioactive iodine is able to cure hyperthyroidism. However, if too much of the thyroid is destroyed, the result is hypothyroidism. Radioactive iodine is used at low enough levels so that no damage is caused to the rest of the body. It isn't given to pregnant women because it may destroy the thyroid gland of the developing fetus.

Larger doses of regular iodine, which does not destroy the thyroid gland, help block the release of thyroid hormones. It is used for the emergency treatment of thyroid storm, and to reduce the excess production of thyroid hormones before surgery.

Anti-thyroid medications (e.g., propylthiouracil* or methimazole) can bring hyperthyroidism under control within 6 weeks to 3 months. These medications cause a decrease in the production of new thyroid hormones by the thyroid gland. Larger doses will work more quickly, but may cause side effects including skin rashes, nausea, loss of taste sensation, liver cell injury, and, rarely, a decrease of blood cell production in the bone marrow.

Surgical removal of the thyroid gland, called *thyroidectomy*, is sometimes necessary. It may be required if there are cancerous nodules; if a non-cancerous nodule is causing problems breathing or swallowing; if the person cannot take radioactive iodine or antithyroid medications, or if these do not work; or if a nodule that contains fluid continues to cause problems. Removing the thyroid gland leads to hypothyroidism, which must then be treated with thyroid hormone therapy for the rest of a person's life.

Sometimes your doctor may recommend other medications to help control symptoms of hyperthyroidism, such as shakiness, increased heart rate, anxiety, and nervousness. However, these won't cure thyroid dysfunction.

Treatment for thyroid cancers often involves some combination of thyroidectomy (surgical removal of the thyroid gland), radioactive iodine, radiation therapy (less common), anticancer medications, and hormone suppression.

9.8. Prevention of Thyroid Diseases

The thyroid is known as your metabolic master because it controls every single cell in the body. Without enough of the crucial thyroid hormone, every system in the body slows down, resulting in fatigue, weight gain, constipation, hair loss, dry skin and more.

9.8.1. Try your best to avoid environmental toxins

According to the National Institute of Environmental Health Sciences, long-term exposure to endocrine disruptors—chemicals that interfere with your body's endocrine system—may trigger endocrine problems in humans. A few to be aware of are perfluorinated chemicals (PFCs) in some carpets, flame-resistant and waterproof clothing, and non-stick cookware, all of which were linked to thyroid disease in a 2010 study. Similarly, in 2011 researchers found that exposure to phthalates (found in fragranced products and soft plastics) and bisphenol-A (found in some hard plastics and canned food linings, although many manufacturers are removing them) could cause disruptions in thyroid hormone levels.

It is also recommended avoiding antibacterial soaps that contain triclosan, an ingredient that has altered hormone regulation in studies of animals (human studies are still ongoing), according to the FDA.

Although it would be impossible to avoid these completely, the key is to reduce your exposure as much as you can, especially if you're pregnant or have little ones in the house—developing fetuses, infants, and children are more vulnerable to any effects of environmental chemicals. Other things you can do include, choosing more fresh or frozen foods over canned, storing food in porcelain or glass rather than plastics, and keeping your home well-ventilated.

9.8.2. Protect yourself against X-rays

The thyroid gland is one of the organs most sensitive to the risk of radiation – whether it's from a dental X-ray, mammogram, MRI or general background radiation. A study from **National Cancer Institute** compared the number of dental X-rays received by a group of thyroid cancer patients prior to their diagnosis with the number received by a group of similar individuals without thyroid cancer. Overall, those who had dental X-rays were twice as likely to develop thyroid cancer. The patients who received more than 10 X-rays had more than five times the risk of developing cancer than someone who had not had any dental X-rays.

To protect yourself it is recommend requesting a thyroid shield (a lead apron that covers your neck area) whenever you have to undergo radiation, especially for children and young adults when the thyroid is still developing.

9.8.3 Stop smoking

Cigarette smoke has various toxins — thiocyanate in particular — that are especially dangerous to the thyroid, and can trigger thyroid disease in susceptible people. Cigarette smokers also are more likely to develop thyroid eye complications of Graves' disease, and treatments for those eye problems are less effective in smokers. One study also suggested that smoking may increase the risk of hypothyroidism in patients with **Hashimoto's thyroiditis**, a common autoimmune disease.

9.8.4. Test for and treat thyroid antibodies

In the study of 21 patients with euthyroid Hashimoto's Thyroiditis, who had normal range TSH, but elevated antibody levels, half of the patients were treated with levothyroxine for a year, the other half were not treated. After 1 year of therapy with levothyroxine, the antibody levels and lymphocytes (evidence of inflammation) decreased significantly only in the group receiving the medication. Among the untreated group, the antibody levels rose or remained the same.

The researchers concluded that preventative treatment of normal TSH range patients with Hashimoto's disease reduced the various markers of autoimmune thyroiditis, and speculated that that such treatment might even be

able to stop the progression of Hashimoto's disease, or perhaps even prevent development of the hypothyroidism.

9.8.5. Detox to save your thyroid

There are thyroid disruptors all around us — in plastic water bottles, pop cans and even lurking in your shampoo bottles. A connection between common chemicals called phthalates and thyroid hormone levels was confirmed by **the University of Michigan** in a large-scale study.

Researchers at the **University of California** also linked canned soups to changes in thyroid hormone levels. They discovered that as BPA levels doubled, participants experienced a decrease in T4 levels, putting them on the path towards hypothyroidism.

9.8.6. Too much soy is not healthy

Overconsumption of soy has been linked to increased risk of thyroid disease. In particular, the craze for soy powders, smoothies, soy patties, and potions—everything from Revival Soy to Isoflavone pills—has been problematic, as too much of these unnatural forms of soy may put a strain on your immune system and trigger thyroid problems in susceptible people. Stick to natural forms of soy— tofu, tempeh, miso soup — in moderation, and you should be fine, but stay away from the pills, powders, smoothies, creams and other may actually ruin your health.

9.8.7. Try Selenium: A Thyroid Super-Nutrient

An inexpensive supplement may help prevent certain forms of thyroid disease. The mineral selenium can help prevent thyroiditis and some autoimmune hypothyroidism conditions.

9.8.8. Keep Potassium Iodide on hand for a Nuclear Emergency

Potassium iodide is an over-the-counter supplement that, when taken within hours after a nuclear accident — or attack on nuclear facilities— may help protect the thyroid from risk of thyroid disease and thyroid cancer.

After the Chernobyl accident, residents of Poland received mass distribution of this supplement in the time when the radioactive cloud was passing over them, while residents of Ukraine and Russia did not. Subsequently, thyroid cancer and thyroid disease rates have skyrocketed in the unprotected areas, while Poland has had no similar increase in thyroid problems.

If you live within 50—100 miles of a nuclear plant, it's wise to have your own stockpile of potassium iodide on hand for each member of your family, and keep some at work, and in the car. Take it only if warnings are issued and the government instructs you via the Emergency Broadcast System to take potassium iodide, and the specific levels recommended.

9.8.9. Watch out for Fluoride: What's good for teeth may be bad for the Thyroid

Fluoride is used as a drug to treat hyperthyroidism, an overactive thyroid, because it makes the thyroid underactive quite effectively. This is why you need to be particularly careful in today's over-fluoridated world, where water supplies, plus toothpastes, plus dental rinses, and other treatments all want to put more fluoride into our systems. Some experts recommend you avoid fluoridated water -- try a bottled water that you've verified is fluoride free, and avoid fluoride treatments and fluoridated toothpaste.

9.9. Conclusion

Thyroid disorders, in particular, goiter, thyrotoxicosis, and hypothyroidism are common in daily clinical practice in Myanmar. Patients with goiter are commonly seen in the northern and eastern parts of the country where most of the people live in hilly regions. Goiter is also common in the delta and coastal region of the country since the soil in these regions is usually deficient in iodine content. On the recommendation of the 24th National Health Committee meeting (1997), the Ministry of Mines issued a regulation which stated that all factories should be licensed for production on iodized salt for animal and human consumption in 1999. Universal Salt Iodization (USI) is the major intervention for elimination of iodine deficiency disorders.

When a goiter becomes very large, it can press against other structures in the neck and cause symptoms including: trouble with swallowing or breathing, hoarseness or voice change, pain in the neck and weakness. So you have to make self-examination of your neck that will help you find Thyroid conditions including goiters, nodules and thyroid cancer. Here is how to do a thyroid neck check.

1. Stand in front of a mirror, so that you can see your neck without any obstruction by taking off scarves, neckties, turtlenecks, so your view of your neck area is clear.
2. Stretch your neck back, chin toward the ceiling.
3. With your neck still stretched back, drink a glass of water.
4. Check your neck, looking for any enlargement in the thyroid area. Feel the area around the thyroid to see if you can detect any enlargement, bumps or lumps.

If you can detect any sort of problem, feel any enlargement or lump, you should see a doctor for evaluation right away. The goiter may be pressing on your jugular vein, windpipe, esophagus, or the nerve that runs to your larynx. The enlarged thyroid requires treatment and may need to be surgically removed. The doctor will usually need to order one or more of the following tests:

- **Thyroid hormone level test.** This **blood test** checks the levels of hormones secreted by the thyroid gland. The hormone levels are usually normal even if there are nodules. However, there are times when abnormal hormone levels are also benign. Therefore, your doctor will probably order other tests.
- **Thyroid ultrasound.** This test uses sound waves to determine if a nodule is solid or a fluid-filled cyst. (The risk of cancer is higher in solid nodules.) This test also checks on the growth of nodules and helps find nodules that are difficult to feel. In addition, thyroid **ultrasound** is sometimes used to help guide placement of the needle during a fine needle biopsy.
- **Fine-needle biopsy of the thyroid gland.** With this test, the doctor uses a very thin needle to take a sample of cells from one or more thyroid nodules. The samples are then sent to a laboratory, and most turn out to be non-cancerous. However, if the test results are “suspicious,” your doctor may repeat this test. The doctor may also

suggest you have surgery to remove the nodules in order to make an accurate diagnosis.

- **Thyroid scan.** In this test, a small amount of radioactive iodine is given orally. The doctor will check to see how much of the radioactive iodine is absorbed by the nodules and how much is absorbed by normal thyroid tissue. This will help diagnose if the nodules are malignant (cancer).
- **CT scan (CAT scan):** A procedure that makes a series of detailed pictures of neck areas, taken from different angles. The pictures are made by a computer linked to an x-ray machine.

As prevention is better than cure, you should avoid radiation exposure to the neck. Thyroid cancer is not preventable, but the possibilities of cancer can be lowered with sensible life style choice such as not smoking and avoid environmental toxins. You can further protect your health by eating a balanced diet, getting enough sleep, exercising several times a week, and getting fresh air and relaxation. Healthy living is an important part of recovery from thyroid condition.

10. Effects of Pesticides on Human Health

10.1. Introduction

Pesticides are chemical and biological products which have been specifically developed to control pests, weeds and diseases particularly in the production of food. Pests, weed and diseases can all have devastating effects on the quantity and quality of crops grown for human consumption and without pesticides we could lose one third of world crops each year. There are several classes of pesticide including insecticides (control insect infestations), fungicides (control the spread of fungal diseases), herbicides (control the competing effects of weeds), molluscicides (control the destructive effects of slugs and snails) and rodenticides (control the activities of rats and mice).

Pesticides are sprayed onto food, especially fruits and vegetables, they secrete into soils and groundwater which can end up in drinking water, and pesticide spray can drift and pollute the air. Pesticides are found as common contaminants in soil, air, and water, and on non-target vegetation in our urban landscapes. Once there, they can harm plants and animals ranging from beneficial soil microorganisms and insects, non-target plants, fish, birds, and other wildlife. Animals may be poisoned by **pesticide residues** that remain on food after spraying, for example when wild animals enter sprayed fields or nearby areas shortly after spraying. Problems which have been identified as a result of mishandling and misuse of pesticides include: health hazard to applicators and workers in pesticide production plants; and residues in food and export crops.

Pesticides can enter the human body through inhalation of aerosols, dust and vapor that contain pesticides; through oral exposure by consuming food and water; and through dermal exposure by direct contact of pesticides with skin. Roughly 90 percent of pesticide intake is ingested with food; much of the remainder has its source in pesticide-contaminated air and water. Across the globe pesticides have been found in human blood, urine, breast milk, semen, adipose tissue, amniotic fluid, infant meconium and umbilical cord blood.

A small amount of some toxins has quick action and can kill within a short period. And other toxins that are slower acting, may take a long time to

cause harm to the human body. Children seem to be greatly susceptible to the toxic effects of pesticides. Researchers report that the dangers of pesticides can start as early as fetal stages of life. What you also need to understand is that toxins from pesticides can remain in the body and build up in the liver. And, even at "safe" levels your reactions can be mild to severe. High levels of exposure can be fatal. How do you know if you're going to be ill? You don't; you just have to hope for the best. How will you be affected? Well, you don't really know how your body will react to the toxins until it happens. Several factors determine how your body will react including your level of exposure, the type of chemical you ingest, and your individual resistance to the chemicals.

10.2. Pesticide Residues

Any pesticide that remains in or on food or feed is called a residue. When a crop is treated with pesticide, a very small amount of pesticide, or indeed what it changes to in their plant (its 'metabolites' or 'degradation product'), can remain in the crop until after it is harvested. This is known as the 'residue'. Pesticide residue may be present in:

- Fresh or tinned fruit and vegetables, or
- Processed food and drink made from the crop (e.g. juice, bread or any other manufactured food or drink),
- Fresh or processed animal products (if the animals have been fed on crop treated with pesticide).
- Fish and aquatic organisms in pesticide contaminated water.

The level or amounts of residues present are expressed in milligrams of the chemical in a kilogram of crop/food/commodity (mg/kg). These are very small amounts. 1 mg/kg is the same as 1 part per million (ppm).

10.2.1. Organochlorine residues in fish from Lake Victoria, Kenya

Eighty-two samples of either Nile perch fish fat or muscle were collected from the Kenyan region of Lake Victoria for detection of organochlorine residues. Nine organochlorine residues were detected in the following percentages: α -BHC/HCB-40%; γ -BHC/HCB-40%; γ -BHC/HCB/lindane-4%; aldrin-9%; dieldrin-1%; p, p'-DDE-73%; p, p'-DDD-9%; o, p'-DDT-170; and p, p'-DDT-11%. All levels of organochlorine residues

were below the Maximum Residue Limit (MRL), apart from just one sample of fish fat which had 4.51 ppm of DDT above MRL (Mitema, 2009).

10.2.2. Analysis of environmental chemical residues in products of emerging aquaculture industry in Uganda

A study was conducted to analyse market-regulated heavy metals (lead, mercury and cadmium) and organochlorine pesticides in samples of 38 farmed fish comprising Nile tilapia (*Oreochromis niloticus*) (20 samples) and African catfish (*Clarias gariepinus*) (18 samples) from ten selected fish farms in Uganda.

- Lead was detected in all the 38 samples (maximum = 1.08 mg kg⁻¹ (dry weight)),
- Mercury in 31 out of 38 samples (maximum= 0.35mg kg⁻¹ (dry weight)),
- Cadmium in two samples (maximum = 0.03 mg kg⁻¹ (dry weight)).
- Pesticides detected were: 4,4'-dichloro-diphenyl-trichloroethane (DDT) and endosulfan sulphate, which were found in one fish sample (both 0.002 mg kg⁻¹ (wet weight)).

The levels of contaminants were below the US Food and Drug Administration (USFDA) action levels and European Union maximum residue limits (MRLs) (Bagumire, 2008).

10.2.3. Organochlorine pesticide residues in paddy fish in Malaysia and the associated health risk to farmers

Paddy fish (*Trichogaster pectoralis* Regan) were collected from five sampling locations in a major paddy-growing area of Malaysia and analysed for organochlorine residues. Pesticide residues found in the fish samples were aldrin/dieldrin, chlordane, HCH, and DDT. Maximum intake level for aldrin/dieldrin approached the acceptable daily intake as recommended by FAO/WHO. However, this study considered only fish. Rice also makes up a large portion of the diet. Published values for DDT in rice show a mean of 200 ng/g. Besides the rice, the farmer may also ingest residues in vegetables, poultry, and pork. The total daily intake of pesticide residues by the

Malaysian paddy farmer may be considerably increased by consumption of other contaminated food. Hence, the sum of all these separate components could exceed the FAO/WHO ADI for some compounds. It is thus not surprising that the mean residue level of organochlorine insecticides in the sera of paddy-farmers (185 ng/g) was nearly twice that found in the general Malaysian population. All possible routes of exposure (i.e., in food, water, and direct) should be considered when assessing the potential health risk to the Malaysian paddy farmers (Donald *et al*, 1984).

10.2.4. Residual Toxicity of different insecticides to *Channa punctata*

In the research work on residual toxicity of different insecticides to freshwater fish, *Channa punctata* it was observed that Kitazin 48% EC remained toxic to the test fish only for 2.4 days. In contrast, DDT 25% EC was degraded very slowly and residual toxicity on fish was 45 days (Table 18). The residual toxicity periods for Lindane L- 20 and Endrin 19.5% EC were 34 days and 28 days respectively. DDT 25% EC, Lindane L-20 and Endrin 19.5% EC were persistent in the water owing to its resistance to biochemical degradation and had long term residual toxicity. **Such pesticides will prolong the exposure period to fish and may also produce delayed mortality and fish may take up some of the pesticide residues into their body.** They should be replaced with insecticides of short term residual toxicity: Kitazin 48% EC, Elsan 50% EC, Padan 50% EC, EPN 45% EC, Diazinon 40% EC, Sumithion 50% EC and Furadan 3-G (Table. 10.1).

Table 10.1. Residual toxicity of 10 different insecticides to *Channa Punctate*. (Kyaw Myint Oo, 1991)

No	Insecticide	Residual toxicity (days)
1	Kitazin 48%EC	2.4
2	Elsan 50% EC	4.8
3	Padan 50% EC	5.6
4	EPN 45% EC	7.0
5	Diazinon 40% EC	8.8
6	Sumithion 50% EC	10.0
7	Furadan 3-G	11.6
8	Endrin 19.5% EC	28.0
9	Lindane L-20	34.0
10	DDT 25% EC	45.0

10.2.5. Residues in fruits and vegetables

Below is a table of 27 fruits and vegetables that were tested by the US. Environmental Working Group for pesticide residue (Table 10.2).

Table 10.2. Residue in fruits and vegetables (US. Environmental Working Group for Pesticide Residue, 2008)

Rank (Worst to best)	Commodity	Combined Score	% of sample tested with detectable pesticides	Average Amount (ppm) of all pesticides found
1	Peaches	100	96.6 %	1.134
2	Apples	96	93.6%	0.894
3	Pepper	86	81.5%	0.138
4	Strawberries	83	92.3%	0.799
5	Cherries	75	91.4%	0.290
6	Pears	65	86.2%	0.586
7	Potatoes	58	81.0%	1.655
8	Carrots	57	81.7%	0.046
9	Green beans	55	67.6%	0.199
10	Cucumbers	52	72.5%	0.057
11	Plums	46	74.0%	0.666
12	Oranges	46	85.1%	0.100
13	Grapes	46	60.5%	0.104
14	Cauliflower	39	84.6%	0.004
15	Mushrooms	37	60.2%	0.158
16	Lemon	31	55.6%	0.188
17	Grapefruit	31	62.9%	0.056
18	Tomatoes	30	46.9%	0.029
19	Sweet Potatoes	30	58.4%	0.198
20	Watermelons	25	38.5%	0.021
21	Papaya	21	23.5%	0.053
22	Cabbage	17	17.9%	0.121
23	Bananas	16	41.7%	0.029
24	Mango	9	7.1%	0.057
25	Pineapples	7	7.1%	0.057
26	Avocado	1	1.4%	0.001
27	Onions	1	0.2%	0.000

The foods are ranked from worst to best (descending). The table will tell you what percentage of that particular fruit/vegetable had pesticides on it and average amount (in ppm) of all pesticides found on each fruit/vegetable. For example, peaches were the worst. 96.6% of peaches had pesticides on them. Average amount of all pesticides found was 1.134 ppm.

10.2.6. Residues of Cypermethrin and Methamidophos on cauliflower at various intervals after treatment

The residues of Cypermethrin and Methamidophos on cauliflower at different time intervals after treatment are shown in Table 3. The data showed that the dissipation rate of methamidophos was slower than that of cypermethrin. The residues of both insecticides were detected even on 15th day after treatment, indicating their prolong persistence nature in cauliflower (Table 10.3).

Table 10.3. Residues of Cypermethrin and Methamidophos on Cauliflower at various intervals after treatment. (Barkat Ali Khan, 2003)

Days after Treatment	Cypermethrin Residues(mg/kg)	Metamidophos Residues(mg/kg)
0	3.74	4.41
1	1.04	2.55
3	0.73	1.62
5	0.35	1.12
7	0.17	0.83
10	0.10	0.68
15	0.07	0.52

10.2.7. Pesticide residues in organisms of Malaysian waters

Organochloride compounds were widely used in rapidly developing countries in South East Asia for agriculture, pest control and for public health purposes. From the study in the Straits of Melacca, pp'-DDE pollution in mussel (*Perna viridis*) tissue from six stations on Penang Island ranged from 3.7 to 17.4 ppb (dry weight basis) and the concentration of 1.2 to 38 ppb for DDT was also found in tissue of the same species (Table 10.4).It can be said

that the level of persistent organic chemicals in the Malaysian waters are still at the acceptable level and comparable to the results in other Southeast Asia countries. Today, it is believed that the level of persistent organic chemicals in Malaysia and Southeast Asia water had been increased because of rapid population growth and urbanization.

**Table 10.4. Pesticide residues in organisms of Malaysian waters.
(Somchit, 2009)**

No	Organism	Location	Concentration
1	<i>Perna viridis</i> (Mussel)	Penang Malaysia	3.7 – 17.4 ppb (dry weight basis) pp'-DDE 1.2 – 38 ppb DDT
2	<i>Anader granosa</i> (Blood Cockles)	Penang, Malaysia	0.21 ppb Lindane 0.08ppb Aldrin 0.15 ppb Endrin

10.2.8. Levels of organochlorine pesticide residues in meat

Organochlorine pesticide residues (Lindane, Aldrin, Dieldrin, Endosulfan, and DDT) were found in beef samples from Buoho abattoirs in Ghana . Results in table 10.5 indicate that the highest concentration of DDT in beef fat from Buoho was 844.28 ug/kg. This is about two times higher than the WHO recommended maximum residue limit of 500 ug/kg. The average concentration of 6.01 ug/kg Dieldrin recorded from beef fat is higher than the maximum level of 6.00 ug/kg recommended by WHO. DDT residues, like the other organochlorines, concentrate more in the fat than in the muscle or lean meat.

Table 10.5. Levels of pesticide residues in beef fat from Buoho, Ghana (ug/kg). (Darco, 2007)

	Lindane	Aldrin	Endosulfan	Dieldrin	DDT
Mean	1.79	4.11	2.28	6.01	403.82
SD	0.38	8.19	1.74	5.14	276.88
Max.	2.11	24.32	6.53	15.37	844.28
Min.	1.24	0.56	0.40	2.21	37.60

10.2.9. Organochlorine Pesticides BHC and DDE in human blood in and around Madurai, India

Blood samples are taken from two groups of people, one that has direct exposure to pesticides (agriculturists & public health workers) the second group, which has indirect exposure to pesticides through food chain. The objective of the investigation was to analyze the blood of the patients with minimum health complaints and skin diseases for the residue of the organochlorine pesticides DDE and BHC using Gas Chromatography. High concentrations of BHC and DDE were noted both in the serum of agricultural and non-agricultural people. The pesticide residue concentration in serum ranges from 0.006 to 0.130 ppm for BHC and 0.002 to 0.033 ppm for DDE (Kallidass, 2006).

The earlier studies on the residues of these pesticides in milk and infant foods showed increased residue levels beyond applicable limit. From 2,205 samples of bovine milk 85% of the milk samples contained HCH isomers (alpha, beta, gamma or delta) above the tolerance limits. In the case of gamma-HCH, 28% of samples were above the tolerance limit of 0.01 mg/kg. In the case of DDT, 82% of the milk samples were contaminated, about 37% of these have above the tolerance limit of 0.05 mg/kg as set out under the Prevention of Food Adulteration Act on a whole milk basis. More over the increased level of the BHC in the non agriculturalists and agriculturalists irrespective of their exposure either direct or indirect shows that this pesticide

residue are highly accumulated in their serum by the contamination of their food by this pesticide rather than their exposure. This may be the reason for the persistence of these pesticides residues in both groups' agriculturalists and non-agriculturalists taken for this study.

10.2.10. Organochlorine pesticide residues in human milk of a Hmong hill tribe living in Northern Thailand

In December 1998 whole breast milk samples from 25 Hmong mothers living in the village of Mae Sa Mai, 40 km north of Chiang Mai City, Northern Thailand, were collected and analysed for DDT, heptachlor, HCB and HCH residues. DDT was detected in all samples with a median and maximum level of 209 and 2012 ng of total DDT isomers per millilitre of milk, respectively. The median and highest percentages of p,p'-DDT were 23.2 and 44.7%. In 15 samples heptachlor was detected in the metabolized form of heptachlor-epoxide with a median value of 4.4 ng/ml.

The estimated daily intakes of DDT, heptachlor and heptachlor-epoxide by the infants exceeded up to 20 times the acceptable daily intakes as recommended by the FAO and WHO. The mean sum-DDT residues with 14.96 mg/kg milk fat, as well as the estimated daily intakes by the infants are one of the highest reported in the 1990s. The fact that the mother breast-feeds her first child and that she originally comes from a region where DDT is still in use as a vector control agent, as well as the former use of organochlorine pesticides (OCPs) in agriculture, seem to be the main factors for high DDT and other OCP residues in the mothers' milk.

10.2.11. Evaluation of organochlorine pesticide residues in human serum from an urban and two rural populations in Portugal

Organochlorine pesticide residues were measured in human serum from an urban and two rural populations in Portugal, in an attempt to evaluate the contamination level of Portuguese population. Serum levels of 12 residues were determined using a validated methodology that included gas chromatography-electron-capture detection. The determination was made as an attempt to point out the differences of contamination between rural and urban populations; and among these, if it could be established a relation with sex and with age of individuals.

*p,p'*DDE, α -hexachlorocyclohexane (HCH), *p,p'*DDD, and β -HCH were the most frequently identified residues. *p,p'*DDE concentrations ranged from undetected to 390.5 $\mu\text{g/l}$ in urban samples, and from undetected to 43.5 $\mu\text{g/l}$ and to 171.2 $\mu\text{g/l}$ in both rural samples. Maximum α -HCH concentration level was 114.4 $\mu\text{g/l}$ in urban samples, 261.3 and 45.5 $\mu\text{g/l}$ in both rural samples. Mean total DDT levels were always higher than mean total HCH levels. About *p,p'*DDE, in all three populations, the majority of the results above the limit of quantification were found among female sex. The analysis of different age groups showed that younger groups continue to reveal contamination. Comparing obtained results with others from Europe, Asia and America, it was observed that Portugal is between the highest levels of contamination (Susana, 2003).

10.3. Effects of Pesticides on Human Health

The effects of pesticides on human health are more harmful based on the toxicity of the chemical and the length and magnitude of exposure. Farm workers and their families experience the greatest exposure to agricultural pesticides through direct contact with the chemicals. But every human contains a percentage of pesticides found in fat samples in their body. Children are most susceptible and sensitive to pesticides due to their small size and underdevelopment.

Fruits and vegetables are known to protect against heart disease and cancer. They are rich in vitamins, minerals, fiber, and health-promoting phytochemicals. However, many fruits and vegetables test positive for pesticide residues, with about one-third of them showing up with multiple residues. Fish are an important part of a healthy diet. Some fish caught in lakes, rivers, oceans and estuarines, however, may contain pesticide residues that could pose health risks if these fish are eaten in large amounts. Eating fish containing pesticide residues may cause birth defects, liver damage, cancer, and other serious health problems.

Researchers reveals that prolonged exposure to pesticide residues may increase the risk of various cancers and neurological problems (such as Parkinson's disease), and impair the immune system. Studies have proved that farmers are at a potentially high risk of developing leukemia, lymphomas, and

cancers of organs like the prostate, stomach, skin and brain. Pesticides are linked to chronic health disorders and ailments. Exposure to pesticides can range from mild skin irritation to birth defects, cancers, blood and nerve disorders, hormone disruption, and even coma or death.

10.3.1. Asthma

Researchers found an association between asthma and use of pesticides by male farmers. Although this study involved adults, it raises concerns about children's exposures to pesticides used in the home or residues brought home on parents' clothes or equipment.

10.3.2. Birth Defects

The commonly used pesticide, chlorpyrifos caused severe birth defects in four children exposed in utero. Chlorpyrifos is used widely as an agricultural chemical, but is also the most common pesticide used indoors to kill termites, fleas, roaches and in pest control strips. A study in Minnesota found significantly higher rates of birth defects in children born to pesticide applicators and in regions of the state where chlorophenoxy herbicides and fungicides are widely used.

In California, mothers living and working in agricultural areas with high pesticide use had a higher risk for giving birth to children with limb reduction defects. A study of pregnant women in Iowa and Michigan found that women exposed to multiple pesticides had an increased risk of giving birth to a child with cleft palate. Researchers found higher rates of numerous birth defects in children born to Norwegian farmers exposed to pesticides, including hormone effects like hypospadias and undescended testicles.

10.3.3. Neurological Effects

Pesticides can be potent neurotoxins. When people are exposed to neurotoxins they may feel dizzy, lightheaded, confused and may have reduced coordination and ability to think. These are the short-term effects, while long term exposure can result in reduced IQ and learning disability, associated with permanent brain damage. In spite of wide reporting of adverse symptoms, until recently, few studies could link permanent brain damage to such

exposures. There is new evidence that prolonged exposure to pesticides in areas where they are used routinely may cause permanent brain damage to children who live in these areas.

Significant reductions in plasma cholinesterase are associated with a number of acute and subacute neurotoxic effects: muscle tremors, twitching and weakness, anorexia, nausea, vomiting, bronchospasm, excessive pupil contraction, blurred vision, headache, cognitive impairment, seizure, and coma.

An intermediate syndrome involving respiratory paralysis and failure may occur 1-5 days after exposure to some organophosphates. Irreversible weakness, ataxia (failure of muscle coordination), and paralysis may occur 2-5 weeks later. This delay is due to degeneration of myelin sheaths covering large nerve fibers. Acute clinical organophosphate and carbamate poisoning is likely to appear when cholinesterase activity is inhibited by 50% or more, and 30% inhibition has been proposed by WHO as a hazard level.

10.3.4. Cancers

The cumulative effects of widespread chronic low-level exposure to pesticides only partially is understood. However, mounting evidence suggests a strong correlation between pesticide exposure and the development of cancer in humans. Of the 80,000 pesticides and other chemicals in use today, 10 percent are recognized as carcinogens. Cancer-related deaths in the United States increased from 331,000 in 1970 to 521,000 in 1992, with as estimated 30,000 deaths attributed to chemical exposure. Farmers are prone to certain cancers, including stomach, prostate, and brain cancer, non-Hodgkin's lymphoma, and leukemia. The National Cancer Institute (NCI) has linked the common weed killer 2,4-D to non-Hodgkin's lymphoma in several studies.

Another NCI study found a link between breast cancer in women and elevated levels of DDE, a metabolite of the pesticide DDT, in their fat tissue. Women with the highest levels of exposure to DDT had four times the breast cancer risk of women with the least exposure. The link is not proven yet. Research also indicates that youngsters in homes where household and garden pesticides are used are seven times as likely to develop childhood leukemia.

A recent study of pesticides and childhood brain cancers has revealed a strong relationship between brain cancers and compounds used to kill fleas and ticks. The specific chemicals associated with children's brain cancers were pyrethrins and pyrethroids (which are synthetic pyrethrins, such as permethrin, tetramethrin, allethrin, resmethrin, and envalerate), and chlorpyrifos.

10.3.5. Hormone Disruption

While some substances cause physical birth defects, others can cause subtle hormonal effects on the developing fetus or affect a child's functional capacities. Hormone disruptors have been linked to many health problems including reproductive cancers. Pesticides like 2,4-D, lindane and atrazine, are known hormone-disruptors. Aside from increases in reproductive cancers, increasing rates of the following conditions are reported.

10. 3.5.1. Endometriosis, a disease in which the uterine tissue grows outside the uterus, and a common cause of infertility was virtually unheard of twenty years ago. It now affects 5.5 million women in the U.S. and Canada, about 10-20% of women of childbearing age.

10.3.5.2. Hypospadias, a condition in which the urethra is near the base of the penis, not the end as it should be, has doubled in the last 10 years.

10. 3.5.3. Undescended testicles, which is linked with later risk of testicular cancer, is increasing. Researchers reported a doubling in cases between 1962 and 1982 in England and Wales.

10.3.5.4. Precocious puberty in girls is now common. A study of 17,077 girls in the US found that the onset of puberty for white girls was 6-12 months earlier than expected and African-American girls experienced puberty 12-28 months earlier than whites.

10.3.5.5. Reduced sperm counts are documented. Between 1938 and 1990, sperm counts dropped 1.5% each year for American men and 3.1% per year for European men. There was no decrease in men from non-western countries. Low sperm count is a marker for testicular cancer.

10.3.5.6. Fertility Problems are becoming more common and now affect more than two million couples in the U.S.

10.4. Home Food Preparation to reduce Exposure to Pesticide Residues on Fresh Fruits, Vegetables, Meats, Poultry and Fish

Consumers can take the following steps to reduce their potential exposure to pesticide residues in food. To reduce the amounts of pesticide residues in food, consumers can wash, peel, cook and dip their food; trim the fat from meat; and eat a variety of foods to avoid repeated exposures to a pesticide typically used on a given crop, however, many pesticides are systemic, which means they penetrate into the fruit and vegetable itself and cannot be washed off. Many pesticides are also by design created to be rain-proof. The following basic food preparation practices and habits can further reduce your exposure to pesticide residues on fresh fruits, vegetables, meats, poultry and fish.

10.4.1. Trim tops and remove the very outer portions of celery, lettuce, spinach and other leafy vegetables that may contain the bulk of pesticide residues.

10.4.2. Washing vegetable and fruit. Household washing procedures are normally carried out with running or standing water at moderate temperatures. You can wash using a very diluted solution of mild dishwashing detergent (1 tsp detergent per gallon, or 4 liters, water). For grapes, strawberries, green beans, and leafy vegetables, swirl the foods in a dilute solution of dish detergent and water at room temperature for 5 to 10 seconds, then rinse with slightly warm water. For the other fruits and vegetables, use a soft brush to scrub the food with the solution for about 5 to 10 seconds, then rinse again with slightly warm water. Carrot, cabbage and cauliflower, can be washed with 1 per cent tamarind solution. Other produce (acorn squash, apples, apricots, carrots, peaches, pears, potatoes, tomatoes) was scrubbed with a brush using a soapy solution of warm water, then rinsed.

10.4.3. Peel fruits with higher residue levels. Peeling fruits, especially peaches, pears and apples, will help remove residues. Be sure to keep the peelings out of the compost. The outer leaves of vegetables often contain residues of pesticides applied during the growing season. Therefore, peeling or trimming procedures reduce the residues levels in leafy vegetables.

Peeling of root, tuber and bulb vegetables with a knife is common household practice. Many examples show that most of the residues concentration is located in or on the peel. Peeling may remove more than 50% of the pesticide residues present in the commodity. Thus, removal of the peel achieves almost complete removal of residues, so leaving little in the edible portions. This is especially important for fruits which are not eaten with their peels, such as bananas or citrus fruits.

Peeling or trimming of carrot reduced the residues of chlorfenvinphos, primiphos-methyl, quinalphos, triazophos. After application of thiometon on cucumbers, no reduction of residue levels could be detected in the peeled cucumbers. Peel and trim fruits like mango, citrus and kiwi and vegetables like gourds to reduce dirt, bacteria, and pesticides, if needed and likely to have high levels of pesticide residue.

10.4.4. Cooking. Several studies were reported on the dissipation of pesticides in crops during cooking. Residues of organophosphorus pesticides chlorfenvinphos, fenitrothion, isoxathion, methidathion and prothiophos decreased during the cooking process corresponding to the boiling time. Cooking of endosulfan (Endoin 35 EC) spiked meat resulted in 58.33–64.59% reduction in α -endosulfan and 55.93–61.60% reduction in β -endosulfan. Among the cooking methods, pressure cooking was most effective in reducing both α - and β -endosulfan. **Don't microwave foods in plastic containers.** Chemicals from the plastic container can become absorbed by food during microwaving. Cover with waxed paper or paper towel instead of plastic wrap to keep food from spattering.

10.4.5. Dipping in chemical solution. Sodium chloride solution is largely used to decontaminate the pesticide residues from different fruits and vegetables. There are several studies to prove the efficacy of salt water washing to dislodge the pesticides from crops. In this process, sample of chopped fruits and vegetables is put in a beaker containing 5% sodium chloride solution. After 15 minutes the plant samples are gently rubbed by hand in salt solution and salt water is decanted.

Dipping of green chillies in 2% salt solution for 10 minute followed by water wash has proved to be effective, facilitating the removal of 32.56 and 84.21% residues correspondingly at 0 and 5 days after spray of triazophos (700g a.i./ha). Following same technique it was also observed that 20.56 and

66.93% reduction correspondingly on 0 and 5 days after spraying of cypermethrin in chillies.

10.4.6. Trim the fat from meat, and fat and skin from poultry and fish. Animal products can contain synthetic hormones, antibiotics and organochlorine chemicals, such as dioxin, DDT and other pesticides, which concentrate in animal fat. The same chemicals that accumulate in animal fats are transferred to our own when we eat them. Then they linger there for years quietly causing damage. Trim all fats and skins and broil meats and fish so that the fats drain away. Avoid frying, which will lock in the contaminants.

10.5. The Health Benefits of Eating Organic Food

We can help protect ourselves from a toxic environment by eating organic food. Fruit and vegetable certified as ‘Organic’ is your best assurance of pesticide-free status. Buying organic, in-season produce from your local market is the best assurance of pesticide-free produce. In the 21st century, a growing number of men and women from around the world are recognizing the benefits of organic food. These people are beginning to appreciate the health benefits that can be derived from organic food. Table 10.6 highlights differences between conventional and organic farming.

Table 10.6. Differences between conventional and organic farming

Conventional farmers	Organic farmers
Apply chemical fertilizers to promote plant growth.	Apply natural fertilizers, such as manure or compost, to feed soil and plants.
Spray insecticides to reduce pests and disease.	Use beneficial insects and birds, mating disruption or traps to reduce pests and disease.
Use chemical herbicides to manage weeds.	Rotate crops, till, hand weed or mulch to manage weeds.
Give animals antibiotics, growth hormones and medications to prevent disease and spur growth.	Give animals organic feed and allow them access to the outdoors. Use preventive measures — such as rotational grazing, a balanced diet and clean housing — to help minimize disease.

Conventional farming practices utilize an extensive array of different types of chemicals. For example, as a general rule in the cultivation of crops of all varieties, the typical farmer utilizes chemical insecticides, herbicides

and fertilizers. Without fail, when these crops are harvested, those remains at least trace amounts of these various chemicals that were utilized during the planting and growing processes. When a consumer purchases and consumes these conventionally grown food items, the trace chemicals end up in a person's body. Over time, these insecticides, herbicides and fertilizers can accumulate in a person's body.

In organic farming, the farmers grow their crop entirely without synthetic pesticides and even without synthetic fertilizers. One of the most significant health benefits associated with eating organic food rests in the fact that these products are free from any potentially harmful chemicals. Organically grown food items are pure and completely wholesome. A number of recent research studies have also considered the benefits of eating organic food. Some of these research endeavors focused on the effects organic foods might have on lowering the incidence of certain diseases, including some types of cancer. It has been demonstrated that organically grown fruits and vegetables have significantly higher levels of antioxidants than do conventionally cultivated food products. Because organically grown fruits and vegetables are higher in antioxidants, these items have been demonstrated to work to reduce the risk of certain types of cancers.

Consumption of processed meat such as bacon, sausages and meat pies has been linked to cancer. By reducing your meat intake you will also be reducing toxic residues such as DDT, dioxin and PCBs which are found in meat fat, especially important for breastfeeding mothers. By eating organic meat, particularly pork, chicken and eggs you are reducing toxic residues, avoiding antibiotics and growth hormones given to the animals.

10.6. Conclusion

Because pesticides have many uses, we may be exposed to them in various ways, through food, water and air. Roughly 90 percent of pesticide intake is ingested with food; much of the remainder has its source in pesticide-contaminated air and water. Many of the pesticides found as residues in our food have serious long term effects including hormonal disruption, cancer, immune system suppression, nervous system damage, genetic damage and birth defects. Children are, as a rule, more vulnerable to toxins than are adults.

So we can help protect ourselves from a toxic environment by doing our best to reduce our exposure to pesticide residues in food.

We can minimize our exposure to pesticide residues by choosing our food and taking some simple steps to prepare them. To reduce the amounts of pesticide residues in food, consumers can wash, peel, cook and dip their food; trim the fat from meat, and fat and skin from poultry and fish. Eat a variety of foods to avoid repeated exposures to a pesticide typically used on a given crop. You may also choose organically grown fruits and vegetables. Organically grown fruits and vegetables have significantly higher levels of antioxidants than do conventionally cultivated food products. Organic foods might have an effect on lowering the incidence of certain diseases, including some types of cancer.

11. Pesticide Residue Monitoring and Food Safety

11.1. Introduction

Pesticides may be used in a variety of different ways during the production of food. They may be used by farmers to control the growth of weeds, or prevent crop damage by insects, rodents and molds. They may be used on food crops after harvest to prolong their storage life. Pesticides may also be used on animal farms to control insect pests. Sometimes, small amounts of pesticides used in these ways can be found in or on foods. The pesticides found in or on foods are called “**residues**”. Some pesticides, even though no longer used, persist and remain in the environment. Residues of these pesticides are sometimes found on food grown on contaminated soil, or in the fish that live in contaminated waters.

Pesticides can enter the human body through inhalation of aerosols, dust and vapor that contain pesticides; through oral exposure by consuming food and water; and through dermal exposure by direct contact of pesticides with the skin. Roughly 90 percent of pesticide intake is ingested with food; much of the remainder has its source in pesticide-contaminated air and water. Across the globe pesticides have been found in human blood, urine, breast milk, semen, adipose tissue, amniotic fluid, infant meconium and umbilical cord blood.

Many fruits and vegetables test positive for pesticide residues, with about one-third of them showing up with multiple residues. Fish are an important part of a healthy diet. Some fish caught in lakes, rivers, oceans and estuaries, however, may contain pesticide residues that could pose health risks if these fish are eaten in large amounts. Eating fish containing pesticide residues may cause birth defects, liver damage, cancer, and other serious health problems.

Researchers reveals that prolonged exposure to pesticide residues may increase the risk of various cancers and neurological problems (such as Parkinson's disease), and impair the immune system. Studies have proved that farmers are at a potentially high risk of developing leukemia, lymphomas, and cancers of organs like the prostate, stomach, skin and brain. Pesticides are linked to chronic health disorders and ailments. Exposure to

pesticides can range from mild skin irritation to birth defects, cancers, blood and nerve disorders, hormone disruption, and even coma or death.

In a developed country like the United States of America, a US delegation consisting of US Department of Agriculture (USDA), US Environmental Protection Agency (EPA) and Food and Drug Administration (FDA) representatives actively participates in setting international limits that are similar to tolerance levels for pesticide residues in food that is traded globally. EPA and FDA reviews the recent findings on pesticide residues in foods, and state and regional intelligence reports on how much pesticide is being used on crops or animals in a region. Similarly, FDA obtains information on pesticide usage from other countries that export foods to the US. The information helps FDA plan on the types of foods to monitor and how many samples to analyze. It prioritizes foods to be tested according to how much of it is typically eaten. The food is also monitored for pesticides that are no longer used in the country but are long lasting in the environment, such as the insecticides DDT, chlordane, dieldrin and toxaphene. FDA has used methods that monitor food samples for different pesticides and break down products. A second type of monitoring is called “Incidence or Level Monitoring”. Under this program, FDA tries to determine how often a certain pesticide is found on a particular crop.

FDA conducts a third kind of food monitoring called the “Total Diet Studies” to analyze pesticide residues that remain in a typical meal. Some examples are: chocolate milk, boiled eggs, chicken nuggets, pork and beans, bread, banana, french fries, macaroni and cheese, ice cream, popcorn, honey, butter, lemonade, and infant and children’s foods.

11.2. Maximum Residue Limits (MRLs)

A Maximum Residue Limits (MRLs) for pesticides are established in most countries to safeguard consumer health and to promote Good Agricultural Practice (GAP) in the use of insecticides, fungicides, herbicides and other agricultural compounds.

Maximum residue limit or MRL is the maximum amount of residue legally permitted on food. Once residues are demonstrated to be safe for consumers, MRLs are set by independent scientists, based on rigorous evaluation of each pesticide legally authorized. They act as an indicator of the

correct use of pesticides, and ensure compliance with legal requirements for low residues on unprocessed food. MRLs are trading standards used to ensure that imported and exported food is safe to eat. In practice, they allow the free movement of goods within the EU and from the rest of the world.

These MRLs vary from country to country depending on the pesticides available, the crops being treated and the way the pesticides are used. Food exporters must comply with these MRLs as a condition of market access.

11.3. The United Nations Codex Alimentarius Commission

The United Nations Codex Alimentarius Commission has recommended international standards for Maximum Residue Limits (MRLs), for individual pesticides in food. Since 1962, the Codex Alimentarius Commission (CAC) has been responsible for implementing the Joint FAO/WHO Food Standards Programme. The Commission's primary objectives are the protection of the health of consumers, the assurance of fair practices in food trade and the coordination of the work on food standards.

The CAC is an intergovernmental body with a membership of 165 Member governments. In addition, observers from international scientific organizations, food industry, food trade and consumer associations may attend sessions of the Commission and of its subsidiary bodies. An Executive Committee, six Regional Coordinating Committees and a Secretariat assist the Commission in administering its work programme and other activities.

The work of the Codex Alimentarius is divided between two basic types of committees: Nine general subject matter(s) Committees that deal with general principles, hygiene, veterinary drugs, pesticides, food additives, labelling, methods of analysis, nutrition and import/export inspection and certification systems and Twelve Commodity Committees which deal with a specific type of food class or group, such as dairy and dairy products, fats and oils, or fish and fish products.

The work of the Committees on hygiene, fish and fishery products, veterinary drugs and import/export inspection and certification systems is of paramount interest to the safety and quality of internationally traded fish and fishery products. The specific Codex food safety provision includes the maximum residue limits for pesticides and veterinary drugs, the maximum

level of use of food additives, the maximum levels of contaminants, and food hygiene requirements of Codex standards.

11.4. Safe Level of a Pesticide Residue

In the United States of America, Environmental Protection Agency (EPA) evaluates tests done in experimental animals, and on plant, human or animal cells growing in the laboratory to estimate the health risk to humans from exposure to pesticides. EPA determines how much the pesticide is likely to remain in foods that are grown using the recommended guideline for pesticide use. It pays extra attention to foods that are eaten by children in large quantities, such as apple juice and milk.

To estimate the health risk to humans from exposure to pesticides, EPA determines how much of the pesticide is likely to remain in foods. A computer program is specially developed to estimate health risks, called the "Dietary Exposure Evaluation Model." EPA considers the exposure through food, drinking water, and home use of pesticides. EPA will set a tolerance level for food if the combined exposure from different sources is 100 to 1,000 times lower than the maximum residue limit (MRL) that shows no harmful effects in experimental animals.

11.5. Monitoring the Residue Levels of Pesticides in Food

In a developed country like the United States of America, Food and Drug Administration (FDA) monitors the levels of pesticides in raw agricultural produce, fish, dairy products and processed foods. Then Food Safety and Inspection Service (FSIS) of US Department of Agriculture (USDA) is responsible for monitoring pesticide residues in meat and certain egg and poultry products.

Both the FDA and the USDA work with state agencies to collect and test for pesticide residues in food from different parts of the country. For imported foods, food samples are collected at the port of entry. If a food is found to have any pesticide residue at, or greater than the tolerance level, federal or state officials can remove the food and destroy it. Any pesticide residue that exceeds tolerance levels, or does not meet EPA regulations is reported as "violative".

11.6. Maximum Residue Limits (MRLs) of some Agricultural Chemicals and Environmental Chemical Contaminants on Fish, Mollusk, Crustaceans and Food

Maximum Residue Limits (MRL) of some agricultural chemicals and environmental chemical contaminants on fish, mollusk, crustaceans and food are shown in Table 11.1 to Table 11.4.

**Table 11.1. Maximum Residue Limits (MRLs)(ppm) of some Agricultural Chemicals on fish in Japan.
(Pesticide and food safety in Japan, 2009)**

No	Agricultural Chemicals	Perciformes*	Salmoniformes**	Anguilliformes***
1	Aldrin	0.1	0.1	0.1
2	Cypermethrin	0.01	0.03	0.01
3	DDT	3.0	3.0	3.0
4	Endosulfan	0.004	0.004	0.004
5	Endrin	0.005	0.005	0.005
6	Heptachlor	0.05	0.05	0.05
7	Lindane	1.0	1.0	1.0
8	Malathion	0.5	0.5	0.5

***Perciformes** - bonito, horse mackerel, sea bass, sea bream and tuna

** **Salmoniformes** –salmon and trout, *****Anguilliformes** –eel

Table 11.2. Environmental chemical contaminants. Maximum Residue Limits (MRLs) in fish, mollusk and crustaceans. (US. Food and Drug Administration, 1998)

No	Chemicals	US (ppm)	EU (mg/kg)	Food commodity
1	Arsenic	76-86	-	Mollusks, crustaceans
2	Cadmium	3-4	0.05-1.0	Fish, mollusks
3	Lead	1.5-1.7	0.2-1.0	Fish, mollusks
4	Methyl mercury	1.0	1.0	all fish
5	PCB	2.0	-	all fish
6	DDT	5.0	-	all fish
7	Dieldrin	0.0	-	all fish
8	Dioxin	-	0.000004	all fish

Table 11.3. Some Maximum Residue Limits (MRLs) in mg/kg on onions registered for use in Australia (DAFF, Australian Government, 2010)

No	Pesticide	Australia	EU	Japan	Malaysia	Singapore
1	Diazinon	0.70	0.05	0.05	Not set	0.50
2	Methyl Parathion	Not set	0.02	1.00	Not set	Not set
3	Aldrin	0.1	0.05	1.00	Not set	Not set
4	DDT	1.0	0.05	0.50	Not set	1.00
5	Endosulfan	0.2	0.05	0.20	Not set	0.20
6	Endrin	Not set	0.01	0.01	Not set	Not set
7	HCH	Not set	0.01	Not set	Not set	Not set
8	Heptachlor	0.05	0.01	0.03	Not set	0.05
9	Lindane	2.0	0.01	2.0	Not set	Not set
10	Cypermethrin	0.01	0.10	0.10	Not set	0.10
11	Pyrethrins	1.0	1.0	1.0	Not set	1.0

Table 11.4. Analysis of wild caught fish for trace elements and persistent organic pollutants. (Food Standards, Australia New Zealand, 2005)

No	Fish	Residue	Average concentration	MRLs
1	Eel	Dioxins Cadmium Copper Lead Mercury DDT PCB	0.00000030 0.005 0.343 0.007 0.211 0.015 0.033	No limit No limit No limit 0.5 0.5 1.0 0.5
2	Lobster	Cadmium Copper Lead Mercury	0.010 2.821 0.005 0.048	No limit No limit No limit 0.5
3	Mackerel	Dioxins Cadmium Copper Lead Mercury	0.00000031 0.023 0.685 0.006 0.072	No limit No limit No limit 0.5 0.5
4	Prawn	Cadmium Copper Lead Mercury	0.02 3.634 0.009 0.040	No limit No limit No limit 0.5
5	Trout	Dioxins Cadmium Copper Lead Mercury	0.00000025 0.006 2.230 0.009 0.126	No limit No limit No limit 0.5 0.5
6	Tuna	Dioxins Cadmium Copper Lead Mercury	0.00000044 0.007 1.515 0.012 0.343	No limit No limit No limit 0.5 1.0

11.7. Analysis of Environmental Chemical Residues in Products of Emerging Aquaculture Industry in Uganda (December 2008)

A study was conducted to analyse market-regulated heavy metals (lead, mercury and cadmium) and organochlorine pesticides in samples of 38 farmed fish comprising Nile tilapia (*Oreochromis niloticus*) (20 samples) and African catfish (*Clarias gariepinus*) (18 samples) from ten selected fish farms in Uganda.

- Lead was detected in all the 38 samples (maximum = 1.08 mg kg⁻¹ (dry weight)),
- Mercury in 31 out of 38 samples (maximum= 0.35mg kg⁻¹ (dry weight)),
- Cadmium in two samples (maximum = 0.03 mg kg⁻¹ (dry weight)).
- Pesticides detected were: 4,4'-dichloro-diphenyl-trichloroethane (DDT) and endosulfan sulphate, which were found in one fish sample (both 0.002 mg kg⁻¹ (wet weight)).

The levels of contaminants were below the US Food and Drug Administration (USFDA) action levels and European Union maximum residue limits (MRLs), indicating that such fish have the potential for export to Markets (Bagumire, 2008).

11.8. Trade problems arising from differing maximum residue levels for veterinary drug and pesticide residues

An international effort is underway to harmonize the procedures and assumptions used for establishing maximum residue levels (MRLs) for residues of veterinary drugs and pesticides. Apparent conflicts in MRLs may not actually reflect differing safety assessments for the residues but may be due to different safety factors, methods of analysis, consumption factors, etc. Equivalence is an important principle in the U.S. position on agricultural trade.

Countries wishing to export food products to the U.S. must demonstrate that their country's residue control programs are equivalent to the U.S. program. From the U.S. export perspective, there have been several

instances of trade problems resulting from different MRLs. The most significant was the decision by the European Economic Community not to accept U.S. beef if the animals were administered anabolic steroids. Another example involved the Japanese rejection of U.S. pork because of sulfamethazine residues. These examples illustrate the importance of harmonization to future efforts to facilitate free trade and reduce the resource burden on government regulatory bodies.

11.9. Conclusion

Government of the Republic of the Union of Myanmar have expressed concerned about the toxic effects of pesticides on human and wildlife and have initiated regulatory policies to safeguard against these adverse effects of pesticides use. **Pesticide Law was enacted in 1990. Formation of Pesticide Registration Board was issued by the Government of the Union of Myanmar on 25th February 1992 by notification No 2/92.** Under Pesticide Registration Board the technical Committee, with technical personnel from ministries concerned, has been set up for evaluation of pesticide efficacy, quality, pesticide residue, toxicology, occupational and public health aspects and to recommend for registration to Pesticide Registration Board. According to the Law and Procedures, all pesticides to be marketed in the country are required to be registered providing documented data of pesticide identity, quality, efficacy, toxicity, handling and disposal prior to domestic use.

Myanmar has established a Pesticide Analytical Laboratory (PAL) with the technical and financial assistance of FAO. PAL is capable of carrying out product and residue analysis. Then pesticides are classified for general and restricted use. Some may be banned or suspended.

Myanmar is participating in PIC procedure implemented by the United Nations Environmental Program/Food and Agriculture Organization (UNEP/FAO) and the Director General of the Department of Agricultural Planning, Ministry of Agriculture and Irrigation is acting as designated national authority for pesticide import. The country is receiving the Decision Guidance Document (DGDs) from UNEP/FAO and responding to the decision of import accordingly. Taking into account of the criteria in DGDs

and the country's experience, pesticides of highly toxic to fish, wildlife and humans are banned from use in Myanmar (Table 11.5).

**Table 11.5. Banned pesticides in Myanmar
(Pesticide Registration Board, 2010)***

No	Pesticide	Used Category
1	Aldrin	Insecticide
2	BHC	Insecticide
3	Captafol	Fungicide
4	Chlordane	Insecticide
5	Chlordimeform	Insecticide
6	Cyhexatin	Insecticide
7	Dieldrin	Insecticide
8	Dinoseb	Herbicide
9	EDB	Fumigant
10	Endrin	Insecticide
11	EPN	Insecticide
12	Inorganic mercury compounds	Fungicide
13	Organic mercury compound	Fungicide
14	Parathion Ethyl	Insecticide
15	Strobane	Insecticide
16	2,4,5-T	Herbicide
17	Toxaphene	Insecticide
18	Monocrotophos	Insecticide

* **Personal communication**

In order to prevent an increasing number of pesticide-related human deaths, Myanmar Pesticide Registration Board is insisting that all pesticide applicators have proper training and certification.

The contamination of food from the indiscriminate use of pesticides is a common problem. The residues in crops at harvest must be maintained below the maximum residue limits. The government officials of Myanmar have expressed concerns over residue problem in both domestic and export markets.

The residues in crops at harvest must be maintained below the **Maximum Residue Limits** (MRLs) set by the Government of the Republic of the Union of Myanmar and international organization. Acceptable residue levels are based on an estimate of level of pesticide residue intake below which the risk to health is too small to be a concern. This level is known as the “Acceptable Daily Intake” (ADI) and is defined as the amount of residue that may be consumed every day during an individual’s active lifetime that no harm will result. The FAO/WHO Codex Alimentarius Commission sets international standards of MRLs and ADIs for agricultural crops intended for international trade. The Pesticide Registration Board of the Government of the Republic of the Union of Myanmar adopts these standards to ensure that safety levels of pesticide residues in food are not violated.

12. Effects of Mosquito Coil Smoke Inhalation on Human Health

12.1. Introduction

A mosquito coil is a mosquito repelling incense, usually shaped into a spiral, and typically made from a dried paste of pyrethrum powder. The coil is usually held at the center of the spiral, suspending it in the air, or wedged by two pieces of fireproof nettings to allow continuous smoldering. Burning usually begins at the outer end of the spiral and progresses slowly toward the centre of the spiral, producing a mosquito-repellent smoke. A typical mosquito coil can measure around 15 cm in diameter and lasts around 8 hours. Mosquito coils are frequently burned indoors in Asia, Africa, Australia and to a limited extent in other parts of the world, including the United States (WHO, 2005).

In 1996, the WHO report estimated the worldwide annual consumption of mosquito coils to be approximately 29 billion pieces. In a study in Taiwanese households, the prevalence of burning mosquito coils is 45% (WHO, 1998). The consumers usually use mosquito coils for at least several months every year, and cumulative effects from long-term exposure to the coil smoke may also be a concern. Despite the fact that mosquito coil smoke may have many potential adverse health effects, large populations in developing countries still use mosquito coils in their daily lives.

Although mosquito coils are recommended for outdoor use, or for use in semi-enclosed patios and porches, coils are often used overnight in sleeping quarters. As a result peoples are exposed to a chemically complex mosquito-coil smoke containing small particles ($< 1 \mu\text{m}$), metal fumes, and vapors that may reach the alveolar region of the lung.

Mosquito coils can be hazardous. In 1999, sparks from mosquito coils ignited a fire that swept through a three-story dormitory building at a summer camp in South Korea; 23 people, including 19 children, died in the blaze. The smoke generated from a burning mosquito coil is of certain health concerns – one burning mosquito coil produces the same amount of particulate mass (diameter up to $2.5 \mu\text{m}$) as 75-137 burning cigarettes would; and the emission

of formaldehyde from one burning coil can be as high as that released from 51 burning cigarettes.

12.2. Mosquito Coil Ingredients

Active ingredients found in mosquito coils may include:

- Pyrethrum - a natural, powdered material from a kind of chrysanthemum plant
- Pyrethrins - an extract of the insecticidal chemicals in pyrethrum
- Allethrin - sometimes d-trans-allethrin, the first synthetic pyrethroid
- Esbiothrin - a form of allethrin
- Butylated hydroxytoluene (BHT) - an optional additive to prevent pyrethroid from oxidizing during burning
- Piperonyl butoxide (PBO) - an optional additive to improve the effectiveness of pyrethroid
- *N*-Octyl bicycloheptene dicarboximide (MGK 264) - an optional additive to improve the effectiveness of a pyrethroid

The most common active ingredients in coils are various pyrethroids, such as allethrin, d-allethrin, pynamin forte and ETOC. Octachlorodipropylether (S-2) is sometimes used as a synergist or active ingredient and use of such coils exposes humans to some level of bis-chloromethyl ether (BCME) which is an extremely potent lung carcinogen.

Although the U.S. Environmental Protection Agency (U.S. EPA) does not register S-2 for any use, some imported mosquito coils contain this chemical, but their use is illegal in the United States, moreover in places like India S-2 is not banned. Other compounds, released during the burning of mosquito coils (aldehydes, formaldehydes, fine and ultrafine particles, benzene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene are also classified by the U.S. EPA as probable human carcinogens.

12.3. Side Effects of using Mosquito Coil

Burning mosquito coils indoors generates smoke that can control mosquitoes effectively. The smoke may contain pollutants of health concern. Breathing in too much smoke will increase the risk of asthma and cause persistent wheezing in children. These are formaldehyde, octachlorodipropyl ether and bischloromethyl ether. The active ingredient in mosquito coils is Pyrethroid insecticides. Pyrethroids are mostly harmless to humans, but they can irritate the skin and eyes. Some people are allergic to them too. According to experts, mosquito repellent mats emit fumes that contain a substance called Allethrin and its products that may cause health hazards like cancer and complications in pregnant women. When inhaled, it may worsen asthma in individuals who suffer from the disease. In others, it may cause nausea, vomiting, diarrhoea and coordination difficulties. It is especially dangerous for infants, young children and pregnant women.

Formaldehyde is a colourless, flammable and strong smelling gas. Inhaling it could cause watery eyes, throat discomfort, coughing, wheezing, nausea and skin irritation. Also, it can cause nasal or sinus cancer and even leukaemia. The amount of formaldehyde released from burning one mosquito coil can be as high as smoking 51 cigarettes. Formaldehyde is not an ingredient of mosquito coils but a by-product of burning them.

Octachlorodipropyl ether, better known as S-2, is a pesticide banned by the US Environmental Protection Agency. When S-2 is burned, it releases bischloromethyl ether, a strong and harmful chemical that causes lung cancer. According to a study by UC Riverside scientists, many mosquito coils – most notably those manufactured in Asia – often contain up to one percent BCME (which stands for bis[chloromethyl]ether, a chemical associated with the breakdown of S-2). BCME has been described as “the most potent lung cancer chemical ever discovered.” And lung cancer is just about the most deadly cancer known. In one Chinese factory where mosquito coils were manufactured, a large fraction of employees were dead within five years of starting their jobs. The cause? Lung cancer.

Researchers from the University of California at Riverside recently tested more than 50 mosquito repellent coils purchased in Indonesia and at several Asian markets in California, and found they contained varying levels of a pesticide, S-2, that releases cancer-causing particles when burned. Of

more concern was that the researchers found S-2 in samples of mosquito coils purchased in California. S-2 shouldn't be in any products sold in the United States because it's banned by the U.S. Environmental Protection Agency either as a main or secondary ingredient, according to the study. Not surprisingly, the coil labels didn't disclose the presence of S-2.

12.4. Exposure to Mosquito Coil Smoke may be a Risk Factor for Lung Cancer in Taiwan (Chen, 2008)

About 50% of lung cancer deaths in Taiwan in 2008 were not related to cigarette smoking. Environmental exposure may play a role in lung cancer risk. Taiwanese households frequently burn mosquito coil at home to repel mosquitoes. The hospital-based case-control study was done to determine whether exposure to mosquito coil smoke is a risk for lung cancer.

Questionnaires were administered to 147 primary lung cancer patients and 400 potential controls to ascertain demographic data, occupation, lifestyle data, indoor environmental exposures (including habits of cigarette smoking, cooking methods, incense burning at home, and exposure to mosquito coil smoke), as well as family history of cancer and detailed medical history.

Mosquito coil smoke exposure was more frequent in lung cancer patients than controls (38.1% vs.17.8%; $p < 0.01$). Risk of lung cancer was significantly higher in frequent burners of mosquito coils (more than 3 times [days] per week) than nonburners (adjusted odds ratio = 3.78; 95% confidence interval: 1.55-6.90). Those who seldom burned mosquito coils (less than 3 times per week) also had a significantly higher risk of lung cancer (adjusted odds ratio = 2.67; 95% confidence interval: 1.60-4.50). Exposure to mosquito coil smoke may be a risk factor for development of lung cancer.

12.5. Mosquito Coil Exposure associated with Small Cell Lung Cancer: A Report of Three Cases (Jie Zhang, 2015)

Mosquito coils, which are commonly used as residential insecticides in Asia, contain different concentrations of octachlorodipropyl ether (S-2) as a synergist or an active ingredient. As bis(chloromethyl) ether (BCME) is an extremely potent lung carcinogen that can be produced by the thermolytic degradation of S-2, contact with mosquito coils is likely to expose individuals

to a certain level of BCME, and therefore increase the risk of lung cancer. However, the significance of exposure is uncertain, as clinical and epidemiological studies concerning mosquito coil users and workers are lacking.

This study described three cases of small cell lung cancer treated at the Shanghai Pulmonary Hospital that were likely to be the result of exposure to mosquito coils. All patients had worked in the mosquito coil manufacturing industry, with a mean occupational duration of 9.1 years, and presented with similar respiratory symptoms, such as cough and dyspnea. Upon diagnosis, no metastasis to other organs was identified in any of the cases. Subsequently, the three patients were treated with chemotherapy as well as radiotherapy in one case; however, all patients succumbed to the disease, with a mean overall survival time of 10.7 months. Contact with mosquito coils is likely to expose individuals to a level of S-2 that may increase the risk of SCLC (small cell lung cancer).

Case one

A 39-year-old male non-smoker presented to the Shanghai Pulmonary Hospital (Tongji University, School of Medicine, Shanghai, China) on March 6, 2008, with a productive cough that had been apparent for one month. Radiography (Fig. 12.1A) and computed tomography (CT) of the chest (Fig. 12.1B) revealed enlarged lymph nodes and a mass measuring 4.8×3.4 cm in the upper lobe of the left lung. Immunohistochemical analysis indicated that the tumor was positive for thyroid transcription factor 1 (TTF-1) and synaptophysin (SYN), but negative for cluster of differentiation (CD)5 and 6 (Figure 12.4). The patient was subsequently diagnosed with SCLC, tumor-node-metastasis (TNM) stage T4N2M0 (IIIb). Following two cycles of chemotherapy with 100 mg/m² etoposide and 75 mg/m² cisplatin on days one to three of three-weekly cycles, the patient exhibited a complete response (CR) (Fig. 12.1C). The six cycles of chemotherapy were completed on September 13, 2008. In March 2009, CT revealed the presence of progressive disease (PD) (Fig. 12.1D) and second-line chemotherapy with 60 mg/m² irinotecan on days one and eight of three-weekly cycles, was subsequently initiated. Due to a poor performance status, the patient proceeded to receive supportive care, but succumbed to the disease on August 17, 2009.

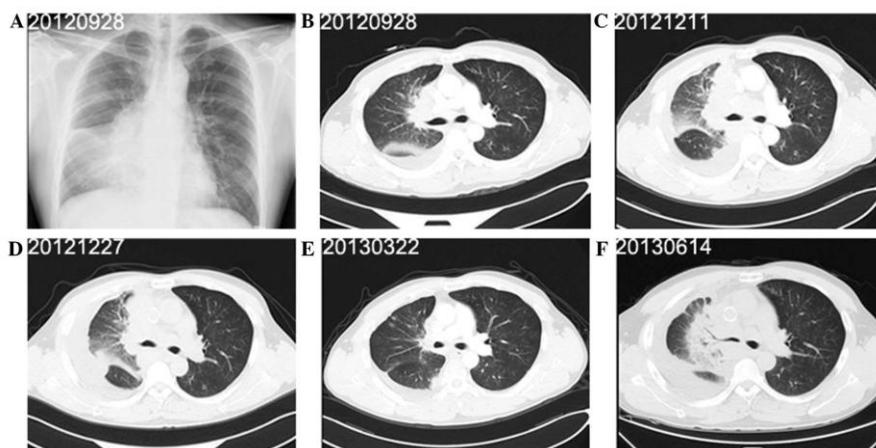


Figure 12.1. Case one: Representative images from radiography and chest CT revealing the presence of a mass in the upper lobe of the left lung. (A) Radiograph prior to treatment. (B) Representative CT prior to treatment. (C) Representative CT image after two cycles of first-line chemotherapy. (D) Representative CT image showing progressive disease after six cycles of first-line chemotherapy. (E) Representative CT image showing progressive disease after six cycles of first-line chemotherapy. (F) Representative CT image showing progressive disease after six cycles of first-line chemotherapy. CT, computed tomography. (Jie Zhang, 2015)

Case Two

A 41-year-old male presented to the Shanghai Pulmonary Hospital on October 20, 2010, with a productive cough and dyspnea. The patient had smoked 10 cigarettes per day for the past 20 years. Radiography (Fig. 12.2A and 12.2C) and CT (Fig. 12.2B) of the chest revealed enlarged lymph nodes and a mass measuring 10.5×7.2 cm in the upper lobe of the left lung. Immunohistochemical analysis indicated that the tumor was positive for TTF-1 and SYN, but negative for CD5/6 (Figure 12.4). The patient was subsequently diagnosed with SCLC, stage T4N2M0 (IIIb). Following two cycles of chemotherapy with 100 mg/m² etoposide and 25 mg/m² cisplatin on days one to three of three-weekly cycles, the patient's condition deteriorated, with evidence of hemoptysis and thrombocytopenia. The patient succumbed to the disease on January 25, 2011.

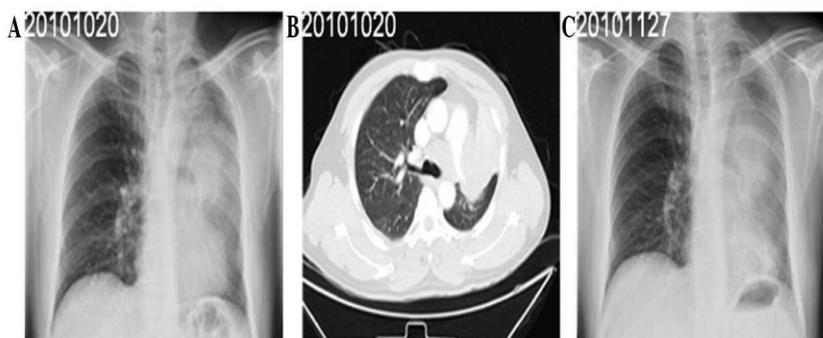


Figure 12.2 - Case two: Representative images from radiography and chest CT revealing the presence of a mass in the upper lobe of the left lung and enlarged lymph nodes. (A) Radiograph prior to treatment. (B) Representative CT image prior to treatment. (C) Radiograph after one cycle of first-line chemotherapy treatment. CT, computed tomography. (Jie Zhang, 2015)

Case Three

A 40-year-old male presented to the Shanghai Pulmonary Hospital on September 27, 2012, with right-sided chest pain, a productive cough and dyspnoea that had been apparent for two weeks. The patient had smoked 15 cigarettes per day for the past 18 years. Radiography (Fig. 12.3A) and CT of the chest (Fig. 12.3B) revealed pleural effusion, enlarged lymph nodes and a mass measuring 9.4×8.0 cm in the middle lobe of the right lung. Immunohistochemical analysis indicated that the tumor was positive for TTF-1, NSE, chromogranin A and Ki-67, but negative for SYN, leukocyte common antigen, p63 and CD5/6 (Figure 12.4). The patient was subsequently diagnosed with SCLC, stage T4N3M1a (IV). Following two cycles of chemotherapy with 100 mg/m² etoposide and 25 mg/m² cisplatin on days one to three of three-weekly cycles, the tumor response was assessed as PD (Figure 12.3C). Superior vena cava stenting and 25 Gy thoracic radiation therapy (2.5 Gy/fraction) were performed in December, 2012 for two weeks. Four cycles of second-line chemotherapy with 60 mg/m² on days one and eight of three-weekly cycles were also administered (Fig. 12.3E). On June 14, 2013, the tumor response was evaluated as PD (Fig. 12.3F), at which time the patient's performance status deteriorated. The patient succumbed to the disease on July 02, 2013.

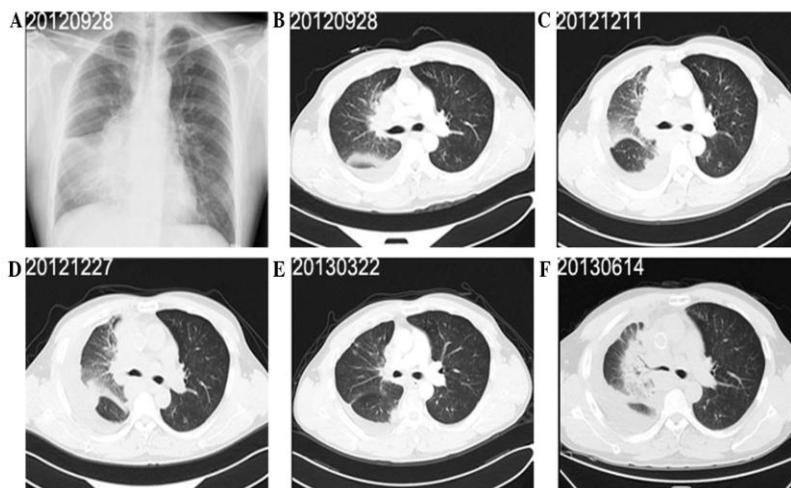


Figure 12.3 - Case three: Representative images from radiography and chest CT revealing the presence of a mass in the middle lobe of the right lung, pleural effusion and enlarged lymph nodes. (A) Radiograph prior to treatment. (B) Representative CT image prior to treatment. (C) Representative CT image after two cycles of first-line chemotherapy. (D) Representative CT image after superior vena cava stenting. (E) Representative CT image after thoracic radiation therapy and four cycles of second-line chemotherapy. (F) Representative CT image showing progressive disease after second-line chemotherapy. CT, computed tomography. (Jie Zhang, 2015)

Based upon the histological evidence, it was hypothesized that the inhalation of S-2 may have been the potential cause of SCLC in the three patients included in the present study. However, the other toxic products released following mosquito coil use have yet to be adequately assessed, therefore, future controlled studies should be conducted in order to evaluate their effects. Exposure is a controllable factor, and workers therefore deserve preventive actions in order to reduce exposure to toxins in the workplace. Furthermore, the effects of daily use and exposure to mosquito coils should be evaluated with respect to further health implications.

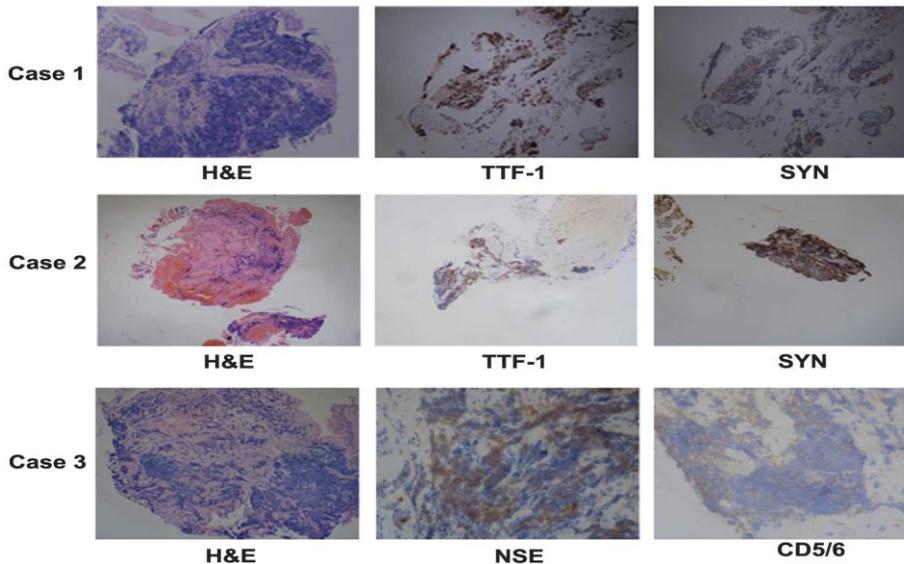


Figure 12.4 - Histological analysis of endobronchial biopsy specimens from cases one, two and three (magnification, $\times 100$). H&E, hematoxylin and eosin; TTF-1, thyroid transcription factor 1; SYN, synaptophysin; NSE, neuron-specific enolase; CD, cluster of differentiation. (Jie Zhang, 2015)

12.6. Toxicological Effects of Inhaled Mosquito Coil Smoke on the Rat Spleen: A Haematological and Histological Study (Garba, 2007)

Study on the effect of inhaling mosquito coil smoke on the haematology and histology of the rats spleen was carried out in the Departments of Human Anatomy and Human Physiology, University of Maiduguri, Nigeria between March and October, 2005. A total of 30 albino rats of the Wister strain were used in this study, they were divided into six groups of five rats each. Rats in Group I served as control (no exposure to mosquito coil smoke). While Groups II-VI were exposed to mosquito coil smoke for 12 h, 7, 14, 21 and 28 days, respectively. At the end of each experimental period, blood was collected from each rat for the analysis of Red Blood Cell (RBC) count, White Blood Cell (WBC) count, Haemoglobin (Hb) concentration, Packed Cell Volume (PCV) and the percentages of Neutrophils, Monocytes, Eosinophils, Basophils and Lymphocytes. The rats were then sacrificed and the spleen obtained, was processed for routine histological analysis. Haematological analysis of the blood obtained revealed a significant ($p < 0.01, 0.05$) increase in WBC count in all exposure periods,

while analysis of differential leucocyte count revealed a significant ($p < 0.05$) increase in basophil and lymphocyte percentages. Histological analysis of the spleen tissue revealed severe congestion of venous sinusoids, hyperplasia and regression of both the red and white pulps. Results from this study demonstrates that mosquito coil smoke inhalation challenges the immune system in experimental rats; however, the precise mechanisms remain to be clarified in more detailed studies.

12.7. Toxic Effects of Mosquito Coil Smoke on Rats: II. Morphological Changes of the Respiratory System (Liu, 1988)

Study of the toxic effect of mosquito coil smoke on the morphological changes of the respiratory system of rats was carried out in the Department of Anatomy, the Chinese University of Hong Kong in 1987. A group of 20 female albino rats was exposed to mosquito coil smoke, 8 h a day, 6 days per week, for 60 days. An additional group receiving air exposure served as control. The smoke-exposed animals had a lower body weight than the controls. Smoke-induced histopathological lesions, including an inflammation of the tracheal epithelium, atelectasis of the lung parenchyma, emphysema, increase of alveolar macrophages in the alveolar space and perivascular infiltration of polymorphonuclear cells were observed in the experimental rats. An elevation of enzyme activities of lactate dehydrogenase, glutamate pyruvate transaminase, glutamate oxoacetate transaminase and acid phosphatase were found in the serum of the smoke-exposed rats indicating the enzymes were released from the damaged tissues into the blood stream.

12.8. Mosquito Coil Smoke Inhalation Effects on Interstitium of Kidney of Albino Rats (Nazia Siddique, 2012)

The study was done in animal house of PGMI Lahore in 2012. Albino rats were provided by Punjab University, Lahore. First step was acclimatization of rats, for which rats were kept in cages for 15 days. Average weight of rats was between 180-200gms. After acclimatization, the animals were randomly divided into three groups A, B and C having 8 rats in each. Group A served as a control so was not exposed to mosquito coil smoke while

Group B and C were exposed to mosquito coil smoke for 8 hours/day. Group B were exposed to mosquito coil smoke for two weeks and group C for four weeks. Mosquito coil smoke was given as whole body inhalation.

Renal interstitium is the space present external to basal laminae of the kidney tubules. The renal interstitium consists of collagen fiber, macrophages and fibroblast. In kidney stroma cellular infiltrate, haemorrhage, congestion of vessels and fibrosis were observed in group B and C which was statistically significant when three groups were compared (Figs. 12.5, 12.6, 12.7 and 12.8) but absent in group A.

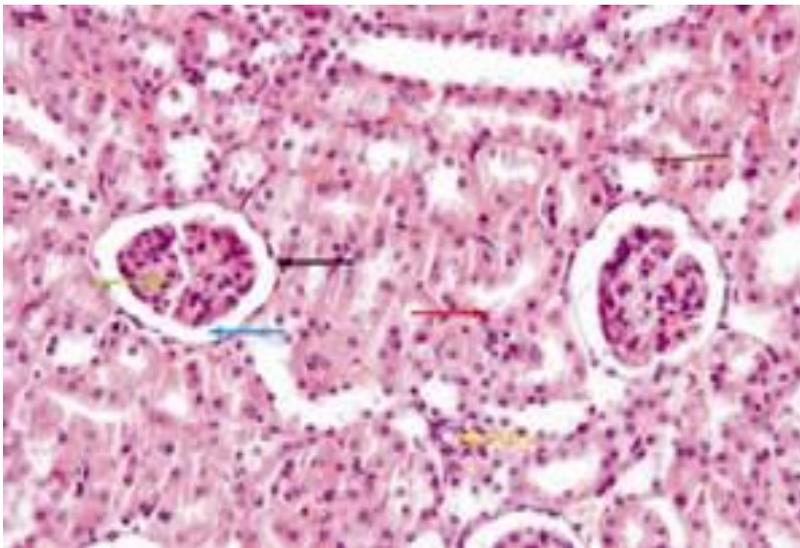


Fig.12.5. Photomicrograph of the kidney from the control group (A) showing renal corpuscles lined by parietal squamous epithelium (black arrow) with central normal glomerulus (green arrow) surrounded by bowman's space (blue arrow). PCT lined by cuboidal epithelium with central nuclei (red arrow) with brush border. DCT lined by cuboidal epithelium (brown arrow). Normal interstitium with blood vessels also seen (yellow arrow). H&E stain X.200 (Nazia Siddique, 2012)

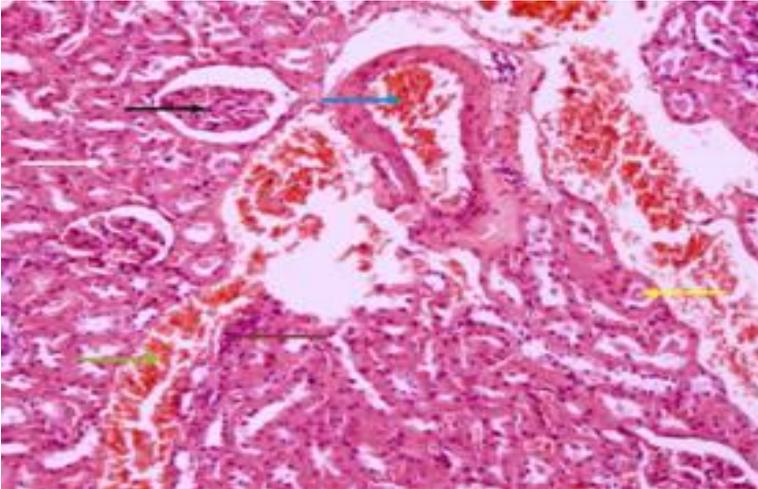


Fig.12.6. Photomicrograph of cortex of kidney from group (B) showing degenerated glomerulus (black arrow).There is necrosis of tubules (white arrow). Cellular infiltrates are seen (brown). In lumen of DCT protein cast is seen (yellow arrow). Interstitial hemorrhage (green arrow) and congested blood vessel (blue arrow) is seen. H&E stain X.100 (Nazia Siddique, 2012)

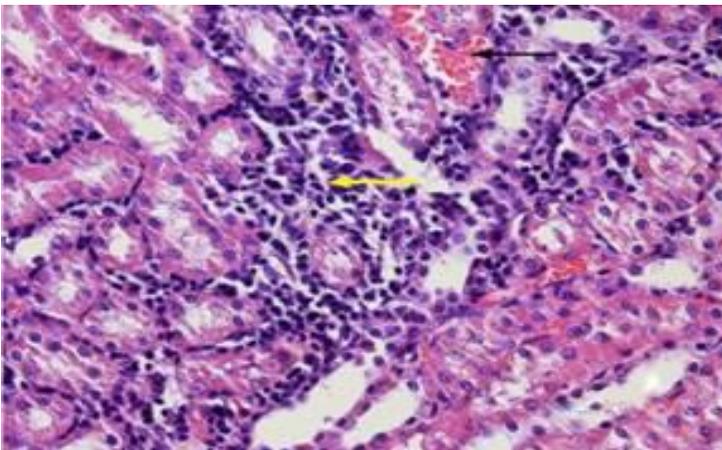


Fig.12.7. Photomicrograph of the kidney from group C showing hemorrhage (black arrow) and cellular infiltrate (yellow arrow) in interstitium of kidney. H&E stain X.400 (Nazia Siddique, 2012)

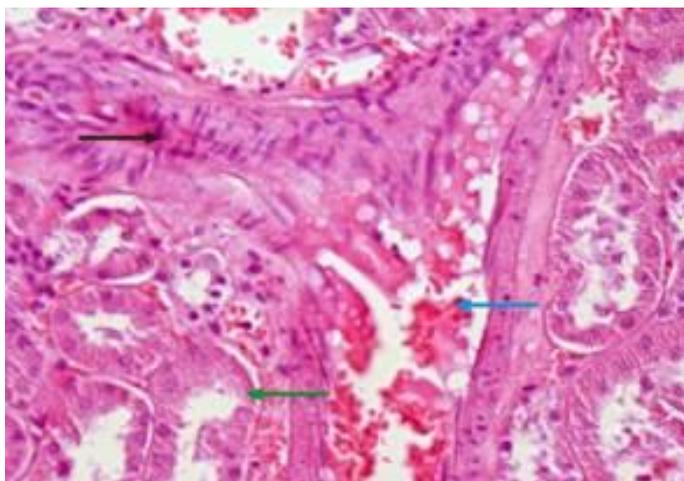


Fig.12.8. Photomicrograph of kidney from group (C) showing renal fibrosis (black arrow), congested blood vessel (blue arrow) and tubular necrosis (green arrow). H&E stain X.400. (Nazia Siddique, 2012)

The results of this study suggest that despite of being the least toxic pesticides, pyrethroids still have harmful effects, as exposure to pyrethroids can cause renal tissue damage . It is hoped that this study will produce an awareness and restricted use of pyrethroids insecticides especially at living places.

12.9. Conclusion

Some recent studies of mosquito coils in Asia and the United States have found an unlisted ingredient in unregistered mosquito coils available for sale from China. This chemical is called Otachlorodipropyl Ether or S-2, this is a synergist used in mosquito coils to increase the effectiveness of the active ingredient. While the active ingredient in mosquito coils, usually one type of Allethrin or another, is safe to humans, the S-2 synergist is highly dangerous to humans. When burnt, the smouldering mosquito coil S-2 degenerates into BCME, Bis (chloromethyl) Ether. BCME is an extremely potent lung carcinogen; some believe it to be the most potent carcinogen known. That is not to say all mosquito coils have this ingredient in them, most don't. If you do choose to use mosquito coils make sure they are approved for use in your country. In Australia, always check for an Australian Pesticide and Veterinary

Medicine Authority (APVMA) approval number, this should be on the front of the box. This ensures the products are safe and approved for use.

In the Philippines, the FDA advised the public to be cautious in buying mosquito coils that may contain hazardous substances. It said unregistered brands of mosquito coils are proliferating in the market. The use of mosquito coils may ward off diseases caused by mosquito bites. But the FDA said unregistered brands may be harmful to the public. Unregistered mosquito coils may not contain any active ingredient (AI) or may not contain the correct strength of the AI. In effect, it will not be able to ward off or kill mosquito that can bite the victim and, thus may transmit the infectious pathogen it carries. It noted that the safety of the active ingredient has not passed the evaluation and approval of the FDA. The active ingredient added in the mosquito coil in high concentration can be hazardous to health when inhaled or accidentally ingested by children. It advised consumers to buy only mosquito coils that are registered with the FDA. The FDA said mosquito coils are designed for use in well-ventilated areas.

In Myanmar, the Food and Drug Administration (FDA) established since 1995, takes care of the safety and quality of Food, Drugs, Medical Devices, Cosmetics and Pesticides in the interests of public safety. Myanmar FDA informed the public that improper use of mosquito coils or mats causes liver damage, cornea (in the eye) damage, Asthma, wheezing, shortness in breath, headache and loss of fertility in both men and women.

In the study conducted at the Taiwanese Institute of Medicine, it was found that a staggering fifty percent of the lung cancer deaths in Taiwan were not related to cigarette smoking, as it was previously thought to be. Instead, the deaths were reported from those households which frequently used mosquito coils. Pyrethrum is the major constituent of a mosquito coil. This chemical stings the mosquitoes in their eyes and makes them blind. But breathing in too much of Pyrethrum can increase the risk of asthma, wheezing and other serious lung disorders. Therefore the public should strictly follow the correct method of using them and avoid excessive use of mosquito coils.

Mosquito coils are to be used in an unenclosed environment. As most users want its maximum effect, they tend to close doors and windows while using this repellent, they forget its dangers. If you can't leave your windows

and doors open, it is advisable to leave the room while the coil burns slowly and then come back and let air fresh in before sleeping in such a room.

The mosquito coils must be lighted half an hour before bed time. It should be done, after closing doors and windows of the room and after switching off fans. After half an hour, all doors and windows should be opened and left open for some time, before closing them and going to bed. Only after that, the room is safe. Use special type of medicated net materials in the market that you can use to cover your windows and also use as mosquito nets at night. Using large mosquito nets is another successful method of preventing mosquito bite in the night. Nets made of cotton wool or any similar material can be used with a fan to avoid excessive heat. Closing down doors and windows early around 5 pm or 5.30 pm will control the problem to a certain extent.

As there are various types of mosquito traps available in the market now. There are many natural plants and leaves that can be used to drive away mosquitoes. There are many natural stuff such as 'Pengiri oil' that you can apply on your body without causing any health problems to avoid mosquito bites. Consume lots of garlic. Mosquitoes can't just stand the stuff. Apply neem oil to your skin. Neem oil is used as a natural insecticide. Neem oil is the best skincare product. Lavender oil is commonly used as a mosquito repellent. The upside of it is the beautiful fragrance it carries. The fragrance is not going to attract mosquitoes.

The smoke from mosquito coil poses both acute and chronic health risks, you should avoid using it, starting from TODAY. Instead, prevent mosquitoes from getting into or breeding around your home, and use natural and safe mosquito eradication methods whenever necessary. Safer alternatives include the use of indoor residual insecticide spraying and/or insecticide treated nets, clearing of bushes and drainage of stagnant bodies of water in your surroundings.

13. Harmful Effects of Genetically modified organisms (GMO's)

13.1. Introduction

Genetically Modified Foods (GM foods) or Genetically modified organisms (GMOs) are defined as organisms (except for human beings) in which the genetic material has been altered in a way that does not occur naturally by mating and/or natural recombination. GMOs have widespread applications as they are used in biological and medical research, production of pharmaceutical drugs, experimental medicine, and agriculture. The use of gene technology in food production has become interesting due to increased needs of food as well as its improved quality. With the application of gene technology to plants and animals, goals can be achieved more quickly than by traditional selection. Consequently, ethical dilemmas are opened concerning the eventual negative effects of production of genetically modified food. It seems that supplementation of nutraceuticals and wild foods as well as wild lifestyle may be protective, whereas western diet and lifestyle may enhance the expression of genes related to chronic diseases.

The prevalence and mortality due to multifactorial polygenic diseases: hypertension, coronary artery disease (CAD), diabetes and cancer vary depending upon genetic susceptibility and environmental precursors because they have identifiable Mendelian subsets. Rapid changes in diet and lifestyle may influence heritability of the variant phenotypes that are dependent on the nutraceutical or functional food supplementation for their expression.

It is possible to recognize the interaction of specific nutraceuticals, with the genetic code possessed by all nucleated cells. There is evidence that South Asians have an increased susceptibility to CAD, diabetes mellitus, central obesity and insulin resistance at younger age, which may be due to interaction of gene and nutraceutical environment. The negative consequences can affect the human health and environment.

13. 2. Effects of Genetically Modified Foods on Human Health

Recombinant DNA technology faces our society with problems unprecedented not only in the history of science, but of life on Earth. Potentially, it could breed new animal and plant diseases.

13.2.1. Cancer

Growth Hormone (GH) is a protein hormone which, when injected into cows stimulates the pituitary gland in a way that produces more milk, thus making milk production more profitable for the large dairy corporations. In 1993, FDA approved Monsanto's genetically-modified rBGH, a genetically-altered growth hormone that could be then injected into dairy cows to enhance this feature, and even though scientists warned that this resulted in an increase of IGF-1 (from 70%-1000%). IGF-1 is a very potent chemical hormone that has been linked to a 2 to 4 times higher risk of human colorectal and breast cancer. Prostate cancer risk is considered equally serious – in the 2.8 to 4 times range. According to Dr. Samuel Epstein of the University of Chicago and Chairman of the Cancer Prevention Coalition, this “induces the malignant transformation of human breast epithelial cells.” Canadian studies confirmed such a suspicion and showed active IGF-1 absorption, thyroid cysts and internal organ damage in rats.

13.2.2. Super viruses

Viruses can mix with genes of other viruses and retroviruses such as HIV. This can give rise to more deadly viruses – and at rates higher than previously thought. One study showed that gene mixing occurred in viruses in just 8 weeks (Kleiner, 1997). This kind of scenario applies to the cauliflower mosaic virus CaMV, the most common virus used in genetic engineering – in Round Up ready soy of Monsanto, Bt-maize of Novartis, and GM cotton and canola. It is a kind of “pararetro virus” or what multiplies by making DNA from RNA. It is somewhat similar to Hepatitis B and HIV viruses and can pose immense dangers.

In a Canadian study, a plant was infected with a crippled cucumber mosaic virus that lacked a gene needed for movement between plant cells. Within less than two weeks, the crippled plant found what it needed from neighboring genes – as evidence of gene mixing or horizontal transfer. This is

significant because genes that cause diseases are often crippled or engineered to be dormant in order to make the end product “safe.” Results of this kind led the US Department of Agriculture to hold a meeting in October of 1997 to discuss the risks and dangers of gene mixing and super viruses, but no regulatory action was taken. A French study also showed the recombination of RNA of two Cuomo viruses, and under conditions of minimal selection and in supposedly virus resistant transgenic plants.

13.2.3. Antibiotic resistance

In recent years health professionals have become alarmed by the increasing number of bacterial strains that are showing resistance to antibiotics. Bacteria develop resistance to antibiotics by creating antibiotic resistance genes through natural mutation. Biotechnologists use antibiotic resistance genes as selectable markers when inserting new genes into plants. In the early stages of the process scientists do not know if the target plant will incorporate the new gene into its genome. By attaching the desired gene to an antibiotic resistance gene the new GM plant can be tested by growing it in a solution containing the corresponding antibiotic. If the plant survives scientists know that it has taken up the antibiotic resistance gene along with the desired gene. There is concern that bacteria living in the guts of humans and animals could pick up an antibiotic resistance gene from a GM plant before the DNA becomes completely digested.

It is not clear what sort of risk the possibility of conferring antibiotic resistance to bacteria presents. No one has ever observed bacteria incorporating new DNA from the digestive system under controlled laboratory conditions. The two types of antibiotic resistance genes used by biotechnologists are ones that already exist in bacteria in nature so the process would not introduce new antibiotic resistance to bacteria. Nevertheless it is a concern and the FDA is encouraging biotechnologists to phase out the practice of using antibiotic resistance genes.

13.2.4. Birth Defects and Shorter Life Spans

rBGH in cows causes a rapid increase in birth defects and shorter life spans and the number of calves born with birth defects to dairy cows has increased significantly. Canada and the European Union have taken precautions and banned the use of rBGH in their dairy cows.

In a very recent study by Cornucopia Institute Research the following information was reported: "...The experience of actual GM-fed experimental animals is scary. When GM soy was fed to female rats, most of their babies died within three weeks—compared to a 10% death rate among the control group fed natural soy. The GM-fed babies were also smaller, and later had problems getting pregnant. When male rats were fed GM soy, their testicles actually changed color—from the normal pink to dark blue. Mice fed GM soy had altered young sperm. Even the embryos of GM fed parent mice had significant changes in their DNA. Mice fed GM corn in an Austrian government study had fewer babies, which were also smaller than normal..."

The American Academy of Environmental Medicine (AAEM) called on 'Physicians to educate their patients, the medical community, and the public to avoid GM (genetically modified) foods when possible and provide educational materials concerning GM foods and health risks.' They called for a moratorium on GM foods, long-term independent studies, and labeling. AAEM's position paper stated, 'Several animal studies indicate serious health risks associated with GM food,' including infertility, immune problems, accelerated aging, insulin regulation, and changes in major organs and the gastrointestinal system. They conclude: 'There is more than a casual association between GM foods and adverse health effects. There is causation,' as defined by recognized scientific criteria. 'The strength of association and consistency between GM foods and disease is confirmed in several animal studies.

13.2.5. Interior Toxins

"Pesticidal foods" have genes that produce a toxic pesticide inside the food's cells. The food is engineered to produce their own built in pesticide in every cell which produces a poison that splits open a bug's stomach and kills them when the bug tries to eat the plant. This represents the first time "cell-interior toxicity" is being sold for human consumption. There is little knowledge of the potential long-term health impacts. However, while some biotech companies claim that the pesticide called Bt has been approved safe and used by farmers for natural insect control, the Bt-toxin in GM plants is thousands of times more concentrated than the natural bug spray, can not be washed off the plants, and has properties of allergens. We are now ingesting this interior plant toxin from GM foods.

As individuals ingest more and more genetically modified foods and organisms into their body it has been shown that the bodies toxicity increases which leads to a ton of other potentially serious health problems. There is a definite link between Obesity, Cancer and Toxicity.

13.2.6. Decreased Nutritional Value

A genetically modified plant could theoretically have lower nutritional quality than its traditional counterpart by making nutrients unavailable or indigestible to humans. For example, phytate is a compound common in seeds and grains that binds with minerals and makes them unavailable to humans. An inserted gene could cause a plant to produce higher levels of phytate decreasing the mineral nutritional value of the plant (GEO-PIE). Another example comes from a study showing that a strain of genetically modified soybean produced lower levels of phytoestrogen compounds, believed to protect against heart disease and cancer, than traditional soybeans (Bakshi, 2003).

A study in the Journal of Medicinal Food showed that certain GM foods have lower levels of vital nutrients – especially phytoestrogen compounds thought to protect the body from heart disease and cancer. In another study of GM *Vicia Faba*, a bean in the same family as soy, there was also an increase in estrogen levels, what raises health issues – especially in infant soy formulas. Milk from cows with rBGH contains substantially higher levels of pus, bacteria, and fat. Monsanto's analysis of glyphosate-resistant soya showed the GM-line contained 28% more Kunitz-trypsin inhibitor, a known anti-nutrient and allergen.

13.2.7. Food Allergy

Food Allergy affects approximately 5% of children and 2% of adults in the U.S. and is a significant public health threat. Allergic reactions in humans occur when a normally harmless protein enters the body and stimulates an immune response. If the novel protein in a GM food comes from a source that is known to cause allergies in humans or a source that has never been consumed as human food, the concern that the protein could elicit an immune response in human increases. Although no allergic reactions to GM food by consumers have been confirmed, in vitro evidence suggesting that

some GM products could cause an allergic reaction has motivated biotechnology companies to discontinue their development

13.3. Effects on Environment

Genetic Engineering is often justified as a human technology, one that feeds more people with better food. Nothing could be further from the truth. With very few exceptions, the whole point of genetic engineering is to increase sales of chemicals and bio-engineered products to dependent farmers.

13.3.1. Soil Sterility and Pollution

In Oregon, scientists found GM bacterium (*klebsiella planticola*) meant to break down wood chips, corn stalks and lumber wastes to produce ethanol – with the post-process waste to be used as compost – rendered the soil sterile. It killed essential soil nutrients, robbing the soil of nitrogen and killed nitrogen capturing fungi. A similar result was found in 1997 with the GM bacteria *Rhizobium melitoli*. Professor Guenther Stotzky of New York University conducted research showing the toxins that were lethal to Monarch butterfly are also released by the roots to produce soil pollution. The pollution was found to last up to 8 months with depressed microbial activity. An Oregon study showed that GM soil microbes in the lab killed wheat plants when added to the soil.

13.3.2. Super weeds

It has been shown that genetically modified Bt endotoxin remains in the soil at least 18 months (according to Marc Lappé and Britt Bailey) and can be transported to wild plants creating super weeds – resistant to butterfly, moth, and beetle pests – potentially disturbing the balance of nature. A study in Denmark and in the UK showed super weeds growing nearby in just one generation. A US study showed the super weed resistant to glufosinate (which differs from glyphosate) to be just as fertile as non-polluted weeds. Another study showed 20 times more genetic leakage with GM plants – or a dramatic increase in the flow of genes to outside species. Also in a UK study by the National Institute of Agricultural Botany, it was confirmed that super weeds could grow nearby in just one generation. Scientists suspect that Monsanto's wheat will hybridize with goat grass, creating an invulnerable super weed. The National Academy of Science's study stated that "concern surrounds the

possibility of genes for resisting pests being passed from cultivated plants to their weedy relatives, potentially making the weed problem worse. This could pose a high cost to farmers and threaten the ecosystem.”

An experiment in France showed a GM canola plant could transfer genes to wild radishes, what persisted in four generations. Similarly, and according to *New Scientist*, an Alberta Canada farmer began planting three fields of different GM canola seeds in 1997 and by 1999 produced not one, but three different mutant weeds – respectively resistant to three common herbicides (Monsanto’s Roundup, Cyanamid’s Pursuit, and Aventis’ Liberty). In effect genetic materials migrated to the weeds they were meant to control. Now the Alberta farmer is forced to use a potent 2,4-D what GM crops promised to avoid use of. Finally Stuart Laidlaw reported in the *Toronto Star* that the Ontario government study indicated herbicide use was on the rise primarily largely due to the introduction of GM crops.

13.3.3. Destruction of Forest Life

GM trees or “super trees” are being developed which can be sprayed from the air to kill literally all of surrounding life, except the GM trees. There is an attempt underway to transform international forestry by introducing multiple species of such trees. The trees themselves are often sterile and flowerless. This is in contrast to rainforests teeming with life, or where a single tree can host thousands of unique species of insects, fungi, mammals and birds in an interconnected ecosphere. This kind of development has been called “death-engineering” rather than “life-” or “bio-engineering.” More ominously pollen from such trees, because of their height, has traveled as much as 400 miles or 600 kilometers – roughly 1/5 of the distance across the United States.

13.3.4. Terminator Trees

Monsanto has developed plans with the New Zealand Forest Research Agency to create still more lethal tree plantations. These super deadly trees are non-flowering, herbicide-resistant and with leaves exuding toxic chemicals to kill caterpillars and other surrounding insects – destroying the wholesale ecology of forest life. As George McGavin, curator of entomology Oxford University noted, “If you replace vast tracts of natural forest with flowerless trees, there will be a serious effect on the richness and abundance

of insects. If you put insect resistance in the leaves as well you will end up with nothing but booklice and earwigs. We are talking about vast tracts of land covered with plants that do not support animal life as a sterile means to cultivate wood tissue. That is a pretty unattractive vision of the future and I for one want no part of it.”

13.3.5. Super pests

Lab tests indicate that common plant pests such as cotton boll worms, will evolve into super pests immune from the Bt sprays used by organic farmers. The recent “stink bug” epidemic in North Carolina and Georgia seems linked to bioengineered plants that the bugs love. Monsanto, on their Farm source website, recommended spraying them with methyl parathion, one of the deadliest chemicals. So much for the notion of Bt cotton getting US farmers off the toxic treadmill. Pests the transgenic cotton was meant to kill – cotton bollworms, pink bollworms, and budworms – were once “secondary pests.” Toxic chemicals killed off their predators, unbalanced nature, and thus made them “major pests.”

13.3.6. Animal Bio-invasions

Fish and marine life are threatened by accidental release of GM fish currently under development in several countries – trout, carp, and salmon several times the normal size and growing up to 6 times as fast. One such accident has already occurred in the Philippines – threatening local fish supplies.

13.3.7. Killing Beneficial Insects

Studies have shown that GM products can kill beneficial insects – most notably the monarch butterfly larvae. Swiss government researchers found Bt crops killed lacewings that ate the cotton worms which the Bt targeted. A study reported in 1997 by New Scientist indicates honeybees may be harmed by feeding on proteins found in GM canola flowers. Other studies relate to the death of bees (40% died during a contained trial with Monsanto’s Bt cotton), springtails and ladybird beetles.

13.3.8. Poisonous to Mammals

In a study with GM potatoes, spliced with DNA from the snowdrop plant and a viral promoter (CaMV), the resulting plant was poisonous to mammals (rats) – damaging vital organs, the stomach lining and immune system. CaMV is a pararetro virus. It can reactivate dormant viruses or create new viruses – as some presume have occurred with the epidemic. CaMV is promiscuous, why biologist Mae Wan-Ho concluded that “all transgenic crops containing CaMV 35S or similar promoters which are recombinogenic should be immediately withdrawn from commercial production or open field trials. All products derived from such crops containing transgenic DNA should also be immediately withdrawn from sale and from use for human consumption or animal feed.”

13.3.9. Animal Abuse

Pig number 6706 was supposed to be a “super pig.” It was implanted with a gene to become a technological wonder. But it eventually became a “super cripple” full of arthritis, cross-eyed, and could barely stand up with its mutated body. Some of these mutations seem to come right out of Greek mythology – such as a sheep-goat with faces and horns of a goat and the lower body of a sheep. Two US biotech companies are producing genetically modified birds as carriers for human drug delivery – without any concern for animal suffering. Gene Works of Ann Arbor, Michigan has up to 60 birds under “development.” GM products, in general, allow companies to own the rights to create, direct, and orchestrate the evolution of animals.

13.3.10. Genetic Pollution

Carrying GM pollen by wind, rain, birds, bees, insects, fungus, bacteria – the entire chain of life becomes involved. Once released, unlike chemical pollution, there is no cleanup or recall possible. As mentioned, pollen from a single GM tree has been shown to travel 1/5th of the length of the United States. Thus there is no containing such genetic pollution. Experiments in Germany have shown that engineered oilseed rape can have its pollen move over 200 meters. As a result German farmers have sued to stop field trials in Berlin.

In Thailand, the government stopped field tests for Monsanto's Bt cotton when it was discovered by the Institute of Traditional Thai Medicine that 16 nearby plants of the cotton family, used by traditional healers, were being genetically polluted. US research showed that more than 50% of wild strawberries growing inside of 50 meters of a GM strawberry field assumed GM gene markers. Another showed that 25-38% of wild sunflowers growing near GM crops had GM gene markers.

A recent study in England showed that despite the tiny amount of GM plantings there (33,750 acres over two years compared to 70-80 million acres per year in the US) wild honey was found to be contaminated. This means that bees are likely to pollinate organic plants and trees with transgenic elements. Many other insects transport the by-products of GM plants throughout our environment, and even falling leaves can dramatically affect the genetic heritage of soil bacteria. The major difference between chemical pollution and genetic pollution is that the former eventually is dismantled or decays, while the latter can reproduce itself forever in the wild.

As the National Academy of Science's report indicated – “the containment of crop genes is not considered to be feasible when seeds are distributed and grown on a commercial scale.” Bioengineering firms are also developing fast growing salmon, trout, and catfish as part of the “blue revolution” in aquaculture. They often grow several times faster (6x faster for salmon) and larger in size (up to 39X) so as to potentially wipe out their competitors in the wild. There are no regulations for their safe containment to avoid ecological disasters. They frequently grow in “net pens,” renown for being torn by waves, so that some will escape into the wild. If so, commercial wild fish could be devastated according to computer models in a study of the National Academy of Sciences by two Purdue University scientists (William Muir and Richard Howard). All of organic farming – and farming per se – may eventually be either threatened or polluted by this technology.

13.3.11. General Economic Harm to Small Family Farms

GM seeds sell at a premium, unless purchased in large quantities, which creates a financial burden for small farmers. Many GM products, such as rBGH, seem to offer a boom for dairy farmers – helping their cows produce considerably more milk. But the end result has been a lowering of prices, again putting the smaller farmers out of business. We can find similar trends

with other GM techniques – as in pig and hen raising made more efficient. The University of Wisconsin's GM brooding hens lack the gene that produces prolactin proteins. The new hens no longer sit on their eggs as long, and produce more. Higher production leads to lower prices in the market place. The end result is that the average small farmer's income plummeted while a few large-scale, hyper-productive operations survived along with their "input providers" (companies selling seeds, soil amendments, and so on).

In an on-going trend, the self-sufficient family farmer is shoved to the very lowest rung of the economic ladder. In 1910 the labor portion of agriculture accounted for 41% of the value of the finally sold produce. Now the figure has been estimated at between 6-9% in North America. The balance gets channeled to agri-input and distribution firms – and more recently to biotech firms. Kristin Dawkins in *Gene Wars: The Politics of Biotechnology*, points out that between 1981 and 1987, food prices rose 36%, while the percentage of the pie earned by farmers continued to shrink dramatically.

13.3.12. Losing Natural Pesticides

Organic farmers have long used "Bt" (a naturally occurring pesticidal bacterium, *Bacillus thuringiensis*) as an invaluable farming aide. It is administered at only certain times, and then sparingly, in a diluted form. This harms only the target insects that bite the plant. Also in that diluted form, it quickly degrades in the soil. By contrast, genetically engineered Bt corn, potatoes and cotton – together making up roughly a third of US GM crops – all exude this natural pesticide. It is present in every single cell, and pervasively impacts entire fields over the entire life span of crops. This probably increases Bt use at least a million fold in US agriculture. According to a study conducted at NYU, BT residues remained in the soil for as much as 243 days. As an overall result, agricultural biologists predict this will lead to the destruction of one of organic farming's most important tools. It will make it essentially useless. A computer model developed at the University of Illinois predicted that if all US Farmers grew Bt resistant corn, resistance would occur within 12 months. Scientists at the University of North Carolina have already discovered Bt resistance among moth pests that feed on corn. The EPA now requires GM planting farmers to set aside 20-50% of acres with non-BT corn to attempt to control the risk and to help monarch butterflies survive.

13.3.13. Monopolization of Food Production

The rapid and radical change in the human diet was made possible by quick mergers and acquisitions that moved to control segments of the US farming industry. Although there are approximately 1500 seed companies worldwide, about two dozen control more than 50% of the commercial seed heritage of our planet. The consolidation has continued to grow. In 1998 the top five soy producers controlled 37% of the market (Murphy Family Foods; Carroll's Foods, Continental Grain, Smithfield Foods, and Seaboard). One year later, the top five controlled 51% (Smithfield, having acquired Murphy's and Carroll's, Continental, Seaboard, Prestige and Cargill). Cargill and Continental Grain later merged.

With corn seed production and sales, the top four seed companies controlled 87% of the market in 1996 (Pioneer Hi-Bred, Holden's Foundation Seeds, DeKalb Genetics, and Novaris). In 1999, the top three controlled 88% (Dupont having acquired Pioneer, Monsanto having acquired Holden's and DeKalb, and Novaris. In the cotton seed market, Delta and Land Pine Company now control about 75% of the market. The concentration is staggering. National farming associations see this dwindling of price competition and fewer distribution outlets as disfavoring and threatening the small family farm. Average annual income per farm has plummeted throughout the last decade. Almost a quarter of all farm operating families live below the poverty level, twice the national average – and most seek income from outside the farm to survive. A similar pattern is developing in Europe.

13.3.14. Impact on Long -Term Food Supply

If food production is monopolized, the future of that supply becomes dependent on the decisions of a few companies and the viability of their seed stocks. Like the example of Peru, there are only a few remaining pockets of diverse seed stocks to insure the long-term resilience of the world's staple foods. All of them are in the Third World. Food scientists indicate that if these indigenous territories are disturbed by biotech's advance, the long-term vitality of all of the world's food supply is endangered.

13.4. Conclusion

Genetically-modified foods have the potential to solve many of the world's hunger and malnutrition problems, and to help protect and preserve the environment by increasing yield and reducing reliance upon chemical pesticides and herbicides. Yet there are many challenges ahead for governments, especially in the areas of safety testing, regulation, international policy and food labeling. Many people feel that genetic engineering is the inevitable wave of the future and that we cannot afford to ignore a technology that has such enormous potential benefits.

What will happen if this technology is allowed to spread? Fifty years ago few predicted that chemical pollution would cause so much vast environmental harm. Now nearly 1/3rd of all species are threatened with extinction (and up to half of all plant species and half of all mammals). Few also knew that cancer rates would skyrocket during this same period. Nowadays approximately 41% on average of Americans can expect cancer in their lifetime. So we must proceed with caution to avoid causing unintended harm to human health and the environment as a result of our enthusiasm for this powerful technology. The use of genetically modified organisms in foods was recently banned in Europe,

14. Effects of Food Additives on Health

14.1. Introduction

A food additive is any substance not commonly regarded or used as food, which is added to, or used in or on, food at any stage to affect its keeping quality, texture, consistency, taste, colour, alkalinity or acidity. Food additives in use today can be divided roughly into three main types: cosmetics, preservatives and processing aids, totalling presently about 3,794 different additives, of which over 3,640 are used purely as cosmetics, 63 as preservatives and 91 as processing aids. The growth in the use of food additives has increased enormously in the past 30 years, totalling now over 200,000 tonnes per year. Therefore it has been estimated that as today about 75% of the Western diet is made up of various processed foods, each person is now consuming on average 8-10 lbs of food additives per year, with some possibly eating considerably more. With the great increase in the use of food additives, there also has emerged considerable scientific data linking food additive intolerance with various physical and mental disorders, particularly with childhood hyperactivity. Food additive is also very dangerous for health. Consuming foods mixed with additives can cause negative effect like insomnia, nervousness, restlessness, irritability, and mood changes.

14.2. Health Effects of Food Additives

Most people who consume food additives will experience in allergies and a lot of them will suffer from irritable bowel syndrome. Harmful effects of food additives will affect the health, mood, skin, your behavior, your bowel movements that make it difficult to defecate. Besides those mentioned above, the harmful effects of food additive is causing migraines. Migraine disease has increased threefold since the seventies. According to the study, this headaches can be overcome by avoiding food additives. Switch to organic foods that are healthy and free from danger. Then, since the seventies, people with autism increased 10-fold worldwide. Based on the study, all of this relates to food additives. Simply put, food additives are dangerous to health even though famous for its natural ingredients. For example, calcium propionate used as a food preservative has been shown to have negative effects such as sleep disturbance, waking at night, depression, bedwetting, anxiety and nasal congestion.

Furthermore, consuming foods that contain salicylates has been proven to cause tinnitus, vertigo, insomnia, hearing loss, behavioral changes in children and others, while food additives such as monosodium glutamate has been proved to be very dangerous, especially for children. The harmful effects of “monosodium glutamate” food additive are inhibiting brain development in children. So, avoid the use of food additives in order to live a healthy life. Consuming organic foods is the solution to better health. Organic foods are best choice for kids.

14.3. Some Food Additives and their Side-Effects

Tartrazine, which is primarily used by the soft drink industry, is one of the colours most frequently implicated in food intolerance studies. Adverse reactions to tartrazine seem to occur most commonly in subjects who are also sensitive to acetylsalicylic acid (ASA), a finding which was also observed by Feingold and his team. Depending on the test protocol followed, it has been found that between 10-40% of aspirin-sensitive patients are indeed usually also affected by tartrazine, the reactions including asthma, urticaria, rhinitis and, as previously mentioned, childhood hyperactivity.

One study found that an oral administration of 50mg tartrazine to 122 patients suffering from allergy-related disorders, evoked the following reactions; feeling of suffocation, weakness, heat sensation, palpitations, blurred vision, rhinorrhoea, pruritus and urticaria. Even though 50mg could be considered as a substantial dose, such a quantity of tartrazine could easily be consumed by an individual drinking only a few bottles of soft drinks per day.

Another carefully conducted double-blind placebo- controlled trial on 76 children diagnosed as hyperactive, showed that tartrazine and benzoates provoked abnormal behaviour patterns in 79% of them. In addition, a double-blind placebo-controlled trial on 10 hyperactive children when compared to controls, found that tartrazine increases urinary zinc secretion, and decreases serum and salivary zinc concentration in the hyperactives, with a corresponding deterioration in their behaviour. This phenomenon was not found among the controls.

It was suggested therefore that tartrazine seems to act as a zinc chelating agent in susceptible individuals. Furthermore, that zinc depletion may also be one of the potential causes of childhood hyperactivity. Although

tartrazine seems to be most frequently associated with adverse reactions, there are also other colouring agents which are known to cause mental and/or physical ill-effects.

Curcumin, used mainly in flour confectionery and margarine, has been found to cause mutations in bacteria and when fed to pigs, it increased the weight of their thyroid glands causing, in high doses, severe thyroid damage.

Sunset Yellow, used in biscuits, has been found to damage kidneys and adrenals when fed to laboratory rats. It has also been found to be carcinogenic when fed to animals.

Carmoisine, used mainly in jams and preserves, was found by the US Certified Color Manufacturers Association to be unavoidably contaminated with low levels of beta- naphthylamine, which is a well known carcinogen; it has also been found to be mutagenic in animal studies.

Amaranth has been found, when fed to laboratory rats, to cause cancer, birth defects, still births, sterility and early foetal deaths. Subsequent work has also found that amaranth can cause female rodents to reabsorb some of their own foetuses.

Ponceau 4R , used mainly in dessert mixes, has been found to exhibit a weak carcinogenic action.

Erythrosine , used in candied cherries and childrens' sweets, has been found to act as a potent neurocompetitive dopamine inhibitor of dopamine uptake by nerve endings when exposed in vitro on a rat brain. Other studies have shown that erythrosine can have an inhibitory action also on other neurotransmitters, resulting in an increased concentration of neurotransmitters near the receptors, thus functionally augmenting the synaptic neurotransmission. There is now some evidence that a reduced dopamine turnover may lead to childhood hyperactivity. Similar findings have been linked with a reduction of noradrenaline. Erythrosine also has been found to have a possible carcinogenic action when tested on animals.

Caramels , of which over 100 different formulations are currently in use, are widely used by the cola drinks industry, as well as the beer and

alcohol industry. It is also used as a colouring agent in crisps, bread, sauces, gravy browning etc. The main recurring problem about the safety of caramels concerns the presence of an impurity called 4-Methylimidazole, produced by processes using ammonia, which leads to convulsions when fed to rats, mice and chicks. It has been also found that ammoniated caramels can affect adversely the levels of white blood cells and lymphocytes in laboratory animals. Furthermore, a study on rabbits provided evidence that even small doses of ammoniated caramels seem to inhibit the absorption of vitamin B₆.

Brown FK is mainly used as a colouring agent in fish, such as kippers. Two of the primary metabolites of this colouring have been found to act as a cardiotoxin. It has been also observed, when fed in the long term to mice, to cause potentially hazardous nodules to form in the liver. Furthermore it has been found to cause mutations in some bacteria, implying that it may also act as a mutagenic and/or carcinogenic agent in humans.

Preservatives/Antioxidants

Benzoates, used mainly in marinated fish, fruit- based fillings, jam, salad cream, soft drinks and beer, have been found to provoke urticaria, angioedema and asthma. Furthermore, they have also been directly linked with childhood hyperactivity.

Sulphites, used mainly in dried fruits, fruit juices and syrups, fruit-based dairy deserts, biscuit doughs, cider, beer and wine, have been linked with pruritus, urticaria, angioedema and asthma. When fed to animals, sulphites have also been found to have a mutagenic action.

Nitrates and nitrites, used in bacon, ham, cured meats, corned beef and some cheeses, have been found to cause headaches in susceptible individuals. In addition, these chemicals have been linked with cancer both in animal and human studies. They have also been found to be mutagenic when fed to mammals.

Butylated hydroxyanisole - BHA , used in soup mixes and cheese spread, has been found to be tumour-producing when fed to rats. In human studies it has been linked with urticaria, angioedema and asthma.

Monosodium glutamate (MSG), a flavour enhancer, used in savoury foods, snacks, soups, sauces and meat products, has been associated with a conjunction of symptoms in susceptible individuals, such as severe chest and/or facial pressure and overall burning sensations, not unlike a feeling that the victim is experiencing a heart attack. MSG has also been found to precipitate a severe headache and/or asthma in susceptible individuals. In susceptible children MSG has been linked with epilepsy-type "shudder" attacks. In animal studies it has been found to damage the brains of young rodents.

Sweeteners

Saccharin, used as sweetening tablets and widely used by the soft drink and sweet food industry, has been shown to produce cancer when tested on animals. Saccharin has also been found to be mutagenic and growth inhibiting, as well causing congenital malformations in animal studies. The fact that any substance which has been found to be carcinogenic also seems to have a mutagenic action, was established by testing 300 different carcinogenic chemicals for mutagenicity. The results showed that of the 300 carcinogenic chemicals tested, 90% were also found to have a mutagenic action.

Aspartame, of which the key ingredient is the amino acid phenylalanine, is also widely used by the soft drink and sweet food industry. When fed to rats, aspartame was found to double the level of phenylalanine in their brains, which re-doubled when other carbohydrates were consumed at the same time. This combination was found to give a great rise in brain tyrosine, followed by a considerable reduction in brain tryptophan levels. Low tryptophan levels have been directly linked with both aggressive and violent behaviour.

Furthermore, as dietary tryptophan acts as a precursor for serotonin (5-hydroxy-tryptamine, 5HT), reduced tryptophan levels will also result in a reduction of brain serotonin levels, which has been directly linked with both hyperactive and aggressive behaviour.

14.4. Who are Affected ?

Young children seem to serve always as the first sentinels of any environmental contamination, because of their immaturity of enzymatic detoxifying mechanism, incomplete function of excretory organs, low levels of plasma protein capable of binding toxic chemicals and incomplete development of physiological barriers such as the blood-brain barrier. The young, developing nervous system seems to be particularly vulnerable. For example, results of some research studies, which incidentally were rather critical of Feingold's claims, found that only the very youngest of the children tested reacted adversely to artificial food additives.

It should be stressed however that the period of organ formation and development stretches long beyond the moment of birth. The Fetal Alcohol Syndrome is a useful example, which arises with fetal exposure to neurotoxic agents such as alcohol.

Similar adverse effects have been attached to maternal smoking, to lead contamination and now, more recently, to food additives. Using animal experiments, it has been found that the fetus may be more susceptible to tumour development than an adult animal.

Evidence is also accumulating that non-carcinogenic substances may cause a variety of biochemical changes, including alterations in the fetal enzyme development at levels at which the mother is asymptomatic.

One class of compounds dangerous to the fetus, often in very low concentration, are the mutagens, which are able to react with and injure chromosomes and genes carrying the genetic code. Furthermore, it has been found that mutagens not only cause mutations but are also capable of damaging and killing living cells, thus inflicting the greatest damage very early in pregnancy or during the weeks before conception.

14.5. Nutritional and Toxic Chemical Influences on Behaviour

Dietary and toxicological factors in behavioural disorders have been sadly neglected by mainstream psychiatry, even though it is known that brain function itself involves subtle chemical and electrical processes, which can be easily altered and modified with the use of various psychoactive drugs.

Therefore, it is difficult to comprehend why the role of nutritional influences on behaviour has been completely ignored, even though the precursors of neurotransmitter molecules, essential for the brain function, are only found in foods. Furthermore, they cannot be synthesized nor stored by the brain, unless introduced by appropriate dietary substances.

When the availability of these dietary precursors are reduced, the neurotransmitter synthesis will become impaired, with the consequent changes in both thinking process and behaviour. When this happens, learning and memory tasks may become impaired or disturbed, intellectual development inhibited and overt behaviours disordered, depending upon which dietary precursor is deficient or missing. In addition, various neurotoxins such as alcohol, heroin, LSD, nicotine, lead, organic solvents, individual food intolerances and some food additives can modify neurotransmitter release, resulting in subtle or exaggerated behavioural changes.

14.6. Food Additives and Malnutrition

Another form of risk posed by additives is the loss of the nutritional value of the food, which can result in inappropriate diets and subclinical malnutrition. The wide use of food additives can contribute to malnutrition in the following ways; the common factor in most foods containing additives is high salt, sucrose and fat content.

Pure sucrose, by definition, contains literally no nutrients, only calories; fat, on the other hand, contains few nutrients and is very high in calories. In addition, foods containing additives are mainly processed foods, which have lost a substantial proportion of their nutritional value through the processing procedure.

Even though some vitamins and/or minerals are sometimes added to some foods after processing, the ratio of essential nutrients to calories is usually still quite inadequate, resulting in a high calorie, but a low nutritional, intake. This type of diet, because of the high calorie and low nutritional content, can result in less than optimum nutrition and therefore subclinical and/or marginal malnutrition.

14.7. Subclinical Malnutrition in Reproduction

Inefficient diet not only affects the brain and behaviour of an individual, it also has serious long-term consequences on reproduction and on the future infant's health, as a good maternal diet is of paramount importance in relation to a healthy fetal development and to a successful pregnancy outcome. For example folic acid deficiency has been directly linked with spina bifida.

Zinc in turn is involved in the process of cell differentiation and replication, therefore zinc deficiency can lead to diverse teratogenic congenital malformations, and premature delivery, as well as small for gestational age babies. The adverse effects of a reduced state of other single essential nutrient compounds on reproduction have also been well documented.

Subclinical maternal malnutrition has also been frequently associated with low birth weight infants, which in turn appears to have a clear negative effect on the infant's future health .

It has been found that infants born with low birth weight are more prone in adult life to suffer from cardiovascular diseases, have a high serum cholesterol concentration, as well as suffer from hypertension, hyperlipidaemia and diabetes mellitus.

Subclinical maternal malnutrition can also lead to reduction of fetal brain development and subsequently to various intellectual deficits. The brain develops much more rapidly than most other organs in the embryo. In fact by about the 20th week of pregnancy it already contains most of the neurons present in the adult brain, excluding the cerebellum which is initially slower to develop but quicker to mature. By mid-pregnancy, almost all the neurons found in the adult brain have been produced.

If the maternal diet is not sufficient during this rapid fetal brain neuronal development, this can permanently reduce the number of neurons formed in the foetal cerebrum with its negative consequences to the future intellect.

14.8. Conclusion

The use of food additives has increased enormously in the last few decades. As the result, it has been estimated that today about 75% of the Western diet is made up of various processed foods, each person consuming on average 8-10 lbs of food additives per year, with some possibly eating even more.

The following adverse effects have been attributed to the consumption of food additives: eczema, urticaria, angioedema, exfoliative dermatitis, irritable bowel syndrome, nausea, vomiting, diarrhoea, rhinitis, bronchospasm, migraine, anaphylaxis, hyperactivity and other behavioural disorders. In order to improve the present situation, the following recommendations are made;

1. All non-essential food additives should be banned, particularly all cosmetic agents such as food colourants.
2. All foods which include additives with carcinogenic, mutagenic and teratogenic properties should be clearly labelled with the appropriate warning.
3. All food additives should be banned from foods which may be consumed by infants and young children.
4. The amount of TV advertising which encourages children to buy and eat unhealthy junk food should be vigorously cut down as children are presently surrounded by images promoting extremely unhealthy eating habits.
5. All foods that have little or no nutritional value should be discouraged from all promotions.
6. Local Education Authorities should include in their health education curricula specific lectures stressing the prime importance of good nutrition in both physical and mental health.

The Government must pass a law refusing permission for the food industries to add continuously into our everyday foods and beverages demonstrably toxic agents for cosmetic purposes only. If not for any other reason, at least in order to protect the health of our significant population of young children, youths, adolescents and adults, as well as the health of our future generation.

15. Effect of Formaldehyde on Human Health

15.1. Introduction

Formaldehyde is a colorless, flammable, strong-smelling chemical that is used in building materials and to produce many household products. It is used in pressed-wood products, such as particleboard, plywood, and fiberboard; glues and adhesives; permanent-press fabrics; paper product coatings; and certain insulation materials. In addition, formaldehyde is commonly used as an industrial fungicide, germicide, and disinfectant, and as a preservative in mortuaries and medical laboratories. Formaldehyde also occurs naturally in the environment. It easily becomes a gas at room temperature, which makes it part of a larger group of chemicals known as volatile organic compounds (VOCs). When an item gives off formaldehyde, it is released into the air through a process called off-gassing.

Formaldehyde is an important precursor to many other materials and chemical compounds. In 1996, the installed capacity for the production of formaldehyde was estimated to be 8.7 million tons per year. It is mainly used in the production of industrial resins, e.g., for particle board and coatings. In view of its widespread use, toxicity, and volatility, formaldehyde poses a significant danger to human health. In 2011, the US National Toxicology Program described formaldehyde as "known to be a human carcinogen".

15.2. Industrial Applications

The textile industry uses formaldehyde-based resins as finishers to make fabrics crease-resistant. Formaldehyde-based materials are key to the manufacture of automobiles, and used to make components for the transmission, electrical system, engine block, door panels, axles and brake shoes. The value of sales of formaldehyde and derivative products was over \$145 billion in 2003, about 1.2% of the gross domestic product (GDP) of the United States and Canada. Including indirect employment, over 4 million people work in the formaldehyde industry across approximately 11,900 plants in the U.S. and Canada. Formaldehyde is also a precursor to polyfunctional alcohols such as pentaerythritol, which is used to make paints and explosives.

Formaldehyde solutions are used as a fixative for microscopy and histology because of formaldehyde's ability to perform the Mannich reaction. Several European countries restrict the use of formaldehyde, including the import of formaldehyde-treated products and embalming. Starting September 2007, the European Union banned the use of formaldehyde due to its carcinogenic properties.

15.3. Contaminant in Food

Formaldehyde occurs naturally and is "an essential intermediate in cellular metabolism in mammals and humans." At high concentrations it is probably unhealthy. Scandals have broken in both the 2005 Indonesia food scare and 2007 Vietnam food scare regarding the addition of formaldehyde to foods to extend shelf life. In 2011, after a four-year absence, Indonesian authorities found foods with formaldehyde being sold in markets in a number of regions across the country. Besides using formaldehyde, they also used borax, but not in combination.

In August 2011, at least at two Carrefour supermarkets, the Central Jakarta Livestock and Fishery Sub-Department found a sweet glutinous rice drink (cendol) contained 10 parts per million of formaldehyde. In 2014, the owner of two noodle factories in Bogor, Indonesia; was arrested for using formaldehyde in noodles. 50 kg of formaldehyde was confiscated. Foods known to be contaminated include noodles, salted fish, and tofu; chicken and beer are also rumored to be contaminated. In some places, such as China, formaldehyde is still used illegally as a preservative in foods, which exposes people to formaldehyde ingestion.

In humans, the ingestion of formaldehyde has been shown to cause vomiting, abdominal pain, dizziness, and in extreme cases can cause death. Testing for formaldehyde is by blood and/or urine by gas chromatography-mass spectrometry. Other methods include infrared detection, gas detector tubes, etc., of which HPLC is the most sensitive. In the early 1900s, it was frequently added by US milk plants to milk bottles as a method of pasteurization due to the lack of knowledge regarding formaldehyde's toxicity.

In 2011 in Nakhon Ratchasima, Thailand, truckloads of rotten chicken were exposed to formaldehyde in which "a large network," including 11 slaughterhouses run by a criminal gang, were implicated. In 2012, 1 billion rupiah (almost USD100,000) of fish imported from Pakistan to Batam, Indonesia, were found laced with formaldehyde.

Formalin use in foods is a crucial problem in Bangladesh. Local stores and supermarkets often sell fruits, fishes, and vegetables that have been treated with formalin to keep them fresh. **However, in 2015, a Formalin Control Bill was passed in the Parliament of Bangladesh with a provision of life-term imprisonment as the maximum punishment and in addition 2,000,000 BDT as fine but not less than 500,000 BDT for importing, production or hoarding of formalin without license.**

15.4. Effects on Human Health

As formaldehyde resins are used in many construction materials it is one of the more common indoor air pollutants. At concentrations above 0.1 ppm in air formaldehyde can irritate the eyes and mucous membranes, resulting in watery eyes. Formaldehyde inhaled at this concentration may cause headaches, a burning sensation in the throat, and difficulty breathing, and can trigger or aggravate asthma symptoms. In concentrations of 100 parts per million or more, formaldehyde is dangerous to breathe in, according to the Occupational Safety and Health Administration (OSHA). It affects the respiratory system to the point where pneumonia, bronchitis, asthma and pulmonary edema can develop. Contact with the skin can lead to contact dermatitis, hives and edema, says the Occupational Safety & Health Administration. Formaldehyde that comes in contact with the eyes can lead to injury to the cornea and blindness.

A 1988 Canadian study of houses with urea-formaldehyde foam insulation found that formaldehyde levels as low as 0.046 ppm were positively correlated with eye and nasal irritation. A recent review of studies has shown a strong association between exposure to formaldehyde and the development of childhood asthma. The primary exposure concern is for the workers in the industries producing or using formaldehyde.

There may also be a link between formaldehyde and leukemia and the abnormal changes in the chromosomes of young white blood cells of people who are exposed to the gas, says the American Cancer Society.

Ingestion of even a diluted amount of formaldehyde can be fatal, says OSHA. It can damage the stomach, liver, kidneys, pancreas, spleen and central nervous system.

15.5. How is the General Population Exposed to Formaldehyde?

According to a 1997 report by the U.S. Consumer Product Safety Commission, formaldehyde is normally present in both indoor and outdoor air at low levels, usually less than 0.03 parts of formaldehyde per million parts of air (ppm). Materials containing formaldehyde can release formaldehyde gas or vapor into the air. One source of formaldehyde exposure in the air is automobile tailpipe emissions.

During the 1970s, urea-formaldehyde foam insulation (UFFI) was used in many homes. However, few homes are now insulated with UFFI. Homes in which UFFI was installed many years ago are not likely to have high formaldehyde levels now. Pressed-wood products containing formaldehyde resins are often a significant source of formaldehyde in homes. Other potential indoor sources of formaldehyde include cigarette smoke and the use of unvented fuel-burning appliances, such as gas stoves, wood-burning stoves, and kerosene heaters.

Industrial workers who produce formaldehyde or formaldehyde-containing products, laboratory technicians, certain health care professionals, and mortuary employees may be exposed to higher levels of formaldehyde than the general public. Exposure occurs primarily by inhaling formaldehyde gas or vapor from the air or by absorbing liquids containing formaldehyde through the skin.

15.6. Can Formaldehyde Cause Cancer?

The formaldehyde theory of carcinogenesis was proposed in 1978. In 1987 the U.S. EPA classified it as a **probable human carcinogen**, and after more studies the WHO International Agency for Research on Cancer (IARC) in 1995 also classified it as a **probable human carcinogen**. Further information and evaluation of all known data led the IARC to reclassify formaldehyde as a **known human carcinogen** associated with nasal sinus

cancer and nasopharyngeal cancer. Recent studies have also shown a positive correlation between exposure to formaldehyde and the development of leukemia, particularly myeloid leukemia. Nasopharyngeal and sinonasal cancers are relatively rare, with a combined annual incidence in the United States of < 4,000 cases. About 25,000 cases of myeloid leukemia occur in the United States each year. Workplace exposure to inhaled chemicals is among the most important risk factors for sinonasal cancers. Professionals exposed to formaldehyde in their occupation, such as funeral industry workers and embalmers, showed an increased risk of leukemia and brain cancer compared with the general population. Other factors are important in determining individual risk for the development of leukemia or nasopharyngeal cancer.

15.7. What has been done to Protect Workers from Formaldehyde?

In 1987, OSHA established a Federal standard that reduced the amount of formaldehyde to which workers can be exposed over an 8-hour workday from 3 ppm to 1 ppm. In May 1992, the standard was amended, and the formaldehyde exposure limit was further reduced to 0.75 ppm.

15.8. How can People Limit Formaldehyde Exposure in their Homes?

Formaldehyde is a chemical used in the production of adhesives, bonding agents and solvents. For this reason, it is commonly found in a variety of consumer products including:

- Pressed-wood products (plywood, particle board, paneling)
- Foam insulation
- Wallpaper and paints
- Some synthetic fabrics (example: permanent press)
- Some cosmetics and personal products

Formaldehyde is also a byproduct of combustion. When burning natural gas, kerosene, gasoline, wood, or tobacco, formaldehyde is produced. Automobile exhaust is a common source of formaldehyde in our environment. Tobacco smoking in the home is another source of the chemical in the indoor environment.

The best way to reduce your exposure is to avoid products that contain formaldehyde, and to not allow cigarette smoking in your home. Look for products that are labeled as 'no' or 'low' VOC or formaldehyde. When purchasing pressed wood products for your home, look for those that are labeled as compliant with American National Standards Institute (ANSI) or California Air Resources Board Air Toxics Control Measure (CARB- ACTM) standards.

When purchasing products that may contain formaldehyde, methods to lower your exposure include:

- **Allow products to off-gas:** Remove the packaging from products and allow them to air out before bringing them into your house. Consider asking the manufacturer or store to leave the product unsealed in their warehouse for a few days before delivery. You may also consider purchasing a floor model where chemicals have already off-gassed.
- **Ventilate your home:** Increase the supply of fresh air to lower the concentration of formaldehyde. This can be done by opening windows, using fans or bringing in fresh air through a central ventilation system (such as a furnace air exchanger).
- **Control the heat and humidity:** Lower the temperature and humidity in the home through air conditioning and dehumidification. The amount of formaldehyde released goes up with increases in air temperature and humidity.

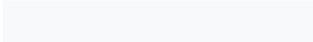
To minimize exposure to combustion by-products, including formaldehyde and carbon monoxide, ensure that combustion sources are properly maintained and vented outdoors. Avoid smoking indoors.

15.9. Conclusion

In the residential environment, formaldehyde exposure comes from a number of different routes; formaldehyde can off-gas from wood products, such as plywood or particle board, but it is produced by paints, varnishes, floor finishes, and cigarette smoking as well. In July 2016, the United States Environmental Protection Agency (EPA) released a prepublication version of its final rule on Formaldehyde Emission Standards for Composite Wood Products. These new rules will impact manufacturers, importers, distributors, and retailers of products containing composite wood, including fiberboard,

particleboard and various laminated products, who will need to comply with more stringent record-keeping and labeling requirements.

The United States Environmental Protection Agency (EPA) allows no more than 0.016 ppm formaldehyde in the air in new buildings constructed for that agency. A U.S. Environmental Protection Agency study found a new home measured 0.076 ppm when brand new and 0.045 ppm after 30 days. The Federal Emergency Management Agency (FEMA) has also announced limits on the formaldehyde levels in trailers purchased by that agency. The EPA recommends the use of "exterior-grade" pressed-wood products with phenol instead of urea resin to limit formaldehyde exposure, since pressed-wood products containing formaldehyde resins are often a significant source of formaldehyde in homes.



16. Arsenic toxicity and human health

16.1. Introduction

Arsenic, a metalloid, occurs naturally, being the twentieth most abundant element in the earth's crust and is a component of more than 245 minerals. The inorganic forms consisting mostly of arsenite and arsenate compounds are toxic to human health. Humans are exposed to arsenic primarily from air, food and water. Drinking water may be contaminated with arsenic from arsenical pesticide, natural mineral deposits or improperly disposed arsenical chemicals. However, elevated arsenic level in drinking water is the major cause of arsenic toxicity in the world. Reports of arsenic contamination in water are available from more than 30 countries in the world.

Arsenic toxicity (arsenicosis) due to drinking of arsenic contaminated ground water is a major environmental health hazard throughout the world. A lot of new information is emerging from extensive research on health effects of chronic arsenic toxicity (CAT) in humans during the last two decades. CAT also produces various systemic manifestations over and above skin lesions, important ones being chronic lung disease like chronic bronchitis, chronic obstructive pulmonary disease and bronchiectasis, liver disease like non-cirrhotic portal fibrosis and other diseases like polyneuropathy, peripheral vascular disease, hypertension and ischaemic heart disease, diabetes mellitus, non-pitting oedema of feet/hands, weakness and anaemia. Cancer of skin, lung and urinary bladder are important cancers associated with chronic arsenic toxicity.

16.2. Sources of Exposure

Arsenic is a natural component of the earth's crust and is widely distributed throughout the environment in the air, water and land. It is highly toxic in its inorganic form. People are exposed to elevated levels of inorganic arsenic through drinking contaminated water, using contaminated water in food preparation and irrigation of food crops, industrial processes, eating contaminated food and smoking tobacco.

Long-term exposure to inorganic arsenic, mainly through drinking contaminated water, eating food prepared with this water and eating food

irrigated with arsenic-rich water, can lead to chronic arsenic poisoning. Skin lesions and skin cancer are the most characteristic effects.

The lethal dose of arsenic in humans is 2-20 mg/kg, or 140 to 1400 mg for an average-sized adult. A 140-mg potentially lethal dose is the same as 0.145 grams. Less than 1/8 teaspoon can be fatal to a healthy adult, while even less could kill a child, an adult with impaired health, or an elderly person.

16.2.1. Background Exposures

Most people are exposed to constant but low levels of arsenic. Normally, background air levels are less than 0.1 $\mu\text{g}/\text{m}^3$ and drinking water less than 5 $\mu\text{g}/\text{L}$, but water levels can be significantly higher. The total average daily exposure to arsenic is about 20 $\mu\text{g}/\text{day}$ from food and water (assuming 2000 mL/day average water consumption at 5 $\mu\text{g}/\text{L}$ arsenic), but this can vary significantly depending on diet and water source.

16.2.2. Food Exposure

Fish, shellfish, meat, poultry, dairy products and cereals can also be dietary sources of arsenic, although exposure from these foods is generally much lower compared to exposure through contaminated groundwater. In seafood, arsenic is mainly found in its less toxic organic form.

Arsenic may be present in grains, fruits, vegetables and other foods. Arsenic exposure from ingested foods usually comes from food crops grown in arsenic-contaminated soil and/or irrigated with arsenic-contaminated water. Most crops don't readily take up much arsenic from the ground, but rice is an exception—it takes up arsenic from soil and water more readily than other grains. In addition, some seafood has high levels of less toxic organic arsenic. Food usually supplies less than 10 $\mu\text{g}/\text{day}$ of arsenic but can be higher with the consumption of rice and fish, and particularly shellfish, which can have arsenic levels up to 30 $\mu\text{g}/\text{g}$.

The US Food and Drug Administration (FDA) has been monitoring the levels of arsenic in foods for decades and increased its testing in 2011. Arsenic levels in apples have been raised as a concern, but FDA's results have confirmed that the amount of arsenic in apple juice is low.

16.2.3. Water Exposure

The greatest threat to public health from arsenic originates from contaminated groundwater. Inorganic arsenic is naturally present at high levels in the groundwater of a number of countries, including Argentina, Bangladesh, Chile, China, India, Mexico, and the United States of America. Drinking-water, crops irrigated with contaminated water and food prepared with contaminated water are the sources of exposure.

Arsenic poisoning from well water remains a serious worldwide human health concern. Tens of millions of people in Bangladesh are estimated to have been exposed to arsenic-contaminated water, resulting in an estimated 24,000 deaths each year. People in Argentina, Chile and Taiwan also have elevated arsenic in their drinking water. This is also relevant to areas of the western United States that have elevated levels of arsenic in drinking water. High levels of arsenic in local soil or rock contaminate the local water supply.

In the United States, the federal government struggled for years to establish standards of arsenic in drinking water. On January 22, 2001, EPA adopted a new standard for arsenic in drinking water of 0.01 mg/l, or 10 parts per billion (ppb), replacing the old standard of 50 ppb.

16.2.4. Industrial processes

Arsenic is used industrially as an alloying agent, as well as in the processing of glass, pigments, textiles, paper, metal adhesives, wood preservatives and ammunition. Arsenic is also used in the hide tanning process and, to a limited extent, in pesticides, feed additives and pharmaceuticals.

16.2.5. Tobacco

People who smoke tobacco can also be exposed to the natural inorganic arsenic content of tobacco because tobacco plants essentially take up arsenic naturally present in the soil. Also, in the past, the potential for elevated arsenic exposure was much greater when tobacco plants used to be treated with lead arsenate insecticide.

16.3. Health Effects

Arsenic occurs in inorganic and organic forms. Inorganic arsenic compounds (such as those found in water) are highly toxic while organic arsenic compounds (such as those found in seafood) are less harmful to health.

16.3.1. Acute effects

The immediate symptoms of acute arsenic poisoning include vomiting, abdominal pain and diarrhoea. These are followed by numbness and tingling of the extremities, muscle cramping and death, in extreme cases.

Ingestion of 70 to 180 mg of arsenic trioxide can be fatal, but initial effects may be delayed for several hours. Acute oral ingestion of lower doses can result in these symptoms:

- Irritation of your stomach and intestines, with symptoms such as stomachache, nausea, vomiting, and diarrhea.
- Decreased production of red and white blood cells, which may cause fatigue, abnormal heart rhythm, blood-vessel damage resulting in bruising, and impaired nerve function causing a "pins and needles" sensation in your hands and feet.
- White bands, called Mees' lines, are visible in the nails.

16.3.2. Long-term effects

The first symptoms of long-term exposure to high levels of inorganic arsenic (e.g. through drinking-water and food) are usually observed in the skin, and include pigmentation changes, skin lesions and hard patches on the palms and soles of the feet (hyperkeratosis). These occur after a minimum exposure of approximately five years and may be a precursor to skin cancer. The nodular forms are encountered most frequently on the thenar and lateral borders of palms, on roots or lateral surfaces of fingers and soles, heels and toes of feet.

In addition to skin cancer, long-term exposure to arsenic may also cause cancers of the bladder and lungs. The International Agency for Research

on Cancer (IARC) has classified arsenic and arsenic compounds as carcinogenic to humans, and has also stated that arsenic in drinking-water is carcinogenic to humans.

Other adverse health effects that may be associated with long-term ingestion of inorganic arsenic include developmental effects, neurotoxicity, diabetes, pulmonary disease and cardiovascular disease. Arsenic-induced myocardial infarction, in particular, can be a significant cause of excess mortality. In China (Province of Taiwan), arsenic exposure has been linked to “blackfoot disease”, which is a severe disease of blood vessels leading to gangrene. This disease has not been observed in other parts of the world however, and it is possible that malnutrition contributes to its development.

Arsenic is also associated with adverse pregnancy outcomes and infant mortality, with impacts on child health¹, and there is some evidence of negative impacts on cognitive development.

Arsenic poisoning or arsenicosis is most often associated with drinking water contaminated with arsenic. Signs and possible impacts of arsenic exposure:

- Skin changes including patches of darkened skin and the appearance of small "corns" or "warts" on the palms, soles, and torso; these are often associated with changes in the blood vessels of the skin.
- Skin cancer
- Cancer of the liver, bladder, kidney or lungs
- Possibly diabetes, high blood pressure, reproductive disorders and lower IQ scores

Inhaling high levels of inorganic arsenic is associated with these signs and symptoms:

- Sore throat and irritated lungs
- Skin effects described above
- Circulatory and peripheral nervous disorders

Direct skin contact with high concentrations of inorganic arsenic compounds may cause the skin to become irritated, with some redness and

swelling. However, it does not appear that skin contact is likely to lead to any serious internal effects.

Much less is known about the health effects of organic arsenic. In animals, ingestion of methyl compounds can result in diarrhea, and lifetime exposure can damage the kidneys. Lifetime exposure to dimethyl compounds can damage the urinary bladder and the kidneys.

These entities have classified arsenic as a cause of cancer:

- The International Agency for Research on Cancer (IARC): inorganic arsenic is carcinogenic to humans.
- The US Department of Health and Human Services (DHHS): inorganic arsenic is known to be a human carcinogen
- US EPA: arsenic is a known human carcinogen.

16.4. Magnitude of the Problem

Arsenic contamination of groundwater is widespread and there are a number of regions where arsenic contamination of drinking-water is significant. Arsenic in Bangladesh has attracted much attention since recognition in the 1990s of its wide occurrence in well-water in that country. Since this time, significant progress has since been made and the number of people exposed to arsenic exceeding the Bangladesh drinking-water quality standard has decreased by approximately 40%. Despite these efforts, it is estimated that about 20 million and 45 million people in Bangladesh are at risk of being exposed to arsenic concentrations that are greater than the national standard of 50 µg/litre and the WHO guideline value of 10 µg/litre respectively

The symptoms and signs caused by long-term elevated exposure to inorganic arsenic differ between individuals, population groups and geographical areas. Thus, there is no universal definition of the disease caused by arsenic. This complicates the assessment of the burden on health of arsenic. Similarly, there is no method to distinguish cases of cancer caused by arsenic from cancers induced by other factors. As a result, there is no reliable estimate of the magnitude of the problem worldwide.

In 2010, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) re-evaluated the effects of arsenic on human health, taking new data

into account. JECFA concluded that for certain regions of the world where concentrations of inorganic arsenic in drinking-water exceed 50–100 µg/litre, there is some evidence of adverse effects. In other areas, where arsenic concentrations in water are elevated (10–50 µg/litre), JECFA concluded that while there is a possibility of adverse effects, these would be at a low incidence that would be difficult to detect in epidemiological studies.

16.5. Reducing Exposure

- Avoid inhalation of sawdust from arsenic-treated lumber, and never burn any treated lumber or sawdust.
- If you have an older deck or other structure made with chromated arsenicals-treated wood, applying a penetrating protective coating (such as oil- or water-based stains) on a regular basis may reduce the leaching of chemicals.
- Testing the soil underneath decks and other structures made with treated wood will indicate whether remediation of soil is needed.
- Wear gloves and other protective clothing when working with treated lumber or contaminated soil.
- Wash your hands after coming in contact with any arsenic-treated product, and supervise children in washing their hands promptly after contact.
- Test well water for arsenic, and if needed, either use bottled water for drinking and cooking or use a filter that removes arsenic.
- Take care to reduce arsenic exposure from rice, especially for children or if you consume more rice than average. In 2014 Consumer Reports investigated which varieties of rice have higher levels, cooking methods that reduce arsenic levels, and alternate grains that are naturally lower in arsenic.

16.6. Prevention and Control

The most important action in affected communities is the prevention of further exposure to arsenic by the provision of a safe water supply for drinking, food preparation and irrigation of food crops. There are a number of options to reduce levels of arsenic in drinking-water.

- Substitute high-arsenic sources, such as groundwater, with low-arsenic, microbiologically safe sources such as rain water and treated surface water. Low-arsenic water can be used for drinking, cooking

and irrigation purposes, whereas high-arsenic water can be used for other purposes such as bathing and washing clothes.

- Discriminate between high-arsenic and low-arsenic sources. For example, test water for arsenic levels and paint tube wells or hand pumps different colours. This can be an effective and low-cost means to rapidly reduce exposure to arsenic when accompanied by effective education.
- Blend low-arsenic water with higher-arsenic water to achieve an acceptable arsenic concentration level.
- Install arsenic removal systems – either centralized or domestic – and ensure the appropriate disposal of the removed arsenic. Technologies for arsenic removal include oxidation, coagulation–precipitation, absorption, ion exchange and membrane techniques. There is an increasing number of effective and low-cost options for removing arsenic from small or household supplies, though there is still limited evidence about the extent to which such systems are used effectively over sustained periods of time.

Long-term actions are also required to reduce occupational exposure from industrial processes. Education and community engagement are key factors for ensuring successful interventions. There is a need for community members to understand the risks of high arsenic exposure and the sources of arsenic exposure, including the intake of arsenic by crops (e.g. rice) from irrigation water and the intake of arsenic into food from cooking water.

High-risk populations should also be monitored for early signs of arsenic poisoning – usually skin problems.

16.7. Conclusion

Arsenic is naturally present at high levels in the groundwater of a number of countries. Arsenic is highly toxic in its inorganic form. Contaminated water used for drinking, food preparation and irrigation of food crops poses the greatest threat to public health from arsenic. Long-term exposure to arsenic from drinking-water and food can cause cancer and skin lesions. It has also been associated with developmental effects, cardiovascular disease, neurotoxicity and diabetes.

The most important action in affected communities is the prevention of further exposure to arsenic by provision of a safe water

supply. Arsenic is one of WHO's 10 chemicals of major public health concern. WHO's work to reduce arsenic exposure includes setting guideline values, reviewing evidence, and providing risk management recommendations. WHO publishes a guideline value for arsenic in its "**Guidelines for drinking-water quality**". The Guidelines are intended for use as the basis for regulation and standard setting worldwide.

World Regulatory Standards are:

- World Health Organization: provisional drinking water guideline of 10 ppb
- US EPA: Drinking water 10 µg/L (10 ppb)
- OSHA: no greater than 10 micrograms of inorganic arsenic per cubic meter of air, averaged over any eight-hour period for a 40-hour work per week
- EPA Reference Dose (RfD) for inorganic arsenic is 0.3 µg/kg/day, based on hyperpigmentation, keratosis, and possible vascular complications in humans. The RfD is an estimate that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime.

The current recommended limit of arsenic in drinking-water is 10 µg/litre, although this guideline value is designated as provisional because of measurement difficulties and the practical difficulties in removing arsenic from drinking-water. Where it is difficult to achieve the guideline value, Member States may set higher values as standards taking into account local circumstances, resources and risks from low arsenic sources that are contaminated microbiologically.

The WHO/UNICEF Joint Monitoring Programme for Water Supply and Sanitation monitors progress towards global targets on drinking water. Under the new 2030 Agenda for Sustainable Development, the proposed indicator of "safely managed drinking water services" calls for tracking the population accessing drinking water which is free of faecal contamination and priority chemical contaminants, including arsenic.

17. Lead poisoning and health

17.1. Introduction

Lead is a naturally occurring toxic metal found in the Earth's crust. Its widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world. Important sources of environmental contamination include mining, smelting, manufacturing and recycling activities, and, in some countries, the continued use of leaded paint, leaded gasoline, and leaded aviation fuel. More than three quarters of global lead consumption is for the manufacture of lead-acid batteries for motor vehicles. Lead is, however, also used in many other products, for example pigments, paints, solder, stained glass, lead crystal glassware, ammunition, ceramic glazes, jewellery, toys and in some cosmetics and traditional medicines.

Drinking water delivered through lead pipes or pipes joined with lead solder may contain lead. Much of the lead in global commerce is now obtained from recycling.

Young children are particularly vulnerable to the toxic effects of lead and can suffer profound and permanent adverse health effects, particularly affecting the development of the brain and nervous system. Lead also causes long-term harm in adults, including increased risk of high blood pressure and kidney damage. Exposure of pregnant women to high levels of lead can cause miscarriage, stillbirth, premature birth and low birth weight, as well as minor malformations.

17.2. Sources and Routes of Exposure

Lead is a common environmental pollutant. Causes of environmental contamination include industrial use of lead, such as is found in facilities that process lead-acid batteries or produce lead wire or pipes, and metal recycling and foundries. Children living near facilities that process lead, such as lead smelters, have been found to have unusually high blood lead levels. In August 2009, parents rioted in China after lead poisoning was found in nearly 2000 children living near zinc and manganese smelters. Lead exposure can occur from contact with lead in air, household dust, soil, water, and commercial

products. Leaded gasoline has also been linked to increases in lead pollution. Some research has suggested a link between leaded gasoline and crime rates. Man made lead pollution has been elevated in the air for the past 2000 years. Lead pollution in the air is entirely due to human activity (mining and smelting).

People can become exposed to lead through occupational and environmental sources. This mainly results from:

- inhalation of lead particles generated by burning materials containing lead, for example, during smelting, recycling, stripping leaded paint, and using leaded gasoline or leaded aviation fuel; and
- ingestion of lead-contaminated dust, water (from leaded pipes), and food (from lead-glazed or lead-soldered containers).

The use of some traditional cosmetics and medicines can also result in lead exposure. Young children are particularly vulnerable because they absorb 4–5 times as much ingested lead as adults from a given source. Moreover, children's innate curiosity and their age-appropriate hand-to-mouth behaviour result in their mouthing and swallowing lead-containing or lead-coated objects, such as contaminated soil or dust and flakes from decaying lead-containing paint. This route of exposure is magnified in children with pica (persistent and compulsive cravings to eat non-food items), who may, for example pick away at, and eat, leaded paint from walls, door frames and furniture. Exposure to lead-contaminated soil and dust resulting from battery recycling and mining has caused mass lead poisoning and multiple deaths in young children in Nigeria, Senegal and other countries.

Once lead enters the body, it is distributed to organs such as the brain, kidneys, liver and bones. The body stores lead in the teeth and bones where it accumulates over time. Lead stored in bone may be remobilized into the blood during pregnancy, thus exposing the fetus. Undernourished children are more susceptible to lead because their bodies absorb more lead if other nutrients, such as calcium, are lacking. Children at highest risk are the very young (including the developing fetus) and the impoverished.

17.3. Health Effects of Lead Poisoning

Lead can have serious consequences for the health of children. At high levels of exposure, lead attacks the brain and central nervous system to cause coma, convulsions and even death. Children who survive severe lead

poisoning may be left with mental retardation and behavioural disorders. At lower levels of exposure that cause no obvious symptoms, and that previously were considered safe, lead is now known to produce a spectrum of injury across multiple body systems. In particular lead affects children's brain development resulting in reduced intelligence quotient (IQ), behavioural changes such as reduced attention span and increased antisocial behaviour, and reduced educational attainment. Lead exposure also causes anaemia, hypertension, renal impairment, immunotoxicity and toxicity to the reproductive organs. The neurological and behavioural effects of lead are believed to be irreversible.

There is no known safe blood lead concentration. But it is known that, as lead exposure increases, the range and severity of symptoms and effects also increases. Even blood lead concentrations as low as 5 µg/dl, once thought to be a "safe level", may result in decreased intelligence in children, behavioural difficulties and learning problems. Encouragingly, the successful phasing out of leaded gasoline in most countries, together with other lead control measures, has resulted in a significant decline in population-level blood lead concentrations. There are now only 3 countries that continue to use leaded fuel.

17.3.1. Complications

Lead affects every one of the body's organ systems, especially the nervous system, but also the bones and teeth, the kidneys, and the cardiovascular, immune, and reproductive systems. Hearing loss and tooth decay have been linked to lead exposure, as have cataracts. Intrauterine and neonatal lead exposure promote tooth decay.

17.3.2. Kidneys

Kidney damage occurs with exposure to high levels of lead, and evidence suggests that lower levels can damage kidneys as well. The toxic effect of lead causes nephropathy and may cause Fanconi syndrome, in which the proximal tubular function of the kidney is impaired. Long-term exposure at levels lower than those that cause lead nephropathy have also been reported as nephrotoxic in patients from developed countries that had chronic kidney disease or were at risk because of hypertension or diabetes mellitus. Lead

poisoning inhibits excretion of the waste product urate and causes a predisposition for gout, in which urate builds up. This condition is known as **saturnine gout**.

17.3.3. Cardiovascular system

Evidence suggests lead exposure is associated with high blood pressure, and studies have also found connections between lead exposure and coronary heart disease, heart rate variability, and death from stroke, but this evidence is more limited. People who have been exposed to higher concentrations of lead may be at a higher risk for cardiac autonomic dysfunction on days when ozone and fine particles are higher.

17.3.4. Reproductive system

Lead affects both the male and female reproductive systems. In men, when blood lead levels exceed 40 $\mu\text{g}/\text{dL}$, sperm count is reduced and changes occur in volume of sperm, their motility, and their morphology. A pregnant woman's elevated blood lead level can lead to miscarriage, prematurity, low birth weight, and problems with development during childhood. Lead is able to pass through the placenta and into breast milk, and blood lead levels in mothers and infants are usually similar. A fetus may be poisoned *in utero* if lead from the mother's bones is subsequently mobilized by the changes in metabolism due to pregnancy; increased calcium intake in pregnancy may help mitigate this phenomenon.

17.3.5. Nervous system

The brains of adults who were exposed to lead as children show decreased volume, especially in the prefrontal cortex, on MRI. Areas of volume loss are shown in color over a template of a normal brain.

Lead affects the peripheral nervous system (especially motor nerves) and the central nervous system. Peripheral nervous system effects are more prominent in adults and central nervous system effects are more prominent in children. Lead causes the axons of nerve cells to degenerate and lose their myelin coats.

Lead exposure in young children has been linked to learning disabilities, and children with blood lead concentrations greater than 10 $\mu\text{g}/\text{dL}$ are in danger of developmental disabilities. Increased blood lead level in children has been correlated with decreases in intelligence, nonverbal reasoning, short-term memory, attention, reading and arithmetic ability, fine

motor skills, emotional regulation, and social engagement. The effect of lead on children's cognitive abilities takes place at very low levels. There is apparently no lower threshold to the dose-response relationship (unlike other heavy metals such as mercury). Reduced academic performance has been associated with lead exposure even at blood lead levels lower than 5 $\mu\text{g}/\text{dL}$. Blood lead levels below 10 $\mu\text{g}/\text{dL}$ have been reported to be associated with lower IQ and behavior problems such as aggression, in proportion with blood lead levels. Between the blood lead levels of 5 and 35 $\mu\text{g}/\text{dL}$, an IQ decrease of 2–4 points for each $\mu\text{g}/\text{dL}$ increase is reported in children. However, studies that show associations between low-level lead exposure and health effects in children may be affected by confounding and overestimate the effects of low-level lead exposure.

High blood lead levels in adults are also associated with decreases in cognitive performance and with psychiatric symptoms such as depression and anxiety. It was found in a large group of current and former inorganic lead workers in Korea that blood lead levels in the range of 20–50 $\mu\text{g}/\text{dL}$ were correlated with neuro-cognitive defects. Increases in blood lead levels from about 50 to about 100 $\mu\text{g}/\text{dL}$ in adults have been found to be associated with persistent, and possibly permanent, impairment of central nervous system function.

Lead exposure in children is also correlated with neuropsychiatric disorders such as attention deficit hyperactivity disorder and anti-social behaviour. Elevated lead levels in children are correlated with higher scores on aggression and delinquency measures. A correlation has also been found between prenatal and early childhood lead exposure and violent crime in adulthood. Countries with the highest air lead levels have also been found to have the highest murder rates, after adjusting for confounding factors. A May 2000 study by economic consultant Rick Nevin theorizes that lead exposure explains 65% to 90% of the variation in violent crime rates in the US. A 2007 paper by the same author claims to show a strong association between preschool blood lead and subsequent crime rate trends over several decades across nine countries. Lead exposure in childhood appears to increase school suspensions and juvenile detention among boys. It is believed that the U.S. ban on lead paint in buildings in the late 1970s, as well as the phaseout of leaded gasoline in the 1970s and 1980s, partially helped contribute to the decline of violent crime in the United States since the early 1990s.

17.4. Symptoms

Symptoms of lead poisoning are varied. They may affect many parts of the body. Most of the time, lead poisoning builds up slowly. It follows repeated exposures to small quantities of lead.

Lead toxicity is rare after a single exposure or ingestion of lead. Signs of repeated lead exposure include:

- abdominal pain
- abdominal cramps
- aggressive behavior
- constipation
- sleep problems
- headaches
- irritability
- loss of developmental skills in children
- loss of appetite
- fatigue
- high blood pressure
- numbness or tingling in the extremities
- memory loss
- anemia
- kidney dysfunction

Since a child's brain is still developing, lead can lead to intellectual disability. Symptoms may include:

- behavior problems
- low IQ
- poor grades at school
- problems with hearing
- short- and long-term learning difficulties
- growth delays

A high, toxic dose of lead poisoning may result in emergency symptoms. These include:

- severe abdominal pain and cramping
- vomiting
- muscle weakness
- stumbling when walking
- seizures
- coma
- encephalopathy, which manifests as confusion, coma, and seizures

If someone has symptoms of severe lead exposure, call emergency medical services. Be sure to have the following information ready to tell the emergency operator:

- the person's age
- their weight
- the source of the poisoning
- the amount swallowed
- the time the poisoning occurred

In non-emergency situations, call poison control to discuss lead poisoning symptoms. They will let you speak with an expert.

17.5. Causes of Lead Poisoning

Lead poisoning occurs when lead is ingested. Breathing in dust that contains lead can also cause it. You cannot smell or taste lead and it's not visible to the naked eye. In the United States, lead used to be common in house paint and gasoline. These products are not produced with lead any longer. However, lead is still present everywhere. It is especially found in older houses.

Common sources of lead include:

- house paint made before 1978
- toys and household items painted before 1976

- toys made and painted outside the United States
- bullets, curtain weights, and fishing sinkers made of lead
- pipes and sink faucets, which can contaminate drinking water
- soil polluted by car exhaust or chipping house paint
- paint sets and art supplies
- jewelry, pottery, and lead figures
- storage batteries
- kohl or kajal eyeliners
- some traditional ethnic medicines

17.6. How is Lead Poisoning Diagnosed?

Lead poisoning is diagnosed with a blood lead test. This test is performed on a standard blood sample. Lead is common in the environment. The National Institute of Environmental Health Sciences reports that no amount of lead in the blood is safe. It is known that levels as low as 5 micrograms per deciliter can be associated with health problems in children. Additional tests could include blood tests to look at the amount of iron storing cells in the blood, X-rays, and possibly a bone marrow biopsy.

17.7. Treatment

The first step of treatment is to locate and remove the source of the lead. Keep children away from the source. If it cannot be removed, it should be sealed. Call your local health department for information on how to remove lead. They can also help you reduce the likelihood of lead exposure. In more severe cases, a procedure known as chelation therapy can be used. This treatment binds to lead that has accumulated in your body. The lead is then excreted in your urine. Activated charcoal can be used to bind the lead in the gastrointestinal tract and encourage elimination via defecation. A chemical called EDTA may also be used. Even with treatment, it can be hard to reverse the effects of chronic exposure.

17.8. Prevention

Simple steps can help you prevent lead poisoning. These include:

- Avoid or throw away painted toys and canned goods from foreign countries.
- Keep your home free from dust.
- Use only cold water to prepare foods and drinks.
- Make sure everyone washes their hands before eating.
- Test your water for lead. If lead levels are high, use a filtering device or drink bottled water.
- Clean faucets and aerators regularly.
- Wash children's toys and bottles regularly.
- Teach your children to wash their hands after playing.
- Make sure any contractor doing work in your house is certified in lead control.
- Use lead-free paint in your home.
- Take young children for blood lead level screening at their pediatrician's office. This is usually done around 1 to 2 years of age.
- Avoid areas where lead-based paint may have been used.

17.9. Conclusion

The Institute for Health Metrics and Evaluation (IHME) has estimated that in 2013 lead exposure accounted for 853 000 deaths due to long-term effects on health, with the highest burden in low and middle income countries. IHME also estimated that lead exposure accounted for 9.3% of the global burden of idiopathic intellectual disability, 4% of the global burden of ischaemic heart disease and 6.6% of the global burden of stroke.

WHO has identified lead as 1 of 10 chemicals of major public health concern, needing action by Member States to protect the health of workers, children and women of reproductive age. WHO has made available through its website a range of information on lead, including information for policy makers, technical guidance and advocacy materials. WHO is currently developing guidelines on the prevention and management of lead poisoning, which will provide policy-makers, public health authorities and health professionals with evidence-based guidance

on the measures that they can take to protect the health of children and adults from lead exposure.

Since leaded paint is a continuing source of exposure in many countries, WHO has joined with the United Nations Environment Programme to form the Global Alliance to Eliminate Lead Paint. This is a cooperative initiative to focus and catalyse efforts to achieve international goals to prevent children's exposure to lead from leaded paints and to minimize occupational exposures to such paint. Its broad objective is to promote a phase-out of the manufacture and sale of paints containing lead and eventually eliminate the risks that such paints pose.

The Global Alliance to Eliminate Lead Paint is an important means of contributing to the implementation of paragraph 57 of the "**Plan of Implementation**" of the World Summit on Sustainable Development and to resolution II/4B of the Strategic Approach to International Chemicals Management (SAICM), which both concern the phasing of lead paint.

18. Health Effects of Exposures to Mercury

18.1. Introduction

Mercury exists in various forms: elemental (or metallic) and inorganic (to which people may be exposed through their occupation); and organic (e.g., methylmercury, to which people may be exposed through their diet). These forms of mercury differ in their degree of toxicity and in their effects on the nervous, digestive and immune systems, and on lungs, kidneys, skin and eyes.

Mercury occurs naturally in the earth's crust. It is released into the environment from volcanic activity, weathering of rocks and as a result of human activity. Human activity is the main cause of mercury releases, particularly coal-fired power stations, residential coal burning for heating and cooking, industrial processes, waste incinerators and as a result of mining for mercury, gold and other metals.

Once in the environment, mercury can be transformed by bacteria into methylmercury. Methylmercury then bioaccumulates (bioaccumulation occurs when an organism contains higher concentrations of the substance than do the surroundings) in fish and shellfish. Methylmercury also biomagnifies. For example, large predatory fish are more likely to have high levels of mercury as a result of eating many smaller fish that have acquired mercury through ingestion of plankton.

People may be exposed to mercury in any of its forms under different circumstances. However, exposure mainly occurs through consumption of fish and shellfish contaminated with methylmercury and through worker inhalation of elemental mercury vapours during industrial processes. Cooking does not eliminate mercury.

Man-made mercury sources. While some mercury emissions occur naturally (such as those released from volcanic eruptions), half of the world's mercury emissions come from man-made sources. According to EarthShare member organization **Natural Resources Defense Council** (NRDC), coal-fired power plants remain one of the largest mercury pollutants, emitting around 33 tons of mercury every year and contributing to half of all mercury emissions. Other sources include chemical manufacturing plants (which emit

another 10–12 tons of mercury per year) and automobile scraps that are melted down for recycling.

Mercury in the environment. According to the **U.S. Environmental Protection Agency (EPA)**, once mercury is released into the air, it can be carried great distances. Once airborne, mercury vapor can be changed into other forms of mercury and can be further transported to water or soil via rain or snow. Some forms of mercury can also be released into water and soil through pesticides and fungicides.

Once in the water, microorganisms like phytoplankton and fungi convert and release inorganic mercury to methylmercury. At this stage, the mercury can enter the food chain. Small fish feed upon organisms and plants containing methylmercury, bigger fish come along and eat the smaller fish. In a process called **bioaccumulation**, mercury builds up in the food chain over time. This means that bigger, older fish tend to carry the most methylmercury, as they've consumed many smaller fish containing mercury.

Not surprisingly, methylmercury is transferred to people as we catch and consume fish from contaminated waters. Eating fish is the main source of human exposure to mercury, according to the **EPA**.

18.2. Exposure to Mercury

All humans are exposed to some level of mercury. Most people are exposed to low levels of mercury, often through chronic exposure (continuous or intermittent long term contact). However, some people are exposed to high levels of mercury, including acute exposure (exposure occurring over a short period of time, often less than a day). An example of acute exposure would be mercury exposure due to an industrial accident.

Factors that determine whether health effects occur and their severity include:

- the type of mercury concerned;
- the dose;
- the age or developmental stage of the person exposed (the foetus is most susceptible);
- the duration of exposure;
- the route of exposure (inhalation, ingestion or dermal contact).

Generally, two groups are more sensitive to the effects of mercury. Foetuses are most susceptible to developmental effects due to mercury. Methylmercury exposure in the womb can result from a mother's consumption of fish and shellfish. It can adversely affect a baby's growing brain and nervous system. The primary health effect of methylmercury is impaired neurological development. Therefore, cognitive thinking, memory, attention, language, and fine motor and visual spatial skills may be affected in children who were exposed to methylmercury as foetuses.

The second group is people who are regularly exposed (chronic exposure) to high levels of mercury (such as populations that rely on subsistence fishing or people who are occupationally exposed). Among selected subsistence fishing populations, between 1.5/1000 and 17/1000 children showed cognitive impairment (mild mental retardation) caused by the consumption of fish containing mercury. These included populations in Brazil, Canada, China, Columbia and Greenland.

A significant example of mercury exposure affecting public health occurred in Minamata, Japan, between 1932 and 1968, where a factory producing acetic acid discharged waste liquid into Minamata Bay. The discharge included high concentrations of methylmercury. The bay was rich in fish and shellfish, providing the main livelihood for local residents and fishermen from other areas.

For many years, no one realised that the fish were contaminated with mercury, and that it was causing a strange disease in the local community and in other districts. At least 50 000 people were affected to some extent and more than 2000 cases of Minamata disease were certified. According to the US Food and Drug Administration (FDA), the risk from mercury by eating fish and shellfish is not a health concern for most people. However, certain seafood might contain levels of mercury that may cause harm to an unborn baby (and especially its brain development and nervous system). In a young child, high levels of mercury can interfere with the development of the nervous system.

The FDA provides three recommendations for young children, pregnant women, and women of child-bearing age:

1. Do not eat shark, swordfish, king mackerel, or tilefish (Gulf of Mexico) because they might contain high levels of mercury.
2. Eat up to 12 ounces (2 average meals) a week of a variety of fish and shellfish that are lower in mercury. Five of the most commonly eaten fish and shellfish that are low in mercury are: shrimp, canned light tuna, salmon, pollock, and catfish. Another commonly eaten fish, albacore or big eye ("white") tuna depending on its origin might have more mercury than canned light tuna. So, when choosing your two meals of fish and shellfish, it is recommended that you should not eat more than up to 6 ounces (one average meal) of albacore tuna per week.
3. Check local advisories about the safety of fish caught by family and friends in your local lakes, rivers, and coastal areas. If no advice is available, eat up to 6 ounces (one average meal) per week of fish you catch from local waters, but consume no other fish during that week.

18.3. Health Effects of Mercury Exposure

The three most common forms of mercury (elemental, inorganic and methylmercury) can all produce adverse health effects at sufficiently high doses. The U.S. Environmental Protection Agency (EPA) has determined that eating mercury-contaminated fish is the primary route of exposure to mercury for most people.

The EPA also concluded that most Americans are not at risk from mercury exposure. Therefore, most people can continue to look to fish as a healthy, low-fat source of protein and other nutrients. However, pregnant women, women who may become pregnant within the next several years, children less than six years old and people who consume unusually large quantities of freshwater sport fish, shark, or swordfish, may be harmed by mercury.

Inhaling elemental mercury, the vapor given off when mercury is heated, can also be dangerous. For example, some people burn mercury in candles as part of rituals, a practice health professionals highly discourage.

Mercury can damage human health because it is toxic to the nervous system — the brain and spinal cord — particularly the developing nervous system of a fetus or young child. And it doesn't take much mercury. One million average-size northern pike from northern Minnesota lakes would contain just a pound of mercury altogether, yet the concentration in each fish would be high enough to call for limits on eating them.

Mercury's effects can be very subtle. Adults who have been exposed to too much methylmercury might begin to experience trembling hands and numbness or tingling in their lips, tongues, fingers or toes. These effects can begin long after the exposure occurred. At higher exposures, walking could be affected, as well as vision, speech and hearing. In sufficient quantities, methylmercury can be fatal.

The greatest risk, however, is for fetuses and young children because their nervous systems are still developing. They are four or five times more sensitive to mercury than adults. Damage occurring before birth or in infancy can cause a child to be late in beginning to walk and talk and may cause lifelong learning problems. Unborn children can be seriously affected even though the methylmercury causes no symptoms in their mothers.

Elemental and methylmercury are toxic to the central and peripheral nervous systems. The inhalation of mercury vapour can produce harmful effects on the nervous, digestive and immune systems, lungs and kidneys, and may be fatal. The inorganic salts of mercury are corrosive to the skin, eyes and gastrointestinal tract, and may induce kidney toxicity if ingested.

Neurological and behavioural disorders may be observed after inhalation, ingestion or dermal exposure of different mercury compounds. Symptoms include tremors, insomnia, memory loss, neuromuscular effects, headaches and cognitive and motor dysfunction. Mild, subclinical signs of central nervous system toxicity can be seen in workers exposed to an elemental mercury level in the air of 20 $\mu\text{g}/\text{m}^3$ or more for several years. Kidney effects have been reported, ranging from increased protein in the urine to kidney failure.

Methylmercury affects more than 630,000 newborns each year, interfering with brain and nervous system development. Deficits in cognitive thinking, memory, language, motor and visual spatial skills have all been

linked to children exposed to methylmercury in the womb. It's because of these risks that women are advised to abstain from eating fish while they are pregnant or nursing.

Mercury poisoning is also a possible health risk to those who consume mercury-laden fish. Immediate symptoms of mercury poisoning include:

- Impairment of peripheral vision
- A “pins and needles” sensation in the hands, feet and around the mouth
- Lack of coordination
- Impairment of speech, hearing and walking
- Muscle weakness

18.4. How to Reduce Human Exposure from Mercury Sources

There are several ways to prevent adverse health effects, including promoting clean energy, stopping the use of mercury in gold mining, eliminating the mining of mercury and phasing out non-essential mercury-containing products.

Promote the use of clean energy sources that do not burn coal. Burning coal for power and heat is a major source of mercury. Coal contains mercury and other hazardous air pollutants that are emitted when the coal is burned in coal-fired power plants, industrial boilers and household stoves.

Eliminate mercury mining, and use of mercury in gold extraction and other industrial processes. Mercury is an element that cannot be destroyed; therefore, mercury already in use can be recycled for other essential uses, with no further need for mercury mining. Mercury used in artisanal and small-scale gold mining is particularly hazardous, and health effects on vulnerable populations are significant. Non-mercury (non-cyanide) gold-extraction techniques need to be promoted and implemented, and where mercury is still used, safer work practices need to be employed to prevent exposure.

Phase out use of non-essential mercury-containing products and implement safe handling, use and disposal of remaining mercury-containing products. Mercury is contained in many products, including:

- batteries
- measuring devices, such as thermometers and barometers
- electric switches and relays in equipment
- lamps (including some types of light bulbs)
- dental amalgam (for dental fillings)
- skin-lightening products and other cosmetics
- pharmaceuticals.

A range of actions are being taken to reduce mercury levels in products, or to phase out mercury-containing products. In health care, mercury-containing thermometers and sphygmomanometers are being replaced by alternative devices.

Dental amalgam is used in almost all countries. A 2009 WHO expert consultation concluded that a global near-term ban on amalgam would be problematic for public health and the dental health sector, but a phase down should be pursued by promoting disease prevention and alternatives to amalgam; research and development of cost-effective alternatives; education of dental professionals and the raising of public awareness.

Inorganic mercury is added to some skin-lightening products in significant amounts. Many countries have banned mercury-containing skin-lightening products because they are hazardous to human health.

18.5. Conclusion

Mercury is a naturally occurring element that is found in air, water and soil. Exposure to mercury – even small amounts – may cause serious health problems, and is a threat to the development of the child *in utero* and early in life. Mercury may have toxic effects on the nervous, digestive and immune systems, and on lungs, kidneys, skin and eyes.

Research suggests that selenium content in fish is protective against the toxic effects of methylmercury content. Fish with higher ratios of

selenium to methylmercury (Se:Hg) are better to eat since the selenium binds to the methylmercury allowing it to pass through the body un-absorbed.

In 2012 the **European Food Safety Authority** (EFSA) reported on chemical contaminants they found in the food of over 20 European countries. They established that fish meat and fish products were primarily responsible for methylmercury in the diet of all age classes. Particularly implicated were swordfish, tuna, cod, pike, whiting and hake. The EFSA recommend a tolerable weekly intake for methylmercury of 1.3 µg/kg body weight.

Mercury is considered by WHO as one of the top ten chemicals or groups of chemicals of major public health concern. People are mainly exposed to methylmercury, an organic compound, when they eat fish and shellfish that contain the compound. Methylmercury is very different to ethylmercury. Ethylmercury is used as a preservative in some vaccines and does not pose a health risk. WHO publishes evidence about the health impacts of the different forms of mercury, guidance on identifying populations at risk from mercury exposure, tools to reduce mercury exposure, and guidance on the replacement of mercury-containing thermometers and blood pressure measuring devices in health care. WHO leads projects to promote the sound management and disposal of health-care waste and has facilitated the development of an affordable, validated, non-mercury-containing blood pressure measuring device.

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