

Kepone and Hopewell, Virginia: A Toxic Tragedy: 1966-1975

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WRITER'S COMMENT: Environmental toxicology includes studying how chemicals move through the environment and the ways in which they interact with living organisms. Our coursework also covers governmental regulation of potentially toxic substances because this process determine how people are exposed to toxicants. Often, regulations are instated after people suffer adverse health outcomes related to a chemical exposure. In this class, we learned about different toxicants and the tragedies they caused—how people were exposed, the effects on people and the environment, and the role of the government and legal system in the event. We were then asked to write a term paper about a compound not covered in class and to explore the aforementioned topics in the context of our compound. I chose Kepone, a pesticide, because I was completely unfamiliar with its story. In this paper, I wanted to capture how events such as the Kepone disaster are not solely the fault of one person or entity, but rather a series of systemic failures, and that it is through these tragedies that we can learn how better to prevent them.

INSTRUCTOR'S COMMENT: Our society is faced with recurring toxic tragedies in the workplace, in consumer products and in the environment. Often society responds slowly, where resolution may require a century of struggle. A legacy is a strong distrust that citizens exhibit toward social institutions (industry, government), reflected today in bitterly partisan opinions. We see a continuing tension between safety and productivity, between regulation and personal liberty. The tragedy of Kepone provides an egregious example of regulatory mal-

function. We must hope that examining such failures of the market system will help us understand how to improve toxic regulation and to reduce the impact of future tragedies. Can we base our actions on reputable science instead of alternative facts and tribal allegiances? Can we dismiss fake tragedies (e.g., vaccination- and GMO-induced disease) and address impending global climate change? Our survival as a species may depend upon the answers.

— Robert Rice & Matthew Wood, Environmental Toxicology

Toxic environmental disasters have been and continue to be inevitable, given our society's industrial dependence, penchant for corporate exploitation of regulatory loopholes, and underfunding of scientific research on the hundreds of thousands of chemicals in use today. These unintentional chemical exposures have lasting impacts on both human populations and the environment, some of which are never fully understood. One of the most well-known toxic tragedies is the Hopewell, Virginia Kepone disaster, in which excessive amounts of a toxic pesticide, Kepone, were produced and disposed of in ways that put the health of workers, community, and surrounding ecosystems at risk. This paper will explore the causes, circumstances, and consequences surrounding this disaster, as well as how Kepone's chemical legacy influences the present.

Chemical Information

To understand why the Kepone disaster was so impactful, it is important to consider the physicochemical properties that played a role in its distribution in humans and the environment. Kepone is a polychlorinated hydrocarbon similar in structure to dieldrin, dichlorodiphenyltrichloroethylene (DDT), and Mirex (Reich and Spong 1983). Like these well-known pesticides, it is lipophilic—so it bioaccumulates in the fat of exposed organisms (Goldfarb 1978). Because of its lipophilicity, Kepone magnifies up the food chain and can have far-reaching ecological consequences (Reich and Spong 1983; Valo 2013). At room temperature, it is a brownish-grey or white powder (Reich and Spong 1983), and its half-life in soil is 700 years (Valo 2013). These properties indicate that Kepone does not readily leave the body

or degrade in the environment. Therefore, the release of Kepone into the environment and human exposure to Kepone can have long-lasting effects.

Production History

Before commercial Kepone production began in 1949, government research revealed that Kepone was highly toxic in rodents, causing acute neurotoxicity that manifested with spasms, tremors, and loss of coordination, but no other health effects were known or investigated at the time. The U.S. Food and Drug Administration ruled that Kepone could not be used on U.S. food products, so most of it was exported to Europe and South America (Goldfarb 1978). Almost all of Kepone export was to West Germany, where it was a component in the production of Kelevan, a pesticide used in South America for control of banana borer weevils (Reich and Spong 1983). In the U.S., Kepone was considered a technical material because it was used in small amounts in insect traps and lawn and garden products, as well as on tobacco and ornamental fruits (Reich and Spong 1983; NRC 1978). From 1949 to 1975, 1.6 million kilograms of Kepone were produced—a small amount in comparison with other organochlorine pesticides at the time, but significant enough to cause adverse health effects (NRC 1978).

Hopewell, Virginia—a small industrial town that sits on the James River—became home to Allied Chemical Company in 1928. By 1975, Allied was the largest company in Hopewell, employing 4,000 workers (Goldfarb 1978). In the 1950s and early 1960s, Allied contracted Kepone production to Nease Chemical Company in State College, Pennsylvania and Hooker Chemicals and Plastics Corporation in Niagra Falls, New York. By 1966, increasing demand for Kepone resulted in increased production in the Hopewell facility (Goldfarb 1978). Allied was aware of Kepone's reported toxicity, so the company enforced safety precautions at the plant—workers kept spills and dust under control and wore personal protective equipment. However, Allied systematically avoided environmental regulations that had been put in place during its Kepone production years. In 1970, the Army Corps of Engineers reinstated the Refuse Act Permit, requiring reporting and treatment of industrial effluent. Allied was discharging untreated Kepone production wastes into a nearby James River tributary and did not want to pay for

treatment, so the company indicated that the discharge was “temporary” to avoid reporting what was in its effluent (Goldfarb 1978). In 1972, with the amendment of the Clean Air Act to include the National Pollution Discharge Elimination System (NPDES) permit program, Allied lied again, stating that the discharge was temporary. In this manner, Allied successfully evaded regulation and withheld knowledge from the government about the contents of its discharge for 8 years, from 1966 to 1974 (Goldfarb 1978).

In 1974, Allied contracted Kepone production again, this time to allow for increased plastics production at its Hopewell plant. Sensing an opportunity, former Allied executives Virgil Hundtofte and William Moore formed Life Science Products Company (LSP) and won Allied’s bid for Kepone production (Goldfarb 1978). From March 1974 to July 1975, LSP was the world’s only producer of Kepone (Cannon et al. 1978). LSP had its own method for avoiding NPDES permitting: discharging wastes into the Hopewell sewer system with permission from the city, even though the city knew that Kepone could not be processed by their treatment facility (Goldfarb 1978). Along with byproduct waste associated with Kepone production, LSP also dumped “bad batches” of Kepone into the sewer system untreated (Reich and Spong 1983). LSP paid approximately 130 men \$3.75 per hour—good money for a chemical production job at that time—to produce 3,000-6,000 pounds of Kepone per day (Foster 2005), so they were unlikely to leave their jobs, regardless of working conditions.

Kepone production was running smoothly at the LSP plant until July 1975, when an employee reported to the company doctor complaining about weight loss and neurological symptoms such as shaking and eye twitching. Toxicologists often use measures such as parts per million (ppm) and parts per billion (ppb) to describe concentrations of compounds in the air, soil, and human samples. A blood sample from the worker sent to the Center for Disease Control (CDC) had 7.5 ppm Kepone (Goldfarb 1978), an unusually high level. This prompted an immediate investigation at the plant, which revealed unhealthful conditions and lack of protective equipment for workers. According to reports from the investigation, workers walked through “puddles of slurry,” the machinery was “caked with up to one-half inch of Kepone powder,” and 95% pure Kepone dust covered almost all surfaces (Reich and Spong 1983). Many of the workers had complained previously

about symptoms of Kepone poisoning, but the company ignored the complaints, suggesting that they were merely “drunks” (Foster 2005). Finally, 18 days after the investigation, LSP “voluntarily” closed the plant (Goldfarb 1978) and razed it within a few weeks (Foster 2005).

Human Health Effects

Immediately following the closure of the LSP plant, governmental agencies became involved in the process of analyzing the situation's full environmental and human health effects. 28 LSP employees were diagnosed with acute Kepone poisoning (Reich and Spong 1983), some with Kepone blood levels as high as 11.8 ppm (Foster 2005). Workers with acute Kepone poisoning had blood levels of Kepone averaging 2.53 ppm, while those without illness averaged only 0.6 ppm (US EPA 1978). Furthermore, in a study of Allied workers who had been involved in Kepone production before the transfer to LSP, 77% had detectable blood levels of Kepone 18 months after exposure (Cannon et al. 1978), indicating that Kepone was not eliminated quickly from the body. The World Health Organization confirmed this persistence; Kepone is eliminated at an estimated rate of 0.075% of body burden (total chemical in the body) per day (IPCS 1984). It builds up in the liver, whole blood, and subcutaneous fat, and its metabolites are most commonly eliminated through the GI tract (IPCS 1984). Given the lipophilicity of Kepone, it is surprising that adipose tissue levels of Kepone in workers were not measured, as this is where it is likely to be stored in the body. Thus, blood levels of Kepone may be low if it has been a long time since the exposure (Cannon et al. 1978), but it is also now known that Kepone binds preferentially to liver and plasma proteins, which reduces its sequestration into fat (Multigner et al. 2016). Kepone has been found in 2.6-8% of breastmilk samples in North Carolina, Alabama, and Georgia at approximately 1-3 ppb (Suta 1977), but this is likely the result of brief exposure to Mirex, as Kepone is a Mirex degradation product (NRC 1978).

Exposure to Kepone was not limited to the workers. Often, their families were exposed to the dust brought home on work clothing, and many community members were exposed to the contaminated soil near the plant. At the time of the investigation at LSP, there was one ton of Kepone on the ground surrounding the plant, creating soil

concentrations up to 20,000 ppm Kepone (Reich and Spong 1983). One block away, the dust was composed of 40% Kepone by weight, and soil levels in a surrounding neighborhood were as high as 940 ppm (Luellen et al. 2006). However, the long-term health effects of Kepone exposure in the LPS workers and community members remain unclear. 20% of blood samples from Hopewell residents had detectable levels of Kepone, ranging from 5-50 ppb, and residents and workers within a one-mile radius of the plant had blood levels of Kepone as high as 32.5 ppb (Suta 1977). Some studies attempted to link Kepone exposure to congenital malformations, low sperm count, and dysfunctional sperm, but the evidence was not strong enough to prove that Kepone caused these effects (Reich and Spong 1983, Cannon et al. 1978). However, as of 1995, some of the workers were still experiencing tremors that began while they were working at LSP (Foster 2005).

Follow-up studies were not performed on Hopewell community members to determine latent effects of their Kepone exposure, but studies on the Caribbean islands of Martinique and Guadalupe, where inhabitants were chronically exposed to Kepone from 1972-1993, provide evidence of potential effects. Kepone was used to control banana weevil borers on the islands, and though no longer used, it has leached from the soil into the surrounding ocean, so exposure to the islands' populations continues through consumption of contaminated seafood (Valo 2013). Epidemiological studies in these Caribbean populations show that exposure to Kepone increases the risk of prostate cancer and that prenatal exposure is associated with motor skill and visual learning challenges (Valo 2013; Multigner et al. 2016). Kepone was not listed as carcinogenic until 1979, even though it was a suspected carcinogen as early as the 1960s and proven to cause liver cancer in mice in 1976 (Valo 2013; Reich and Spong 1983). Furthermore, in rodents, prenatal exposure to Kepone stimulates uterine and vaginal development through estrogen-receptor-mediated action, increases the likelihood of preterm birth, and causes many developmental challenges (Multigner et al. 2016; Eroschenko and Mousa 1979). Now, it is widely accepted that Kepone is a xenoestrogen—a compound that stimulates the body in a manner similar to estrogen—and a more potent one than o,p'-DDT, one of the most well-known xenoestrogens (Hammond et al. 1979). It is possible that Hopewell community members, especially childbearing women, were not exposed to high enough concentrations over a long enough

period of time for effects of Kepone exposure to manifest there the way they have in the Caribbean, but there are no studies to confirm or reject this hypothesis. A basic principle in toxicology is that the dose of toxin, or toxicant, and the time course of exposure have great impacts on manifestation of toxicity, so those factors may have played a critical role in the differences in observed health outcomes in these two populations.

Environmental Effects

One of the most devastating effects of the Hopewell Kepone disaster was the impact it had on the local ecosystems and fishing industries. Over the nine years of Kepone production in Hopewell, about 100,000 kg of Kepone were released into the environment (Luellen et al. 2006), and a frozen fish library showed that Kepone contamination in the James River dated to 1967, proving that Allied dumped Kepone and other toxic wastes illegally into the river well before LSP began production (Foster 2005). The U.S. EPA did not actively mitigate the contamination—most Kepone was bound to sediment, not dissolved in the water—so they let the Kepone become buried in the riverbed through the process of sedimentation (US EPA 1978; Luellen et al. 2006). By the time the situation was discovered, much Kepone effluent had already spread downstream on suspended sediment and in fish, resulting in contamination as far away as the Chesapeake Bay and southern parts of New York State (US EPA 1978; Reich and Spong 1983).

Kepone levels in James River sediment are still of concern today. Installation of a new powerline near Hopewell sparked a debate about whether it should be buried, risking disturbing Kepone-contaminated sediments, or constructed as an above-ground eyesore. Those in favor of burial cited a 1990's dredging event in which Kepone was not found in sediments, but others doubt that it could have degraded or dissipated that quickly given its extremely long degradation half-life (Dietrich 2005). Either way, the debate is a primary example of how environmental disasters can impact a community decades after the original contamination event.

In December 1975, the U.S. EPA and State Water Control Board (SWCB) reported high levels of Kepone in the James River, so the governor of Virginia banned finishing for the 80 miles of the river from Hopewell to the Atlantic Ocean (Reich and Spong 1983). This ban remained in place until 1982, when some restrictions were lifted; fishing was not

restored completely until 1989 (Luellen et al. 2006). Many fishermen sued Allied, LSP, and the City of Hopewell, resulting in the payment of millions of dollars in damages (Reich and Spong 1983). Oystermen were particularly affected and won \$1.2 million for damages to oyster seeding grounds (Reich and Spong 1983).

Environmental effects were not limited to aquatic organisms. The widespread distribution of Kepone-contaminated fish meant that animals that eat fish were exposed to Kepone. This was true in the case of Chesapeake Bay bald eagles, which had Kepone blood levels as high as 83 ppm in the early 1970s (US EPA 1978). Some suspect that Kepone played a role in the famous bald eagle eggshell thinning associated with DDT at the time (Foster 2005). DDT's effect on eggshell thinning was thought to be mediated through the estrogen receptor. The pivotal study that implicated DDT in eggshell thinning did not include Kepone, and it is now known that Kepone is an estrogenic compound, so it would be plausible for Kepone to have played a role in that phenomenon.

Responsibility, Consequences, and Legacy

It is indisputable that the majority of the blame for the Kepone disaster belongs to Allied Chemical Company and Life Science Products. These companies purposefully evaded regulatory laws and permitting systems put in place to protect people and the environment, with the knowledge that their actions could be harmful. Allied and LSP were indicted three times on hundreds of counts relating to lack of reporting on discharges and lying to the government; perhaps surprisingly, they pleaded not guilty (Goldfarb 1978). At the end of the proceedings, Allied was fined \$13.2 million; LSP \$4 million; Hundtofte and Moore \$25,000 each; and the City of Hopewell \$10,000 (Goldfarb 1978). These fines, however, were hardly significant given the financial statuses of the companies and individuals as well as the degree of environmental damage caused by the disaster. LSP was financially unable to pay its fine, and the actual cost to Allied was about \$4 million after various amounts were deducted from the fine, such as funds for the creation of the Virginia Environmental Endowment (Reich and Spong 1983, Foster 2005). However, former employees successfully sued Allied and Hooker Chemical Company, winning a total of \$173 million in damages (Reich and Spong 1983)—a much more impactful amount than the small fine

initially imposed by the courts.

Despite the fact that Allied and LSP were the entities most responsible for the Kepone disaster, the U.S. EPA and Occupational Safety and Health Administration (OSHA) also failed in many regards. The EPA never investigated the “temporary” discharge at Allied even though it had been filed as such for years, and they spent six months debating whether Kepone was a pesticide product or component, delaying much needed action on the situation (Reich and Spong 1983). A terminated worker filed a health complaint with OSHA in 1974, but OSHA failed to investigate LSP because of a “procedural error” (Reich and Spong 1983; Foster 2005). Even though inspections were not required at the time, it would have been prudent to do so following employee complaints about conditions.

Although many did not consider the financial punishment for Allied and LSP to be adequate, the Kepone disaster did contribute to the creation of many important regulatory laws. It stimulated an OSHA-approved occupational safety and health plan, and it was the catalyst for the passage of other Virginia environmental regulations (Cannon et al. 1978). The Virginia Environmental Endowment, created with a portion of Allied’s fine, is still around today and promotes pollution control (Foster 2005). Less directly, the disaster contributed to the passage of the Toxic Substances Control Act in 1976 and the EPA Superfund in 1980. Today, Wonder City Motors occupies the former LSP office space. Hopewell has a new sewage system, and the local government is actively involved with monitoring the town’s chemical plants in the hope of preventing future toxic tragedies (Foster 2005).

While there do not seem to be any lasting health effects from the Kepone exposures in Hopewell, or extremely noticeable present-day impacts on the James River ecosystem, this disaster serves as an important lesson in how lack of regulatory guidelines and enforcement can result in undesirable chemical releases. Through the passage of the aforementioned regulatory laws, Kepone’s legacy can be viewed in a positive light, though these laws are far from perfect. Toxic tragedies are events that our society still faces on a regular basis, and they may never completely cease occurring. What is most important is that we learn from each of these events as much as we can and apply that knowledge and experience to better handle future environmental disasters.

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