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Kofi Asante-Duah

Public Health Risk Assessment for Human Exposure to Chemicals

Second Edition

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To: Dad—George Kwabena Duah
To: Mom—Alice Adwoa Twumwaa
To: Kojo Asante-Duah (a.k.a., K.J.)
To: Kwabena Asante Duah (a.k.a., Daddy-K./Kobby)
To: Adwoa Twumwaa Asante-Duah (a.k.a., Obaa-Sima A.T./Naana)
To: My Extraordinary Families (of Abaam, Kade, and Nkwantanan)
—and all the Off-springs & Off-shoots

&

To the Everlasting and Ever-Loving Memories of:
Daddy, George Kwabena Duah (a.k.a., GKD/Agya Duah)
Grandma, Nana Martha Adwoa Oforiwaa & Grandpa
Grandma, Nana Abena Nketia Owusua & Grandpa
Atta Kakra (a.k.a., Emmanuel Asare Duah)
Atta Panin (a.k.a., Ebenezer Asare Duah)
Osagyefo Dr. Kwame Nkrumah @GH

Preface

Risk to human health as a consequence of toxic materials found in modern societies is a matter of grave concern to the world community. What is more, risks to humans that arise from chemical exposures from a multiplicity of sources are a complex issue with worldwide implications. The effective management of human exposure to a variety of chemicals present in various sectors of society has therefore become a very important public health policy issue that will remain a growing social challenge for years to come. In fact, with a reasonable control and containment of most infectious conditions of the past millennium having been realized in most developed countries, and with the accompanying increase in life expectancies, much more attention seems to have shifted to degenerative health problems typically attributable to environmental or 'social' chemicals so very often encountered in modern societies. Many of the degenerative health conditions have indeed been linked to thousands of chemicals regularly encountered in human living and occupational/work environments. It is important, therefore, that human health risk assessments are carried out on a consistent basis—in order to be able to determine the potential impacts of the target chemicals on public health. Overall, risk assessment promises a systematic way for developing appropriate strategies to aid public health risk policy decisions in the arena of human exposures to chemicals.

Risk assessment generally serves as a tool that can be used to organize, structure, and compile scientific information to help identify existing hazardous situations or problems, anticipate potential problems, establish priorities, and provide a basis for regulatory controls and/or corrective actions. A key underlying principle of public health risk assessment is that some risks are somehow tolerable—a reasonable and even sensible view, considering the fact that nothing is wholly safe per se. In fact, whereas human exposures to large amounts of a toxic substance may be of major concern, exposures of rather limited extent may be trivial and hence should not necessarily be a cause for alarm. In order to be able to make a credible decision on the cut-off between what really constitutes a 'dangerous dose' and a 'safe dose', systematic scientific tools—such as those afforded by risk assessment—may be utilized. In this regard, therefore, risk assessment seems to represent an important

foundation in the development of effectual public health risk management strategies and policies.

This book provides a concise, yet comprehensive overview of the many facets/aspects of human health risk assessments in relation to chemical exposure problems. It presents some very important tools and methodologies that can be used to address chemical exposure and public health risk management problems in a consistent, efficient, and cost-effective manner. On the whole, the book represents a collection and synthesis of the principal elements of the risk assessment process that may be used to more effectively address issues pertaining to human exposures to chemicals found in modern societies. This also includes an elaboration of pertinent risk assessment concepts and techniques/methodologies for performing human health risk assessments. A number of illustrative example problems are interspersed throughout the book, in order to help present the book in an easy-to-follow, pragmatic manner.

Meanwhile, it is noteworthy that even though the main focus of this title is on risk assessment of the potential human health effects associated with chemical exposures, the same principles may be extrapolated to deal with other forms of human exposure problems (such as exposures to radionuclides and pathogens). Thus, the chemical risk assessment framework may be adapted and applied to human exposures to other agents—albeit many unique issues may have to be addressed for exposures to the new hazard/agent under consideration. In fact, the subject matter of this book can generally be used to aid in the resolution of a variety of environmental contamination and public health risk management problems.

On the whole, this book should serve as a useful reference for many professionals encountering risk assessment in relation to environmental contamination and public health risk management programs; it offers an understanding of the scientific basis of risk assessment and its applications to public health policy decisions. The specific intended audience includes public and occupational health practitioners and other public health and environmental health professionals, public policy analysts, environmental consulting professionals, consumer product manufacturers, environmental attorneys, environmental and health regulatory agencies, environmental and public health NGOs, and a miscellany of health, environmental, and consumer advocacy interest groups. The book is also expected to serve as a useful *educational/training resource for both students and professionals in the health-related and environmental fields*—particularly those who have to deal with human exposures to chemicals, public health risk assessment issues, and/or environmental health management problems. Written for both the novice and the experienced, the subject matter of this book is an attempt at offering a simplified and systematic presentation of public health risk assessment methods and application tools—all these facilitated by a design/layout that will carefully navigate the user through the major processes involved.

Finally, a key objective in preparing this revised edition to the book has been to, insofar as practicable, incorporate new key developments and/or updates in the field since the previous version was last published. Another notable feature of the revised edition is the sectional re-organization that has been carried out for some

topics—all meant to help with the overall flow of the presentations, but especially to facilitate a more holistic learning process/experience afforded by this book. All in all, the book is organized into five parts—consisting of 15 chapters and a set of 5 appendices, together with a bibliographical listing. It is the hope of the author that the five-part presentation offered by this title will provide adequate guidance and direction for the successful completion of public health risk assessment programs that are to be designed for any type of chemical exposure problem, and at any geographical location. The structured presentation should also help with any efforts to develop effectual classroom curricula for teaching purposes. Ultimately, the systematic protocols presented in this volume should indeed aid many a public health and related environmental professional to formulate and manage chemical exposure and associated problems more efficiently.

Washington, DC
8 August 2016

Kofi Asante-Duah

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The support of the Publishing, Editorial, and Production staff at Springer in helping to bring this book project to a successful conclusion is very much appreciated. I also wish to thank every author whose work is cited in this book—for having provided some pioneering work to build on.

Finally, it should be acknowledged that this book benefited greatly from review comments of several anonymous individuals, as well as from discussions with a number of professional colleagues. Any shortcomings that remain are, however, the sole responsibility of the author.

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Part I
Problem Diagnosis: A General Overview of the Origins and Nature of Chemical Exposure Problems

This part of the book encompasses the following three specific chapters:

- Chapter 1, *Introduction*, presents a general background discussion on the wide-ranging sources/origins of environmental contamination and chemical exposure problems often encountered in practice, as well as elaborate on the likely implications/consequences of such types of problem situations. This chapter also provides a broad overview on the general types of issues that may have to be addressed in order to establish an effective risk management and/or corrective action program for chemical exposure problems.
- Chapter 2, *Anatomical and Physiological Perspectives on Human Exposure to Chemicals*, looks at the major human contact sites, target organs, and exposure scenarios that can be expected to become key players in the assessment of human exposure to, and response from, chemical hazards—all the while recognizing that several characteristics of the target chemicals of concern/interest, as well as the human contact sites, will typically provide an indication of the critical attributes of a given exposure.
- Chapter 3, *Archetypical Chemical Exposure Problems*, apprises the typically significant exposure scenarios that can be expected to become key players in the assessment of human exposure to, and response from, chemical hazards; it goes on to provide a general framework that may be used to guide the formulation of realistic exposure scenarios, as necessary to generate credible risk assessments.

Chapter 1
Introduction

In the landmark book—*Silent Spring*—from the early 1960s, Rachel Carson wrote: "For the first time in the history of the world, every human being is now subjected to contact with dangerous chemicals, from the moment of conception until death" (Carson 1962, 1994). What is more, this statement of some more than five decades ago is not about to change, given our dependency—maybe even obsession—with a so-called 'modern way of life'. Indeed, in everyday living, peoples around the world—directly or indirectly—are exposed to myriad sources and cocktails of chemical hazards. Ultimately, these endemic chemical exposure problems may pose significant risks to global populations because of the potential health effects; for instance, pesticides are believed to have accounted for some of the most advanced and persistent cases of variant human chemical sensitivity that became known to some clinicians and physicians in the fairly recent past (Ashford and Miller 1998; Randolph 1962, 1987). Risks to human health as a result of exposure to toxic materials present or introduced into our living and work environments are, therefore, a matter of grave concern to modern societies. To borrow again from Rachel Carson's *Silent Spring*, 'if we are going to live so intimately with these chemicals—eating and drinking them, taking them into the very marrow of our bones'—then at the very least, we should be able to determine the risks that we are exposed to, as well as know how to manage such risks, in order to ensure a worthwhile quality to our lives (Carson 1962, 1994).

In fact, it has become overwhelmingly apparent that many of the degenerative health conditions seen in modern societies may be linked to the innumerable chemicals regularly encountered in human living and occupational/work environments. What is more, with a reasonable control and containment of most infectious conditions and diseases of the past millennium having been realized in most developed countries, and with the consequential increase in life expectancies, much more attention seem to have shifted to degenerative health problems typically attributable to environmental or 'social' chemicals so very often encountered in modern societies. It is important, therefore, that human health risk assessments are undertaken on a consistent basis—in order to reasonably ascertain the potential

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impacts of the target chemicals of concern on public health. Overall, risk assessment promises a systematic way for developing appropriate strategies to aid public health risk policy decisions in the arena of human exposures to chemicals.

This book focuses on the holistic application of effectual risk assessment concepts and principles to support responsible and credible public health risk management programs as relates to chemical exposure problems. On the whole, it offers a good understanding of the scientific basis of the risk assessment paradigm and attributes, as well as its applications to public health policy decisions for chemical exposure situations.

1.1 Chemical Origins: Coming to Terms with the Several Chemicals in Modern Society

As a quintessential part of the story often told about chemicals prevalent in modern societies, synthetic pesticides became the symbols of progress during the postwar years and provided an unprecedented level of control over one type of environmental risks—more specifically, pest-related risks. As a notable example, the discovery of the insecticidal properties of dichlorodiphenyl trichloroethane [DDT] in 1939 by the Swiss scientist and Nobel Prize recipient, Paul Müller, began the modern chemical industrial revolution—and which then became a turning point in the shaping of both public health and agricultural history. In fact, as an important specific example, when the World Health Organization (WHO) was established in 1945, it relied primarily on DDT to control mosquito-borne diseases, especially malaria; the results of the WHO efforts were considered extraordinary for much of that period of time. However, as subsequently became quite apparent, these benefits were not realized without some significant (even if intangible) costs; among other things, growing mosquito-resistance to DDT necessitated the use of higher application rates, as well as the development and use of other related chlorinated compounds with similar attributes/concerns. Ultimately, DDT and its analogs became associated with significant environmental impacts globally—most notably, the apparent decline of certain avian species due to the chemical effects on egg shell integrity, etc. Indeed, to affirm how serious a problem the likely impacts generally had been, it is noteworthy that even in the far removed Arctic regions, it has been established that contamination of the arctic aquatic food-chain by organochlorine compounds and other anthropogenic chemicals has occurred (see, *e.g.*, Barrie et al. 1992; Dewailly et al. 1993; Lockhart et al. 1992; Muir et al. 1992; Thomas et al. 1992).

Now, making what seems like quantum leaps into the future with respect to the significant advances in the germane scientific fields associated with the chemical exposure problems of yesterdays does not appear to have insulated most biological organisms from the potential chemical impact or vulnerability problems seen today. In fact, in contemporary societies, it appears that there is no escape from potential

chemical exposure problems in any part of the world—especially with regards to those resulting from possible environmental contamination, and also from the usage of a wide variety of consumer products. After all, chemicals seem to have become an integral part of the global economy—providing key building blocks for the many products that seem to have proven beneficial to much of society. Still, depending on their use (or misuse), chemicals may have significantly harmful impacts on human health and the environment; for instance, evidence seems to be mounting about the believe that some chemicals found in everyday consumer products (*e.g.*, some plastic bottles and containers; liners of metal food cans; detergents; flame retardants; foods; toys; cosmetics; pesticides; etc.) may disrupt the endocrine system and affect the development of children and sensitive ecological species.

Broadly speaking, the key environmental chemicals of greatest concern are believed to be anthropogenic organic compounds. These typically include pesticides—*e.g.*, lindane, chlordane, endrin, dieldrin, toxaphene, and dichlorodiphenyl trichloroethane [DDT]; industrial compounds—*e.g.*, solvents such as trichloroethylene (or, trichloroethene) [TCE] and fuel products derived from petroleum hydrocarbons; and byproducts of various industrial processes—*e.g.*, hexachlorobenzene [HCB], polychlorinated biphenyls [PCBs], polychlorinated dibenzodioxins (or, polychlorodibenzo-*p*-dioxins) [PCDDs], and polychlorinated dibenzofurans (or, polychlorodibenzofurans) [PCDFs] (see, *e.g.*, Dewailly et al. 1993, 1996; Walker 2008). Many industries also produce huge quantities of highly toxic waste byproducts that include cyanide ions, acids, bases, heavy metals, oils, dyes, and organic solvents (Table 1.1). Further yet, other rather unsuspecting sources of environmental contaminants are beginning to add to the multitude of chemical exposure problems that contemporary societies face. For instance, low levels of reproductive hormones, birth control pills, steroids, antibiotics, analgesics, antidepressants, antineoplastics, parasiticides, and numerous other prescription and nonprescription drugs (in relation to both human medicinal and veterinary products), as well as some of their metabolites, have been detected in various water bodies around the world in recent times. In fact, a number of scientists and regulatory agencies around the world have come to recognize/acknowledge pharmaceuticals to be an emerging environmental problem of significant concern—culminating in the development of regulatory frameworks to address this issue; within such framework, it has been determined that approximately 10% of pharmaceutical products currently in use may potentially pose significant environmental risks (Küster and Adler 2014). At any rate, pharmaceuticals have probably entered, and been present in our environments since their use began (*i.e.*, for well over a century now)—albeit it has only recently been recognized as a significant environmental issue. What is more, given the rather continual and diffuse nature of pharmaceutical releases into the environment (usually through various point and nonpoint sources, and typically via municipal/domestic waste streams and/or sewage systems), trace levels of pharmaceuticals in the environment are not unexpected in most locales. Along with the pharmaceuticals, products used in everyday life (such as food additives, cosmetics, fragrances, plasticizers, cleaners, detergents, disinfectants, insect repellants, pesticides, fire retardants, etc.) are also turning up in

Table 1.1 Examples of typical potentially hazardous waste-streams from selected industrial sectors.

Sector/source	Typical hazardous waste-stream
Agricultural and food production	Acids and alkalis; fertilizers (e.g., nitrates); herbicides (e.g., dioxins); insecticides; unused pesticides (e.g., aldicarb, aldrin, DDT, dieldrin, parathion, toxaphene)
Airports	Hydraulic fluids; oils
Auto/vehicle servicing	Acids and alkalis; heavy metals; lead-acid batteries (e.g., cadmium, lead, nickel); solvents; waste oils
Chemical/pharmaceuticals	Acids and alkalis; biocide wastes; cyanide wastes; heavy metals (e.g., arsenic, mercury); infectious and laboratory wastes; organic residues; PCBs; solvents
Domestic	Acids and alkalis; dry-cell batteries (e.g., cadmium, mercury, zinc); heavy metals; insecticides; solvents (e.g., ethanol, kerosene)
Dry-cleaning/laundries	Detergents (e.g., boron, phosphates); dry-cleaning filtration residues; halogenated solvents
Educational/research institutions	Acids and alkalis; ignitable wastes; reactives (e.g., chromic acid, cyanides; hypochlorites, organic peroxides; perchlorates, sulfides); solvents
Electrical transformers	PCBs
Equipment repair	Acids and alkalis; ignitable wastes; solvents
Leather tanning	Inorganic chemicals (e.g., chromium, lead); solvents
Machinery manufacturing	Acids and alkalis; cyanide wastes; heavy metals (e.g., cadmium, lead); oils; solvents
Medical/health services	Laboratory wastes; pathogenic/infectious wastes; radionuclides; solvents
Metal treating/manufacture	Acids and alkalis; cyanide wastes; heavy metals (e.g., antimony, arsenic, cadmium, cobalt); ignitable wastes; reactives; solvents (e.g., toluene, xylenes)
Military training grounds	Heavy metals
Mineral processing/extraction	High-volume/low-hazard wastes (e.g., mine tailings); red muds
Motor freight/railroad terminals	Acids and alkalis; heavy metals; ignitable wastes (e.g., acetone; benzene; methanol); lead-acid batteries; solvents
Paint manufacture	Heavy metals (e.g., antimony, cadmium, chromium); PCBs; solvents; toxic pigments (e.g., chromium oxide)
Paper manufacture/printing	Acids and alkalis; dyes; heavy metals (e.g., chromium, lead); inks; paints and resins; solvents
Petrochemical industry/gasoline stations	Benzo-a-pyrene (BaP); hydrocarbons; oily wastes; lead; phenols; spent catalysts
Photofinishing/photographic industry	Acids; silver; solvents
Plastic materials and synthetics	Heavy metals (e.g., antimony, cadmium, copper, mercury); organic solvents
Shipyards and repair shops	Heavy metals (e.g., arsenic, mercury, tin); solvents

(continued)

Table 1.1 (continued)

Sector/source	Typical hazardous waste-stream
Textile processing	Dyestuff heavy metals and compounds (e.g., antimony, arsenic, cadmium, chromium, mercury, lead, nickel); halogenated solvents; mineral acids; PCBs
Timber/wood preserving industry	Heavy metals (e.g., arsenic); non-halogenated solvents; oily wastes; preserving agents (e.g., creosote, chromated copper arsenate, pentachlorophenol)

a number of aquatic environments (Erickson 2002; NRC 1999). Indeed, it is probably reasonable to assume that pollutants from pharmaceuticals and other everyday products have been in the human environments for as long as they have been in use—albeit it is only recently that proper analytical methods have been developed to detect them at the low levels typically found in the environment. Regardless, there currently are a number of uncertainties associated with the determination of risks associated with pharmaceuticals released into various environments—especially because of the inadequacy (or even lack) of knowledge concerning their fate in waste streams, and the variant environments in which they are typically found; their uptake, metabolism and excretion (*viz*., pharmacokinetics) upon entry into ecosystems; and their target affinity and functional effects (*viz*., pharmacodynamics) in non-target species or organisms (Arnold et al. 2014). Still, if pharmaceuticals in the environment are investigated and evaluated in a reasonably holistic fashion, then there is a better chance of properly accounting for their potential effects—even if not in a fully quantitative manner.

1.1.1 The Wide-Ranging Scope of Chemical Hazard Problems: A General Overview

A general review of various chemical materials and their usage in social contexts reveals that hazards from several of the commonly encountered 'social chemicals' could be problematic with respect to their potential human health impacts; this is illuminated by a limited number of the select examples enumerated below.

- *Arsenic* [*As*]. A poison famous from murder mysteries, arsenic [As] has been used in insecticides (among other uses, such as in alloying agents and wood preservatives)—and these have resulted in extensive environmental contamination problems. Also, there have been a number of medicinal, agricultural, and industrial uses for arsenic compounds; for example, arsenic has been used extensively in medicine (*viz*., Fowler's Solution) for the treatment of leukemia, psoriasis, and asthma, as well as in the formulation of anti-parasitic drugs. It is also noteworthy that arsenic is a naturally-occurring element distributed throughout the environment. Arsenic is indeed a ubiquitous element on earth

with metalloid properties and an overall complex chemistry. As a consequence, arsenic is introduced into waters through the dissolution of natural minerals and ores—and thus concentrations in groundwater in some areas are elevated as a result of releases from local rocks. Still, industrial effluents also contribute arsenic to waters in some areas. Accordingly, drinking water tends to pose the greatest threat to public health from arsenic exposures—with severe health effects having been observed in populations drinking arsenic-rich water over extended periods of time. Exposure at work, as well as mining and industrial emissions may also be significant in some locations. Meanwhile, it worth mentioning here that inorganic arsenic can occur in the environment in several forms; in natural waters—and thus in drinking-water—it is mostly found as trivalent arsenite, As(III) or pentavalent arsenate, As(V). Also notable is the fact that organic arsenic species—which is more common in seafood—are far less harmful to human health, and are also readily eliminated by the body.

Overall, human exposure to arsenic can result in serious health effects; for instance, large doses can cause gastrointestinal disorders—and even small quantities may be carcinogenic. Following long-term exposure, the first changes are usually observed in the skin—namely, pigmentation changes, and then thickening (hyperkeratosis). Cancer tends to be a late phenomenon, and usually estimated to take more than ten years to develop. Also, some studies have reported hypertensive and cardiovascular diseases, diabetes, and reproductive effects. On the other hand, absorption of arsenic through the skin is believed to be minimal—and thus hand-washing, bathing, laundry, etc. with water containing arsenic do not appear to pose significant human health risk. In any case, the relationship between arsenic exposure and other health effects is not quite as clear-cut; for instance, according to a 1999 study by the US National Academy of Sciences (NAS), long-term exposure to arsenic in drinking water causes cancer of the skin, lungs, urinary bladder, and may cause kidney and liver cancer. The NAS study also found that arsenic harms the central and peripheral nervous systems, as well as heart and blood vessels, and causes serious skin problems; it also may cause birth defects and reproductive problems. In particular, other fairly recent studies appear to strengthen the evidence of a link between bladder and lung cancer and exposure to arsenic in drinking water. Indeed, even very low concentrations of arsenic in drinking water are believed to be associated with a higher incidence of cancer. Additionally, some research by the US EPA's Office of Research and Development has shown that arsenic can induce an interaction of arsenic compounds with DNA, causing genetic alterations. The study found that methylated trivalent arsenic derivatives (which can be produced by the body in an attempt to detoxify arsenic) produce reactive compounds that cause DNA to break.

- *Asbestos.* A known human carcinogen, asbestos found a wide range of uses in various consumer products for a considerable period of time. Indeed, processed asbestos had typically been fabricated into a wide variety of materials used in consumer products (such as cigarette filters, wine filters, hair dryers, brake linings, vinyl floor tiles, and cement pipes), and also in a variety of construction

materials (*e.g.*, asbestos-cement pipes, floorings, friction products, roofing, sheeting, coating and papers, packing and gaskets, thermal insulation, electric insulation, etc.). Notwithstanding the apparent useful commercial attributes, asbestos emerged as one of the most complex, alarming, costly, and tragic environmental health problems (Brooks et al. 1995). Among other things, its association with lung cancer has been proven—and notably with synergistic effect observed in relation to cigarette smoke exposures.

It is noteworthy that, there are two general sub-divisions of asbestos: the serpentine group—containing only chrysotile (which consists of bundles of curly fibrils); and the amphibole group—containing several minerals (which tend to be more straight and rigid). Anyhow, because asbestos is neither water-soluble nor volatile, the form of concern with respect to human exposure relates to the microscopic fibers (usually reported as, or measured in the environment in units of fibers per m^3 or fibers per cc). In the end, for asbestos fibers to cause any disease in a potentially exposed population, they must gain access to the potential receptor's body. Since they do not pass through the intact skin, their main entry routes are by inhalation or ingestion of contaminated air or water (Brooks et al. 1995)—with the inhalation pathway apparently being the most critical in typical exposure scenarios. In fact, for asbestos exposures, inhalation is expected to be the only significant exposure pathway worth expending resources to appraise. Consequently, potential human exposure and intake is derived based on estimates of the asbestos concentration in air, the rate of contact with the contaminated air, and the duration of exposure. Subsequently, the intake can be integrated with the toxicity index for asbestos to determine the potential risks associated with any exposures; this then forms a basis for developing appropriate public health risk management actions.

- *Bisphenol-A* (*BPA*). A rather familiar example of a chemical finding widespread use in varieties of consumer products, BPA is a human-made chemical used in linings of metal food cans/containers to prevent the degradation of the metal, as well as in some plastic food packaging and other plastic products (particularly in hard polycarbonate plastics). The critical concern with such applications, though, relates to the fact that the chemical constituent is believed to act as a weak estrogen in the body—purported to impact biological systems even in very low doses. Indeed, BPA is generally shown to be a weak endocrine disruptor that mimics the effects of natural estrogen in the body, which at high doses can lead to adverse developmental and reproductive effects in humans; even so, there seems to be significant controversy surrounding the evaluation of this chemical's effects at low doses—i.e., those levels similar to or lower than typical human exposures in practice.

  Overall, it is notable that BPA has been studied extensively for several decades now; indeed, evaluating potential risks associated with food packaging materials in particular has been a scientific challenge for centuries—perhaps going back to the beginning of modern civilization. Even so, there still does not appear to be clear consensus on its standing with respect to public health implications associated with its use in consumer products.

- *Lead* [*Pb*]. Inorganic lead is one of the topmost anthropogenic pollutants—and is now deemed one of the most ubiquitous toxic substances (Chakraborty et al. 2012; Snape and Townsend 2008; Lobinski and Marczenko 1996); it has been used since antiquity, but its use seems to have increased exponentially during the twentieth century (Levallois et al. 1991; Harrison and Laxen 1981). Most commonly, lead has been used in water supply systems, gasolines, automobile batteries, and paints for a long time in modern human history; this, in turn, has resulted in extensive releases into the environment. The typical sources of environmental lead contamination include industry (such as metal smelters and lead-recycling facilities), paints, and exhaust from motor vehicles that used leaded gasoline. Domestic water supply systems have also been a major source of human exposure to lead. As a result of past and current industrial uses, lead has in fact become a common environmental pollutant globally, and is often more problematic in economically disadvantaged and minority-populated areas or regions globally.

  Overall, various uses of lead—such as in storage batteries and as organic anti-knocking additives (tetraalkyllead) to petrol/fuels, cables, solders, steel products, ammunition, shielding systems from radiation and X-rays, circuit boards in computers and electronics equipment, superconductor and optical technology, insecticides, pigments, paints, ceramics, enamels, glass, plastics and rubber products, coal-fired power plants/stations, wastes from runoff and incineration, as well as other industrial effluents—have contributed significantly for the widespread distribution of lead in the environment (Ritson et al. 1999; Hansmann and Koppel 2000). Meanwhile, it is noteworthy that, although legislations have been implemented in various jurisdictions to enforce the use of alternative petroleum additives and recover lead from used batteries in contemporary times, the uses of lead seem to somehow continue unabated in other areas of application—including, for instance, from some planes flying on leaded aviation fuels, smelting plants, industrial boilers, battery makers, coal-burning power plants, and road surfaces. Further elaboration on this subject matter is presented below in Sect. 1.1.2.

  Known, among others things, to be neurotoxic as well as a cause of anaemia, lead has indeed come to be recognized as a primary public health hazard globally (see, *e.g.*, Needleman and Gatsonis 1990; Pirkle et al. 1985; Schwartz 1994). In part, this is due to the fact that Pb can harm a wide variety of organ systems—including the nervous, cardiovascular, kidney, immune, hematological, reproductive, and developmental systems; indeed, exposure to Pb is also likely to result in cancer effects. Meanwhile, it is noteworthy that lead's biggest risks seem to be towards young children—and particularly to their developing nervous systems; in fact, there seem to be significant evidence of cognitive effects even in populations with relatively low mean blood-Pb levels (of between 2 and 8 μg/dL—thus suggesting there may not quite be any known threshold below which scientists could be confident that there will not be any harmful cognitive effects from Pb exposures.

Nutritionally or physiologically lead is not an essential nutrient for either humans or other organisms; on the contrary, it is toxic, bioaccumulative and persistent. In general, lead toxicity derives from the fact that it is absorbed through respiratory or digestive routes, and then preferentially binds to RBCs for distribution to the body tissues. Common observable human health effects include nausea and irritability at low levels, and brain damage at large doses. Of special significance is the storage of lead in the human bone, where its half-life may be in excess of twenty years. Also, the threat of lead poisoning in children and pregnant women is of particular public health concern; ultimately, lead poisoning can cause a number of adverse human health effects—but this is particularly detrimental to the neurological development of children. Further discussion of the effects of lead is provided below in Sect. 1.1.2.

- *Mercury* [*Hg*]. A nervous system toxin, mercury [Hg], is a significant environmental pollutant in several geographical regions/areas (although far less common than the more ubiquitous lead)—especially because of its use in: measuring instruments (*e.g.*, thermometers and manometers); medicines (as antiseptics); dental practice; lamps; and fungicides. Remarkably, Hg can exist in different forms which control its availability, complex distribution, and toxicity; it can be present in both organic and inorganic forms in the environment.

  The typical major sources of Hg to the human environment generally consist of the release of elemental Hg from manometers used to measure the flow of natural gas through pipelines and distribution systems, electrochemical industries, and certain fungicides (Henke et al. 1993; Stepan et al. 1995). Potential sources of airborne Hg releases include combustion of fossil fuels, chlor-alkali plants, waste incineration, mining and smelting of Hg ores, and industrial processes involving the use of Hg (ATSDR 1999a, b; Porcella 1994). Inorganic Hg may [also] be present in soil due to atmospheric deposition of Hg released from both natural and anthropogenic sources as elemental or inorganic Hg vapor, or as inorganic Hg adsorbed to particulate matter. Mercury is indeed a widely distributed hazardous pollutant and has received enormous attention globally because of its persistence in environments, high toxicity to organisms, reactivity and tendency to form more toxic organic mercury compounds, as well as biomagnifications capability along the food web (Jiang et al. 2006; Craig 1986; Beckvar et al. 1996). Typically, Hg released into the environment will persist for a long time—and during which intervening periods the Hg can change between the organic and inorganic forms. Of special interest, one form of organic Hg—namely, methylmercury—can produce a buildup in certain fish; thus, even very low levels of Hg in the ocean and lakes can contaminate the target fish to the point of being a significant environmental and public health concern.

  Overall, the form of Hg and the manner of human exposure determine the nature and/or type of the consequential health effects. Long-term exposure to either organic or inorganic Hg can permanently damage the brain, kidneys, and developing fetuses. Commonly observable human health effects from exposure to large doses of organic Hg compounds include brain damage, often fatal.

- *Organochlorine Compounds/Persistent Organic Pollutants* [*POPs*]. Most organochlorine compounds—including the chlorinated aromatic hydrocarbons, such as PCBs (that have been widely used in electrical transformers) and DDT (that has been widely used as a powerful pesticide/insecticide)—have proven to be notoriously persistent in the environment. PCBs and DDT are indeed persistent lipophilic chlorinated organic compounds that have been used rather extensively globally—as noted in the additional discussions offered below. Meanwhile, it is also noteworthy here that, in various organisms, DDT is slowly transformed to the even more stable and persistent DDE (dichlorodiphenyl dichloroethylene). In view of the intransigent characteristics, these types of chemicals generally qualify for classification as part of the group often referred to as *persistent organic pollutants* [POPs].

  PCBs are the family of chemicals formed by attaching one or more chlorine atoms to a pair of connected benzene rings; depending on the number and position of chlorine atoms attached to the biphenyl ring structure 209 different PCB congeners can be formed—with the chemical and toxicological properties of the PCBs varying from one congener to the next. Traditionally, PCBs found use in heat exchange and dielectric fluid; as stabilizers in paints, polymers, and adhesives; and as lubricants in various industrial processes. More specifically, in the past, PCBs had been used in the manufacture of electrical transformers and capacitors due to the fact that they generally exhibit low flammability, high heat capacity, and low electrical conductivity—and are indeed virtually free of fire and explosion hazards. PCBs also found several 'open-ended applications' (referred to as such, due to the relative ease with which the PCB may enter the environment during use, in comparison to a 'closed system' for transformer/capacitor use) in products such as plasticizers, surface coatings, ink and dye carriers, adhesives, pesticide extenders, carbonless copy paper, dyes, etc. For instance, they gained widespread use in plasticizers because PCBs are permanently thermoplastic, chemically stable, non-oxidizing, non-corrosive, fire resistant, and are excellent solvents. Also, PCBs have been used in laminating adhesive formulations involving polyurethanes and polycarbonates to prepare safety and acoustical glasses; the PCBs have been used in adhesive formulas to improve toughness and resistance to oxidative and thermal degradation when laminating ceramics and metals. Furthermore, PCBs have been used in paints and varnishes to impart weatherability, luster, and adhesion. Broadly speaking, PCBs have also been used in 'nominally closed systems' (due to the relative ease with which the PCB may enter the environment during use, when compared to a 'closed system' such as for transformer/capacitor use) as hydraulic fluids, heat transfer fluids, and lubricants.

  Meanwhile, it is noteworthy that the primary non-occupational source of PCB exposure is food—especially fish from contaminated waters; indeed, ATSDR has noted that the primary route of exposure to PCBs in the general population appears to involve the consumption of contaminated foods, particularly meat, fish and poultry. Thus, recreational and subsistence fishers who eat large amounts of locally caught fish might be at increased risk for exposure to

PCBs. Small amounts of PCBs can also be found in almost all outdoor and indoor air, soil, sediments, surface water, and animals—albeit people are exposed to PCBs primarily from contaminated foods and breathing contaminated air. In the final analysis, the high lipophilicity and the resistance to biodegradation of most organochlorine compounds allow the bioaccumulation of these chemicals in fatty tissues of organisms and their biomagnification through food chains (Dewailly et al. 1996). Anyhow, as a consequence of humans being located at the top of most food chains, therefore, relatively high levels of these compounds have been found in human adipose tissues, blood lipids, and breast milk fat.

DDT, which belongs to the chlorinated insecticide family, was used extensively from the early 1940s to about the early 1970s for agricultural and public health purposes. It is noteworthy that, although its use has long been banned or curtailed in most industrialized nations, leftover DDT products are suspected to have had continued applications to a degree of concern in some parts of the world even long after the ban, especially in the developing nations.

Overall, POPs have become environmental disaster stories, especially in view of their potential to cause severe health effects. For instance, some PCB congeners and DDT isomers possess an endocrine-disrupting capacity, and are believed to contribute to breast cancer risk and various reproductive and developmental disorders (Colborn et al. 1993; Davis et al. 1993; Dewailly et al. 1994a, b, 1996; Falck et al. 1992; Wolff et al. 1993). Indeed, there are several adverse health effects associated with both PCBs and DDT—as, for example, tests on animals show that PCBs can harm reproduction and growth, as well as can cause skin lesions and tumors. Furthermore, when PCB fluid is partially burned (as may happen in the event of a transformer fire), PCDDs and PCDFs are produced as byproducts—and these byproducts are indeed even much more toxic than the PCBs themselves. For instance, dioxin is associated with a number of health risks, and has been shown to cause cancer of the liver, mouth, adrenal gland, and lungs in laboratory animals; furthermore, tests on rats have shown that furans can cause anemia and other blood problems.

By and large, most of the POPs often encountered tend to persist in the environment, as the 'group name' suggests—generally concentrating upward in the food-chain; for instance, most PCB congeners have half-lives ranging from months to several years. Indeed, persistent chemicals have continued to present ongoing challenges to global environmental communities. Consequently, in May 2004, the 'Stockholm Convention' was put in place—in an attempt to stem the tide, so to speak; this international treaty codified a worldwide effort to eliminate POPs—focusing first on twelve of the most prominent chemicals (including DDT, dioxins, PCBs, and certain pesticides). What is more, there is the growing realization that at least certain POPs constitute a global problem that need to be addressed on a global scale. In fact, by virtue of their physiochemical properties, many of the POPs are subject to global environmental transport and distribution—with some passing through food chains (that ultimately may accumulate in some species that serve as food sources for