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## Public health decisions: Actions and consequences\*

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### Abstract

The goal of public health is to promote the best possible health for the whole population. Public health issues are numerous and can be unbelievably complex in form, scope, and possible consequence. Most public health decisions involve assessing several different options, weighing the respective benefits and risks of those options, and making difficult decisions that hopefully provide the greatest benefit to the affected populations. Many risk management decisions involve a variety of societal factors which modify risk assessment choices. The purpose of this paper is to point out difficulties in making decisions that impact public health. The intent of such decisions is to improve public health, but as illustrated in the paper, there can be unintended adverse consequences. Such unplanned issues require continued attention and efforts for responsible officials in the protection of environmental public health. This article presents examples of such events, when in the past, it was necessary to assess and regulate a number of potentially hazardous chemicals commonly used as insecticides, gasoline additives, and wood preservatives.

### Keywords

Chemical regulations; Insecticides; Gasoline additives; Wood preservatives; Risk management decisions

## 1. Introduction

Most facets of 21st century living involve complex public health issues and correspondingly difficult public health decisions. One of the more complex and perhaps least understood of these is that of exposure to hazardous chemicals. There are millions of chemicals known to man, over 100,000 currently in production (Encyclopedia of Global Change, 2001). Many have never been evaluated for toxicity. Those charged with protecting public health are often tasked with evaluating potential risks associated with various chemicals in order to set acceptable exposure standards and rules for use. Often these decisions are not straightforward. Information on toxicity may be missing or incomplete and inter- and intra-species differences in sensitivity may further complicate the interpretation of both risks and

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There is no conflict of interest.

benefits. Decisions that affect public health can be difficult, not only because of their complexity, but also because they have the potential to impact both positively and negatively the well-being of a great number of people. The purpose of this paper is to point out the difficulties in making decisions that impact public health. The intent of such decisions is to improve public health, but as illustrated in the paper, there can be unintended adverse consequences.

## 2. Examples of chemicals in our lives and their regulation

### 2.1. Insecticides: dichlorodiphenyl trichloroethane (DDT)

**2.1.1. Problem definition: mammalian toxicity of bio-persistent chemicals versus increased mortality from malaria that is preventable via vector (e.g., mosquitoes) control**—DDT was first synthesized in 1874, but it was not until 1939 that Müller and his coworkers discovered its insecticidal properties (ATSDR, 2002). Restrictions put in place for DDT use in the 1970's (EPA, 1972) were mainly due to concerns regarding the chemical's persistence in the environment, bioaccumulation in biota and exposure-related health effects such as cancer, neurological, reproductive, and developmental effects (ATSDR, 2002). EPA assigned DDT, DDE, and DDD a weight-of-evidence classification of B2, probable human carcinogens (IRIS, 2008). Studies in humans support the conclusion that DDT and the metabolites are endocrine disruptors (ATSDR, 2002). There is sufficient information from laboratory animal studies that DDT (and the metabolites) causes reproductive and developmental effects (ATSDR, 2002). Similar reports come from observations in wildlife (Blomquist et al., 2006; Guillette et al., 1994; Hamlin and Guillette, 2010; Kolaja and Hinton, 1977).

Results from the National Health and Nutrition Examination Survey (NHANES) indicate that mean serum levels of DDT and DDE in the U.S. population are declining (CDC, 2012). Anderson et al. (1998) estimated that, since the 1970's, levels have declined up to ten-fold. For example, serum *p,p'*-DDT (lipid adjusted) levels in the 95th percentile of the population were 28.0 ng/g (ppb) in 1999–2000, but had dropped to 19.5 ng/g (ppb) in 2003–2004. However, occupational exposures are much higher. For example, the mean DDT serum level in a group of 26 malaria control sprayers in Brazil was 76.9 µg/L and ranged from 7.5 to 473.5 µg/L, whereas 16 unexposed workers had a mean serum level of 16.1 µg/L (range: 5.1–32.9 µg/L) (Minelli and Ribeiro, 1996). *p,p'*-DDT and *p,p'*-DDE serum levels in the exposed workers ranged from 1.6 to 62.9 and 5.9–405.9 µg/L, respectively.

DDT alternatives for fighting malaria include textile nets, impregnated with the pyrethroid insecticide permethrin, that are used on windows and doors of dwellings (Trembley, 2006). Permethrin-treated nets decreased malaria-related mortality in children by 20%. Permethrin, like all synthetic pyrethroids, kills insects by strongly exciting their nervous systems (ATSDR, 2001a). The mode of action is similar to that of DDT, and permethrins display a large variety of mammalian toxicity, as well. New methods for malaria control such as transmission-blocking vaccines and genetically modified mosquitoes are being developed (Ito et al., 2002; Richie and Saul, 2002); however, their usefulness has been questioned. Alternative environmental modification programs to eradicate mosquito larvae have also been investigated (Chanon et al., 2003; Guimaraes et al., 2007). They include methods such

as intermittent irrigation in agriculture, removal of emerging vegetation to eliminate mosquito breeding sites, or introduction of larvicidal biological agents (e.g., bacteria, fungi, and some algae).

The use of DDT has eradicated malaria in different parts of the world (Najera et al., 2011).

However, with restrictions on DDT use in place, the global malaria control policy changed in favor of methods utilizing adulticides (i.e., insecticides killing adult mosquitoes) with domestic preferences to less toxic and less effective chemicals. Following the change, the incidence of malaria increased. A clear causal link between decreased spraying of homes with DDT and increased malaria was reported in South America (Roberts et al., 1997). According to the WHO, there were 300–500 million clinical cases of malaria each year resulting in 1.5–2.7 million deaths – mostly in sub-Saharan Africa (Hileman, 2006; IDRC, 1996). More than half of the deaths were children. Since 1972, malaria has killed over 50 million people worldwide. The effectiveness of DDT was demonstrated in South Africa which used DDT and found that malaria cases were kept very low (Hileman, 2006). In 1996, South Africa switched to other pesticides and the incidence of malaria rose sharply thereafter. In December 2000, South Africa approved a ruling allowing for the continued use of DDT in malaria vector control as the United Nations Environment Program concluded the negotiations on 12 persistent organic pollutants (POPs) (MFI, 2000). The program decided that DDT can still be used for spraying interior walls in regions where malaria is a problem (SCPOP, 2001). The US EPA participated in those negotiations. In September 2006, the World Health Organization (WHO) declared its support for the indoor use of DDT in African countries where malaria remains a major health problem, citing that benefits of the pesticide outweigh the health and environmental risks (WHO, 2006). This is consistent with the Stockholm Convention on POPs, which bans DDT for all uses except for malaria control.

According to the latest WHO (2013) data, there were about 219 million malaria cases (with an uncertainty range of 154–289 million) and an estimated 660,000 malaria deaths (with an uncertainty range of 490,000–836,000) in 2010 world-wide. Increased prevention and control measures have resulted in a decrease in malaria mortality rates by more than 25% globally since 2000 and by 33% in the WHO African Region.

In addition to being effective, DDT is a relatively cheap tool to fight malaria. For example, the cost of spraying one house with DDT per year in Ecuador was estimated as \$1.44 (Roberts et al., 1997). Alternative insecticides are much more costly (e.g., malathion is five times more expensive than DDT and has its own toxicity issues ATSDR, 2004). This is a major problem for developing countries with limited resources. On a greater scale, eradication of malaria brings economic growth and prosperity. When Gallup and Sachs (2001) analyzed the growth in gross domestic products per capita between 1965 and 1990, they reported that countries with substantial occurrence of malaria grew 1.3% per year less than countries with little or no malaria and that a 10% reduction in malaria was associated with 0.3% higher growth per year.

When DDT was first recognized as an environmental hazard, it was considered to be harmful to use in vector control efforts. However, with a greater understanding of both the adverse impacts and potential benefits of this chemical, those views are now changing. DDT usage is a prime example of choosing a course of action that will provide the best net benefit for adversely-affected populations in specific circumstances. These beneficial effects are now widely recognized, and DDT application in localized areas and situations has been shown to reduce morbidity and mortality from vector-transmitted diseases.

## 2.2. Gasoline additives: lead, methyl tert-butyl ether (MTBE), and ethanol

**2.2.1. Problem definition: mammalian toxicity of gasoline additives and potential for increased environmental pollution**—The evolution of the United States society in the 20 century is inextricably tied to the evolution of the automobile as a part of individual and family life. However, the necessary application of additives to gasoline in powering the automobile has resulted in a variety of challenges in the public health sector. (McGarity, 2004)

Gasoline is a mixture of organic chemicals including straight-chain alkanes, branched alkanes, cycloalkanes, aromatics (benzene less than 1% in U.S.), and alkenes (ATSDR, 1995). Although gasoline can induce a wide range of toxic effects, major discussions related to public health effects of gasoline are not about gasoline per se but about additives that serve to make gasoline a better product.

Tetraethyl lead was initially added to gasoline (from 1920's) to prevent non-uniform combustion differential that can potentially damage the engine. (Seyferth, 2003) Prior to EPA regulation of lead content in gasoline during the early 1970s, approximately 250,000 tons/year of organic lead were added to gasoline in the United States (Giddings, 1973). By 1984, combustion of leaded gasoline was responsible for approximately 90% of all anthropogenic lead emissions.

With increasing lead levels in the environment concerns about lead toxicity emerged. Neurotoxicity of lead in humans is well established and children are even more sensitive to lead toxicity than adults (ATSDR, 2007). An extensive compilation of pediatric patients identified encephalopathy at levels in the range of 90–800 µg/dL in blood (NAS, 1972); signs include hyperirritability, ataxia, convulsions, stupor, and coma. Histopathologic findings in fatal cases of lead encephalopathy in children include cerebral edema, altered capillaries, and perivascular glial proliferation. Non-fatal cases are at great risk for neurological and cognitive impairments. Renal effects, hypertension, and decreased fertility were reported at level 40 µg/dL (ATSDR, 2007). Several cross-sectional studies of asymptomatic children with relatively high lead body burdens were published in the 1970s that identified a pattern of IQ and neuropsychologic deficits at blood lead levels of 40–60 µg/dL (ATSDR, 2007).

In 1973, after determining that lead particle emission presented a significant health risk to urban populations, the EPA initiated a phase-down program designed to minimize the amount of lead in gasoline. The elimination of lead additives was also encouraged by the development of catalytic converters, devices to reduce undesirable byproducts of combustion

but devices incompatible with lead-containing gasoline. (Seyferth, 2003) By 1988, the phase-down program had reduced the total lead usage in gasoline to less than 1% of the amount of lead used in the peak year of 1970 (EPA, 1996a). In 1990, a congressional amendment to the Clean Air Act (CAA) banned the use of gasoline containing lead or lead additives as fuel in motor vehicles (effective date January 1, 1996) (ATSDR, 2007).

The national prevalence rate for adults with blood lead levels 25 µg/dL or higher was 7.5 per 100,000 adults (CDC, 2006). The prevalence rate has reportedly been declining annually since 1994. Jones et al. (2009) reported that, based on a comparison of NHANES 1988–1991 to NHANES 1999–2004, the prevalence of blood lead levels of 10 µg/dL or greater in children decreased from 8.6% to 1.4%, an 84% decline.

Over the years a number of oxygenated solvents have been used as substitute for the lead additive in enhancing engine performance. The use of such materials was further encouraged by the passage and implementation of the Clean Air Act requiring special fuel blends for areas of the country with documented poor air quality. When first introduced in the 1970s, MTBE, or methyl tertiary-butyl ether, seemed like an ideal choice for gasoline additive. MTBE is manufactured by combining two readily available compounds-methanol and isobutylene. MTBE enhances performance and reduces the production of undesirable combustion products such as carbon monoxide, benzene and other volatile organic compounds. Nearly all the MTBE produced in the United States is used as octane boosters and oxygenating agents in reformulated gasoline and these uses are the only ones for which reliable production figures are readily available (ATSDR, 1996). Starting in the late 1980s, MTBE production increased rapidly. The 1990 Clean Air Act created a guaranteed market of some 400,000 barrels per day for all types of reformulated fuel oxygenated additives. For MTBE, the estimated production capacity in the United States during 1995 was 240,100 barrels (about 62.2 million pounds or 28.2 million kg) per day (CMR, 1995).

Unfortunately the introduction of MTBE occurred at a time frame where significant problems were occurring with underground storage tanks and facilities used by the gasoline distribution system. Historically poor design or implementation resulted to undiscovered releases of petroleum products in the environment. This situation was compounded by several factors associated with the chemical MTBE. This additive has a distinct unpleasant odor which is readily recognized upon releases and that most people find disagreeable (Angle, 1991; Gilbert and Calabrese, 1992). The chemical characteristics of MTBE are such that it is readily soluble in water and hence it becomes widely distributed upon underground release, which inevitably results in significant contamination of drinking water. The EPA has established a Drinking Water Health Advisory for MTBE of 20–40 ppb or below to protect consumer acceptance of the water resource and also provide a large margin of exposure (safety) from toxic effects. (EPA, 1997) MTBE can also be emitted to the ambient air, primarily from pre-combustion volatilization. This can then lead to low-level background exposure potentials over a large geographical area.

According to much of the existing literature, MTBE poses no severe human health threats at the exposure levels anticipated for the general population (ATSDR, 1996). In some studies, motorists and workers have reported symptoms of coughing, burning sensations in the nose

and throat, headache, nausea or vomiting, dizziness, and feelings of disorientation that may be associated with MTBE exposure (Chandler and Middaugh, 1992). However, other studies have not shown these effects Cain et al., 1994; Prah et al., 1994). MTBE blood levels reflect recent exposure. In the NHANES 2001–2002, 2003–2004, and 2005–2006 subsamples, MTBE was detectable in most of the population; however, geometric means have decreased over time from 16.4 to 6.16 pg/mL (CDC, 2012). In studies of U.S. automobile drivers when MTBE was used as a fuel additive, blood levels were about 10–100 times higher (depending in part on the concentration of MTBE in the gasoline) than those in NHANES subsamples taken after removal of MTBE from gasoline (Lemire et al., 2004; Mannino et al., 1995; White et al., 1995).

Despite the lack of consensus for overt MTBE toxicity at environmental levels, several multimillion dollar lawsuits were filed on behalf of communities that had MTBE in their water supply. Currently, several states in the U.S. restrict or ban MTBE use in gasoline (EIA, 2003). However, these states still have to meet the EPA's Clean Air Act requirements. As has been noted (Davis and Farland, 2001), the irony of the situation is that the introduction of a substance into widespread use to enhance performance and reduce air pollution has resulted in significant environmental contamination of drinking water systems.

The attention for a fuel additive for enhanced performance and air pollution reduction moved to use of ethanol once the issues associated with MTBE became apparent. Ethanol is a oxygenated compound similar to MTBE and its satisfactory properties as a gasoline additive have been known for a number of years. Ethanol has advantages in being a renewable resources. Significant programs in Brazil in the 1980's also demonstrated the domestic production through agriculture could reduce a country's dependence on imported oil (Rohter, 2006). Significant efforts after 1985 were made through environmental programs to reduce leaking underground tanks, with limited success. However in the case of ethanol, the health effects in any environmental contamination are well known.

The use of ethanol as a gasoline additive is not without its challenges. Ethanol is produced from corn and its production relies on heavy subsidies for farmers. In addition, corn used for ethanol production is not available for food production, driving up the cost of food. According to the Congressional Budget Office (CBO, 2009), the corn prices increased more than 50% between April 2007 and April 2008 due to the increased demand for ethanol production. Consequently, the food prices have increased by 10 to 15% during the same time period.

A detailed cost/benefit analysis study of reformulated gasoline with MTBE, ethanol, and toluene or isooctane (i.e., non-oxygenated) additives was conducted for the State of California (Fernandez and Keller, 2001). Regarding air quality damages, the study found that the use of reformulated gasoline with MTBE will increase the air concentration of formaldehyde, a combustion byproduct of MTBE. Formaldehyde is known to be a human carcinogen (NTP, 2011) and can induce other health effects in exposed populations. The authors predicted that the total cost of decreased air quality may be up to \$27 million in terms of increased costs of illness and mortality. Similarly, the combustion by-product of ethanol, acetaldehyde, can decrease the air quality substantially. As a direct cost, the fuel



price increase was calculated as \$135–\$675 million and \$290–\$991 million for MTBE and ethanol, respectively. Ethanol is marketed as the future “clean” additive. But ethanol is manufactured from corn and, in U.S., 75% of the field corn acreage is treated with the herbicide atrazine (EPA, 2013). About 76.4 million pounds of atrazine are applied annually. Atrazine has been reported to pollute 91–98% of the U.S. surface waters (ATSDR, 2003). Atrazine and its metabolite deethylatrazine were the top chemicals found in mixtures of pollutants detected in wells used for drinking water in the U.S. (Squillace et al., 2002). The EPA (2002) concluded that these chemicals affect reproductive function and development, and has set the drinking water standard (maximum contaminant level = MCL) of 0.003 mg/L (3 ppb) for atrazine. In many states (e.g., Illinois, Nebraska, Iowa and Minnesota), the MCL is often exceeded. For example, data obtained from public water systems by the Environmental Working Group (EWG) show that 1.3 million people in Indiana were exposed to atrazine in their drinking water above state or federal health-based limits between 1998 and 2003 ([www.ewg.org/tapwater/index.php](http://www.ewg.org/tapwater/index.php)). However, even with the dramatic increase in corn production, the economic advantages of atrazine use have been questioned (Ackerman, 2007). Further increases in water contamination with atrazine due to the demand for ethanol from corn have the potential for widespread adverse impacts on public health and the environment.

In addition, according to the recent news, ethanol-fueled cars affect food supplies and prices. A direct quote from the Economist (2007) claimed that “This year’s biofuels will take a third of America’s (record) maize harvest. That affects food market directly: fill up an SUV’s fuel tank with ethanol and you have used enough maize to feed a person per year. And it affects them indirectly, as farmers switch to maize from other crops. The 30 m tones of extra maize going to ethanol this year amounts to half the fall in the world’s overall grain stocks”. Proper evaluation of all benefits and risks is important not only for the regulators, but also for those who formulate and implement public policy.

Some of the characteristics of ethanol have made it less attractive to refiners than MTBE. Ethanol has higher volatility which makes it more difficult to meet emissions standards. However, ethanol contains more oxygen so only about half as much ethanol (by volume) is needed for reformulated gasoline. In contrast, gasoline with ethanol is more expensive in comparison to gasoline with MTBE (EIA, 2003).

Ethanol also poses challenges in its introduction into the distribution system. Ethanol cannot be shipped in the general pipeline systems due to its chemical characteristics, and the location of pipelines is not conducive in shipping from production sites (Thompson, 2006). Accidents involving tanker trucks and any resulting fires pose a dangerous situation to responders because of the heightened temperatures created and unique characteristics of ethanol combustion.

In this example of gasoline additives, eliminating lead as a gasoline additive provides the greatest health benefit to most people, and especially the most vulnerable – children and the unborn. The effort spent on settling on an alternative additive illustrates that there are challenges that are likely to arise and must be considered with major regulation decisions that impact public health.

### 2.3. Wood preservatives: creosote, pentachlorophenol, and Chromated Copper Arsenate (CCA)

#### 2.3.1. Problem definition: mammalian toxicity of bio-persistent chemicals versus decay of lumber used for constructions and associated economic loss in light of increased understanding of human health impacts—

Wood is an economical, renewable resource for many outdoor applications. Unfortunately the maintenance of reasonable lifespan for structures requires the use of chemical agents to minimize structural degradation from various sources-termites, fungi, etc. The changes in use of various agents reflect increased understanding of human health impacts, and the allowable and acceptable decisions associated with that understanding. In general, there are three types of preservatives used: creosotes, oilborne preservatives (e.g., pentachlorophenol, copper naphthenate), and waterborne preservatives (e.g., Chromated Copper Arsenate (CCA)).

Creosotes are mixtures of chemicals containing mostly condensed aromatic ring compounds (IARC, 1985). They are obtained from the distillation of coal tars. Coal tar creosote has been widely used as a wood-treatment pesticide since the turn of the 20th century. In the environment, coal tar creosote components are slowly released from treated wood products by oil exudation, rain-water leaching, and by volatilization of the lighter fractions (Henningsson, 1983). Losses of creosote from impregnated wood are dependent on the kind of coal used to produce the coal tar, the kind of coke oven used to make the coal tar, and the conditions under which the wood is used (Leach and Weinert, 1976). USDA (1980) reported that the major components of creosote were not detected in soil samples taken to a depth of 6 inches within 2–24 inches from treated poles, presumably as a result of biotransformation of mobilized components by soil microorganisms.

PAHs (polycyclic aromatic hydrocarbons), components of creosote, are used as markers of body burdens following exposure to creosotes (ATSDR, 2002). Human studies indicate that dermal exposure to creosote used in wood treatment or in coking oven processes contribute more significantly to the total body burden than respiratory exposures (Klingner and McCorkle, 1994; Malkin et al., 1996; Van Rooij et al., 1993). Dermal exposure is also important, as was demonstrated in a study that measured internal levels of PAHs (using pyrene as a biomarker) in workers at a creosote wood impregnation plant (Van Rooij et al., 1993). For workers protected by Tyvek coveralls worn beneath outer work-clothes, dermal pyrene contamination was approximately 35% less than that of unprotected workers, and urinary levels of 1-hydroxypyrene were 3.2  $\mu\text{g}$ . In comparison, unprotected workers had total pyrene skin contamination of 47–1510  $\mu\text{g}/\text{day}$  and urinary levels of 1-hydroxypyrene of 6.6  $\mu\text{g}$ . Volatile pyrene in the breathing-zone air was measured at 0.3–3.0  $\mu\text{g}/\text{m}^3$ . In this study, it was concluded that 15 times more pyrene was absorbed through dermal uptake than through respiratory uptake. In addition, workers who manually install creosote treated wood products have been reported to be exposed mainly through dermal contact (USDA, 1980).

The major health concern is induction of cancer. The EPA has determined that creosote is a class B1 carcinogen (probable human carcinogen) (IRIS, 2008). The National Toxicology Program classifies coal tar (coke oven emissions, coal tar, coal tar pitch, and creosotes) as a



known human carcinogen (NTP, 2011). Creosotes are still commonly used in the United States. However, designation of creosote products as restricted use pesticides requires that only certified applicators use this product (mainly on railroad ties) (EPA, 1986). It is of note that in 2002 creosote had been identified in at least 46 of the 1613 hazardous waste sites that had been proposed for inclusion on the EPA National Priorities List. (ATSDR, 2002b).

The most common oil-based preservative is pentachlorophenol. It is applied usually in a 5% solution of light petroleum solvents (ATSDR, 2001b). Typically, commercial grade pentachlorophenol is 86% pure. Contaminants generally consist of other polychlorinated phenols, polychlorinated dibenzo-p-dioxins (CDDs), and polychlorinated dibenzofurans (CDFs), which are formed during the manufacturing process. Pentachlorophenol was one of the most heavily used pesticides in the United States, but is now regulated as a restricted-use pesticide (EPA, 1984a) and its use is restricted to the treatment of utility poles, railroad ties, and wharf pilings. Pentachlorophenol can leach into soil from the treated woods and has been detected in soil within 12 inches of treated utility poles at concentrations ranging from 3.4 to 654 ppm (Arsenault, 1976). Pentachlorophenol is stable to hydrolysis and oxidation, but it is quickly photolyzed by sunlight and can be metabolized by microorganisms, animals, and plants. In humans, dermal, inhalation and oral routes of exposure are relevant. Among the general adult population, in an NHANES 2003–2004 subsample, the 95th percentile urinary pentachlorophenol levels were about 3.40 µg/L (CDC, 2012). However, some segments of general population may have higher exposures. For example, a mean pentachlorophenol blood serum level of 420 µg/L was reported for residents of log homes built with pentachlorophenol-treated logs (Cline et al., 1989). Accordingly, their urinary levels of pentachlorophenol were hundreds fold higher than in the general population. In an occupational exposure study, Ferreira et al. (1997) compared the concentration of pentachlorophenol in the urine and blood of a group of workers occupationally exposed to pentachlorophenol at a wood-transformation unit to those of a control group with no known exposure to pentachlorophenol. The mean level of pentachlorophenol in the occupationally-exposed group was 1197 and 1273 µg/L in urine and blood, respectively. The mean concentration of pentachlorophenol in the control group was considerably lower at 6.4 and 15.3 µg/L in urine and blood, respectively. Major health effects of pentachlorophenol include hepatic, immunologic, and endocrine (thyroid) effects in humans (ATSDR, 2001b). Pentachlorophenol is classified as a probable human carcinogen by EPA (IRIS, 2008).

In the United States, pentachlorophenol treated wood was identified as the major source of dioxins in the environment and the cause of increased cancer risk. For example, contact with pentachlorophenol contaminated soils leached from treated woods represents a cancer risk to children as high as  $2.2 \times 10^{-4}$  (EPA, 1999).

It has been estimated that there are about 60 million utility-owned wood poles and 54 million crossties (in railroads) in the U.S., most of them preserved by pentachlorophenol or creosote (EPA, 2008). More than a half of the utility poles in service (about 36 million) are treated with pentachlorophenol. One older source quoted the cost of a utility pole as \$271 and re-treatments costing about \$30–35 per pole (Feldman, 2001).

With restrictions on use in place for creosotes and pentachlorophenol, a question arose regarding the treatment of a wide variety of wooden structures outdoors such as decks, playground equipment, picnic tables, garden-bed borders, and docks. To preserve these wooden structures and protect them against termites and fungi, CCA treated wood (also called pressure-treated wood) began to be used for their construction. By 1995, CCA represented 94% of all the waterborne preservatives applied (Felton and De Groot, 2003). Around that time, about 90% of all arsenic used in the United States was used to preserve wood. All three metals of CCA treated wood can leach into the environment. In the soil below CCA-treated wooden decks, arsenic, copper, and chromium levels were found to be twenty, four, and two times higher, respectively, than normal background levels (Stilwell and Gorny, 1997). No increases were found at the depths over 6 inches. There is a concern for exposure of the general population, especially children. It was estimated that exposure of children per playground visit is in the range of 24–630  $\mu\text{g}$  for arsenic, 35–288  $\mu\text{g}$  for chromium, and 17–181  $\mu\text{g}$  for copper (CDHS, 1987). Based on the weight-of-evidence for exposure-effect scenarios, the playground exposure to arsenic may be a health concern for children with high end exposures. Arsenic is known to cause a large range of effects, including gastrointestinal, neurological, and cancer (inorganic arsenic is classified as a human carcinogen by EPA IRIS, 2008). The EPA classified CCA as a restricted-use pesticide and ordered stores that sell CCA-treated woods to have copies of consumer information sheet that describes safe handling recommendations. Since January 2004, EPA has banned CCA as a preservative for wood intended for residential use (except for the lumber that is used in permanent wood foundations) (EPA, 2003).

The quest for less toxic but efficient preservatives continues. Waterborne preservatives that are on the market today include ammoniacal copper quat, amine copper quat (uses ethanolamine instead of ammonia to act as the treating solution carrier), copper azole (contains boron), and borate oxide (water soluble-leach; not recommended for water or soil media) (Morrison, 2004). However, more copper has to be added to make the newer waterborne preservatives sufficiently effective. Some preservatives increased the copper content from 18% to as much as 96% (Morrison, 2004). This translates into a higher cost of the treated wood ranging from 15% to 35%. An induction of copper-related toxicity associated with exposures is also a possibility (Morrison, 2004).

As discussed above, regulatory perspectives on wood-preserving chemicals have changed over time as new information about potential health impacts has become available. Careful consideration of all the potential pro's and con's of using a specific chemical for a specific purpose is a good public health policy. It seems that the jury may still be out in regards to which wood preservatives may be the greater hazard and which the lesser hazard. The search continues for adequate wood preservatives with minimal human and environmental impacts.

### 3. Conclusion

Many new chemicals are introduced into the marketplace every week. The number of structure-searchable chemicals already exceeds 10 million (Elsevier, 2007) and the overall number of registered substances is in excess of 30 million (CAS, 2007). Use of chemicals is essential in meeting the social and economic goals of the world's population. Advances in

industrial chemistry and in pharmacology contributed tremendously to the development and improved quality of life of modern societies around the world. However, exposures to environmental chemicals can also adversely affect life on this planet. Everyone carries a body burden of chemicals that range from primary elements and radioactive materials to synthetic, persistent chemicals. The major task for public health professionals is to balance the benefits versus the risks of chemical exposures. With more studies published regarding behavior of chemicals in the environment as well as information on their toxicity and toxicokinetics, we may need to adjust our views on their health impacts. Also, we need to realize that sometimes it is unavoidable to incur some degree of risk in order to protect the most individuals. Evaluation of public health risks associated with chemical exposures has had an interesting and thought-provoking history, and there is little reason to expect that to change.

## References

- Ackerman F. The economics of atrazine. *Int J Occup Environ Health*. 2007; 13:441–449.
- Anderson HA, Falk C, Hanrahan L, et al. Profiles of Great Lakes critical pollutants: a sentinel analysis of human blood and urine. *Environ Health Perspect*. 1998; 106(5):279–289. [PubMed: 9560354]
- Angle CR. If the tap water smells foul, think methyl-*tert*-butyl ether. *J Am Med Assoc*. 1991; 266(21): 2985–2986.
- Arsenault, RD. Pentachlorophenol and Contained Chlorinated Dibenzodioxins in the Environment: A Study of Environmental Fate, Stability, and Significance When Used in Wood Preservation. Alexandria, VA: American Wood-Preservers Assoc; 1976. p. 122-147.
- ATSDR. Toxicological Profile for Lead. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2007. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Malathion. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2004. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Atrazine. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2003. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for DDT, DDE, DDD. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2002a. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Wood Creosote, Coal Tar Creosote, Coal Tar, Coal Tar pitch, and Coal Tar Pitch Volatiles. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2002b. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for .Pyrethrins and Pyrethroids. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2001a. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Pentachlorophenol. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 2001b. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Methyl *tert*-Butyl Ether (MTBE). Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 1996. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]
- ATSDR. Toxicological Profile for Automotive Gasoline. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services; 1995. <http://www.atsdr.cdc.gov/toxprofiles> [accessed June 2013]

- Blomquist A, Berg C, Holm L, Brandt I, Ridderstrale Y, Brunstrom B. Defective reproductive organ morphology and function in domestic rooster embryonically exposed to o, p'-DDT or ethynyl estradiol. *Biol Reprod.* 2006; 74(3):481–486. [PubMed: 16280416]
- CAS (Chemical Abstracts Service). Registry Number and Substance Counts. Columbus OH: American Chemical Society; 2007. Available on the Internet at: [www.cas.org/cgi-bin/cas/regreport.pl](http://www.cas.org/cgi-bin/cas/regreport.pl) [accessed October 2007]
- Cain, WS., Leaderer, BP., Ginsbert, GL., et al. Human Reactions to Brief Exposures to Methyl Tertiary-Butyl Ether (MTBE). In: John, B., editor. New Haven, CT: Pierce Laboratory; 1994.
- Chandler, B., Middaugh, J. Potential Illness Due to Exposure to Oxygenated Fuels, Anchorage, Alaska. Anchorage, AK: State of Alaska Department of Health and Social Services; 1992 Dec 23.
- Centers for Disease Control and Prevention (CDC). Updated Tables. National Health and Nutrition Examination Survey (NHANES); 2012. Fourth National Report on Human Exposure to Environmental Chemicals. [www.cdc.gov/exposurereport/](http://www.cdc.gov/exposurereport/) [accessed March 2014]
- Centers for Disease Control and Prevention (CDC). Adult blood lead epidemiology and surveillance—United States, 2003–2004. *MMWR Morb Mortal Wkly Rep.* 2006; 55(32):876–879. [PubMed: 16915221]
- Cline RE, Hill RH Jr, Phillips DL, et al. Pentachlorophenol measurements in body fluids of people in log homes and workplaces. *Arch Environ Contam Toxicol.* 1989; 18:475–481. [PubMed: 2774665]
- CBO (Congressional Budget Office). The Impact of Ethanol Use on Food Prices and Greenhouse Gas Emissions. 2009. [www.cbo.gov/publication/41173](http://www.cbo.gov/publication/41173)
- CDHS. Report to the Legislature. 1987. Evaluation of hazards posed by the use of wood preservatives on playground equipment. State of California Department of Health Services (CDHS).
- Chanon KE, Mendez-Galvan JF, Galindo-Jaramillo JM, Olguin-Bernal H, Borja-Aburto VH. Cooperative actions to achieve malaria control without the use of DDT. *Int J Hyg Environ Health.* 2003; 206:387–394. [PubMed: 12971694]
- CMR. Chemical Marketing Report (03.04.94). 1995. Chemical Profile: MTBE.
- Davis JM, Farland WH. The paradoxes of MTBE. *Toxicol Sci.* 2001; 61:211–217. [PubMed: 11353129]
- Economist. The end of cheap food. *Econ.* 2007; 385(8558):11–12.
- Encyclopedia of global change. *Encyclopedia of Global Change: Environmental Change and Human Society.* In: Goudie, AS., editor. Hazardous Waste. Oxford University Press; NY: 2001. p. 579
- EIA (Energy Information Administration). [accessed November 2006] Status and Impact of State MTBE Bans. 2003. <http://tonto.eia.doe.gov/ftproot/service/mtbe.pdf>
- Elsevier (Elsevier Information Systems GmbH). [accessed October 2007] CrossFire Beilstein Database Exceeds Ten Million Compounds. 2007. Press release August 20th 2007. Available on the Internet at: ([www.beilstein.com/PR\\_Beilstein%2010m\\_Final.pdf](http://www.beilstein.com/PR_Beilstein%2010m_Final.pdf))
- EPA (Environmental Protection Agency). Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Methyl Tertiary-Butyl Ether (MtBE) EPA-822-F-97-009. 1997.
- EPA (Environmental Protection Agency). Atrazine Background. 2013. [http://www.epa.gov/pesticides/factsheets/atrazine\\_background.htm](http://www.epa.gov/pesticides/factsheets/atrazine_background.htm)
- EPA (Environmental Protection Agency). [accessed November 2006] Chromated Copper Arsenate (CCA): EPA Testimony on CCA Treated Wood. 2003. <http://www.epa.gov/oppa001/reregistration/cca/ccatestimony1.htm>
- EPA. The Grouping of a Series of Triazine Pesticides Based ON A Common Mechanism of Toxicity. Washington, DC: U.S. Environmental Protection Agency. Office of Pesticides Programs; 2002. <http://www.epa.gov/opsrdd1/cumulative/triazines/triazinescommonmech.pdf>
- EPA. U.S. Environmental Protection Agency. Science Chapter for the Reregistration Eligibility Decision Document for Penta (PC Code: 063001. Registration Case Number 2505). Washington, DC: 1999.
- EPA. Pentachlorophenol Revised Risk Assessments; Notice of Availability and Solicitation for Risk Reduction Options. *Fed Reg.* 2008; 73(74):20638–20640.
- EPA (Environmental Protection Agency). Code of Federal Regulations. U.S. Environmental Protection Agency; 1996a. 40 CFR 268, Appendix XI

- EPA (Environmental Protection Agency). Code of Federal Regulations. U.S. Environmental Protection Agency; 1996b. 40 CFR 745
- EPA (Environmental Protection Agency). Fed Reg. Vol. 51. U.S Environmental Protection Agency; 1986. Creosote, Pentachlorophenols, and Inorganic Arsenicals: Amendment of Notice of Intent to Cancel Registrations; p. 1334-1348.
- EPA (Environmental Protection Agency). Notice of Intent to Cancel Registration of Pesticide Products Containing Creosote, Pentachlorophenol (Including its Salts), and the Inorganic Arsenical. 1984a 49 CAR 28666 (13.07.84).
- EPA (Environmental Protection Agency). Position Document 4. Washington, DC: U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances; 1984b. Wood preservative pesticides: creosote, pentachlorophenol, inorganic arsenicals.
- EPA (Environmental Protection Agency); U.S. Environmental Protection Agency. Consolidated DDT Hearings: Opinion and Order of the Administrator. Federal Register. 1972; 37(131):13369.
- Feldman. Petition for Suspension and Cancellation of Pentachlorophenol. 2001. <http://www.beyondpesticides.org/wood/Penta%20Petition.htm>
- Felton CC, De Groot RC. The recycling potential of preservative treated wood. For Prod J. 2003; 46(7/8):20–25.
- Ferreira AG, Vieira DN, Marques EP, et al. Occupational exposure to pentachlorophenol: the Portuguese situation. Ann NY Acad Sci. 1997; 837:291–299. [PubMed: 9472347]
- Fernandez L, Keller AA. Cost benefit analysis of MTBE and Alternative gasoline formulations. J Environ Sci Pollut. 2001; 3:173–188.
- Gallup JL, Sachs JD. The economic burden of malaria. Am J Trop Med Hyg. 2001; 64(1/2):85–96. [PubMed: 11425181]
- Giddings, JC., editor. Chemistry, Man, and Environmental Change: An Integrated Approach. New York, NY: Harper & Row, Publishers Inc; 1973. Lead in gasoline; p. 351-353.
- Gilbert, CE., Calabrese, EJ. Developing a standard for methyl butyl ether in drinking water. In: Gilbert, CE., Calabrese, EJ., editors. Regulating Drinking Water Quality. Lewis Publishers; 1992. p. 231-252.
- Guillette LJ, Gross TS, Masson GR, Matter JM. Developmental abnormalities of the gonad and abnormal sex hormone concentrations in juvenile alligators from contaminated and control lakes in Florida. Environ Health Perspect. 1994; 102:680–688. [PubMed: 7895709]
- Guimaraes RM, Asmus CI, Meyer A. DDT reintroduction for malaria control: the cost-benefit debate for public health. Cad Saude Publica. 2007; 23(12):2835–2844. [PubMed: 18157325]
- Hamlin HJ, Guillette LJ. Birth defects in wildlife: the role of environmental contaminants as inducers of reproductive and developmental dysfunction. Syst Biol Reprod Med. 2010; 56(2):113–121. [PubMed: 20377310]
- Henningsson B. Environmental protection and health risks in connection with the use of creosote. Holz als Roh- und Werkstoff. 1983; 41:471–475.
- Hileman B. Malaria control. Chem Eng News. 2006; 84(3):30–31.
- IDRC (International Development Research Center). [accessed November 2006] Statistics on Malaria. 1996. (<http://archve.idre.ca/books/reports/1996/01-07e.html>)
- IARC. Polynuclear Aromatic Compounds. Part 4. Bitumens, Coal-Tars and Derived Products, Shale Oils and Soots. Vol. 35. Lyon, France: World Health Organization, International Agency for Research on Cancer; 1985. Monographs on the evaluation of the carcinogenic risk of chemicals to humans; p. 104-140.
- IRIS. Integrated Risk Information Systems. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; 2008. <http://www.epa.gov/iris/subst> [accessed November 2008]
- Ito J, Ghosh A, Moreira LA, Wimmer EA, Jacobs-Lorena M. Transgenic anopheline mosquitoes impaired in transmission of malaria parasite. Nature. 2002; 417(6887):452–455. [PubMed: 12024215]
- Jones RL, Homa DM, Meyer PA, et al. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988–2004. Pediatrics. 2009; 123:e376–e385. [PubMed: 19254973]



- Klingner TD, McCorkle T. The application and significance of wipe samples. *AIHA J Short Commun.* 1994;251–254.
- Kolaja GJ, Hinton DE. Effects of DDT on eggshell quality and calcium adenosine triphosphatase. *J Toxicol Environ Health.* 1977; 3(4):699–704. [PubMed: 145496]
- Leach, CW., Weinert, JJ. A report to the Environmental Task Group, Sub-Group No. 5 (creosote) of the American Wood Preservers Institute. 1976.
- Lemire S, Ashley D, Olaya P, et al. Environmental exposure of commuters in Mexico City to volatile organic compounds as assessed by blood concentrations, 1998. *Salud Publica Med.* 2004; 46(1): 32–38.
- Malkin R, Kiefer M, Tolos W. 1-Hydroxypyrene levels in coal-handling workers at a coke oven. *J Occup Environ Med.* 1996; 38(11):1141–1144. [PubMed: 8941904]
- Mannino DM, Schreiber J, Aldous K. Human exposure to volatile organic compounds: a comparison of organic vapor monitoring badge levels with blood levels. *Int Arch Occup Environ Health.* 1995; 67:59–64. [PubMed: 7622282]
- McGarity TO. MTBE: a precautionary tale. *Harv Environ Law Rev.* 2004; 28:281–342.
- MFI (Malaria Foundation International). [accessed November 2006] Our Campaign to Prevent a Ban of DDT for Malaria Control has been Successful. 2000. <http://www.malaria.org/DDTpage.html>
- Minelli E, Ribeiro M. DDT and HCH residues in the blood serum of Malaria control sprayers. *Bull Environ Contam Toxicol.* 1996; 57:691–696. [PubMed: 8791542]
- Morrison DS. Pressure-treated wood: the new generation. *Fine Homebuilding.* 2004; 160:82–85.
- Nájera JA, González-Silva M, Alonso PL. Some lessons for the future from the Global Malaria Eradication Programme (1955–1969). *PLoS Med.* 2011; 8(1):e1000412. [PubMed: 21311585]
- NAS. Lead: Airborne Lead in Perspective: Biologic Effects Atmospheric Pollutants. Washington, DC: National Academy of Sciences; 1972. p. 71-177.p. 281-313.
- NTP. Summary. 12. Research Triangle Park, NC: National Toxicology Program, U.S. Department of Health and Human Services; 2011. Report on carcinogens; p. 195
- Prah JD, Goldstein GM, Devlin R, et al. Sensory symptomatic, inflammatory, and ocular responses to and the metabolism of methyl tertiary butyl ether in a controlled human exposure experiment. *Inhal Toxicol.* 1994; 6:521–538.
- Rohter, L. [accessed 12.07.14] With Big Boost from Sugarcane, Brazil is Satisfying its Fuel Needs. 2006. <http://www.nytimes.com/2006/04/10/world/americas/10brazil.html?pagewanted=1&sq=Bush%20Brazil%20ethanol&st=nyt&scp=5&r=0>
- Richie TL, Saul A. Progress and challenges for malaria vaccines. *Nature.* 2002; 415(6872):694–701. [PubMed: 11832958]
- Roberts DR, Laughlin LL, Hsheih P, Legters LJ. DDT, global strategies and malaria control crisis in South America. *Emerg Infect Dis.* 1997; 3(3):295–302. [PubMed: 9284373]
- Seyferth D. The rise and fall of tetraethyl lead. 2. *Organometallics.* 2003; 22:5154–5178.
- Squillace PJ, Scott JC, Moran MJ, et al. VOCs, pesticides, nitrate, and their mixtures in groundwater used for drinking water in the United States. *Environ Sci Technol.* 2002; 36:1923–1930. [PubMed: 12026972]
- Stilwell DE, Gorny KD. Contamination of soil with copper, chromium, and arsenic under decks built from pressure treated wood. *Bull Environ Contam Toxicol.* 1997; 58:22–29. [PubMed: 8952921]
- SCPOP (Stockholm Convention on Persistent Organic Pollutants). [accessed November 2006] 2001. (<http://www.pops.int>)
- Thompson S. *Rural Cooperatives.* 2006; 73:19–21.
- Trembley JF. *Chem Eng News.* 2006; 84(22):19.
- USDA. The biologic and economic assessment of pentachlorophenol, inorganic arsenicals, creosote, vol. 1. Wood Preservatives. Washington, DC: U.S. Department of Agriculture; 1980. p. 193-227. Technical Bulletin No. 1658-1
- Van Rooij JGM, Van Lieshout EMA, Bodelier Bade MM. Effect of the reduction of skin contamination on the internal dose of creosote workers exposed to polycyclic aromatic hydrocarbons. *Scand J Work Environ Health.* 1993; 19(3):200–207. [PubMed: 8367698]



- White MC, Johnson CA, Ashely DL, et al. Exposure to methyl tertiary butyl ether from oxygenated gasoline in Stamford, Connecticut. *Arch Environ Health*. 1995; 50(3):183–189. [PubMed: 7618951]
- WHO. [accessed June 2013] WHO Gives Indoor Use of DDT a Clean Bill of Health for Controlling Malaria. 2006. (<http://www.who.int/mediacentre/news/releases/2006/pr50/en/>)
- WHO. [accessed June 2013] Malaria. 2013. (<http://www.who.int/features/factfiles/malaria/en/>)

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