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Is Agent Orange a Poison? : Vietnamese Agent Orange Litigation and the New Paradigm of Poison

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> All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.
>
> Paracelsus

INTRODUCTION

On March 30, 2005, after just over a year of proceedings, Judge Jack B. Weinstein of the Federal District Court in the Eastern District of New York dismissed the case *The Vietnamese Association for Victims of Agent Orange/Dioxin v. Dow Chemical et al*. (hereafter *VAVA v. Dow et al*.)[1] This result was disappointing for many and infuriating for many more.[2] Some insinuated there was political pressure from the US government behind Weinstein's decision (e.g., Moto 2008). But within Weinstein's statement of dismissal, perhaps, what vexed these commentators most was his

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insistence that Agent Orange and the other chemical herbicides used by the US military in the Vietnam War "should be characterized as herbicides and not poisons" (Weinstein 2005, 60).

Was Agent Orange a poison? Except for a few industry lobbyists such as Michael Gough (1997) and legal technicalists such as Judge Weinstein, most of us, I think, would probably now respond with a resounding yes. Deployed by the US military and its allies as one of the defoliants to remove forest cover of the Viet Cong guerrillas, in its heyday, Agent Orange and America's herbicidal warfare, code named Operation Ranch Hand, gave birth to a new concept of war crime called "ecocide," or ecological genocide (Galston, in Knoll and McFadden 1970, 71–72; Zierler 2011). Agent Orange and its contaminant, dioxin, were later known to have caused–and continue to cause–many illnesses, including cancers and birth defects, among the exposed population.

For most of us fed by these popularized images of Agent Orange, Weinstein's nit-picky distinction between an "herbicide" and a "poison" may appear as a ridiculous, but innocuous, rhetorical point. But for the Vietnamese plaintiffs the verdict that Agent Orange is not a poison meant more. Clad in the protective shield of a "government contractor defense" (an argument that goes, "because the government made us do it, it is not our fault") the defendants, such as Dow Chemical and Monsanto, were already virtually immune to ordinary product liability laws. The "not a poison" verdict made them more invincible by effectively barring the plaintiffs' principal allegation that they committed a war crime by supplying the US military with a poison during the Vietnam War.

"Agent Orange, not a poison"—a "hairsplitting" argument indeed![3] But my confidence wanes if I consider whether it was a poison during the time it was in use in Vietnam. In the 1960s, just as the US military was beginning to spray Vietnamese forests with Agent Orange and other chemical herbicides, the idea of what is a poison and the field of toxicology was going through a transition. Under the new paradigm of poison, dosage was still important in understanding the consequences of poisoning; but it no longer differentiated a poison from a remedy. Beneath the threshold of lethal dosage and acute poisoning, the world discovered previously undetected risks of various insidious and horrendous diseases, such as cancers, autoimmune diseases, and birth defects. In the United States, Rachel Carson's (1962) *Silent Spring* was instrumental in publicizing this new threat. Synthetic chemicals such as DDT and the component chemicals of Agent Orange, 2,4-D and 2,4,5-T, and the latter's

contaminant, dioxin, accumulated in the food chain and human bodies and insidiously poisoned the population. This type of poisoning could have latency of up to several decades, and the primary concerns, as would be chanted in the 1970s, were their *carcinogenic, teratogenic*, and *mutagenic* potential (e.g., Galston 1971). What complicated the issue was that these "poisonous" substances were often unavoidable contaminants or active agents of chemical pesticides and pharmaceutical products, which also brought many benefits to society so that it was not possible to outright reject them. In this context, how to define and assign accountability for the damages and risks incurred by toxic substances such as chemicals and radiation became an important scientific, legal, and sociological question (Jasanoff 1997).

In the field of Science and Technology Studies one of the common ways to approach toxic tort litigation is to look at the use of scientific evidence of causation in court (Jasanoff 1987, 1997). Since the 1980s, in the United States, the awareness that courts need to reckon with increasingly sophisticated and controversial scientific evidence has led to various studies that try to bridge the fields of science and law (Berger 2003, 2005; Berlivet 2005; Caruth and Goldstein 2001; Eaton 2003; Jasanoff 1992, 2006). Toxic tort litigation is particularly "science heavy," because ordinarily the scientific evidence of causation linking plaintiffs' injuries to a specific chemical is often at the heart of the dispute (Jasanoff 1997). In earlier Agent Orange litigations filed by US veterans of the Vietnam War in the 1980s, this question of evidence was important (Schuck 1986). However, in the Vietnamese Agent Orange litigation, the question of causation never became the main point of contestation; the plaintiffs' claims were barred from proceeding further before the litigation reached this stage. In the Vietnamese case, the main issue was the applicability of international law that prohibits the use of poison in war. And the bone of contention was over whether or not Agent Orange was categorically a poison. The intention and knowledge of the manufacturers and the military purportedly answered this question.

In this article, I analyze the implications of the historical shift in the notion of poison in the United States in the 1960s for the question of legal responsibility in the context of *VAVA v. Dow et al*. First, I discuss how the Vietnamese plaintiffs' attorneys were boxed into making a rather strained argument that insisted on there being a malicious intention *embodied in* the substance itself (Agent Orange) while suspending the question about the US military's intent to poison. Second, I discuss how the possibility of

knowledge of a toxic substance changed, in particular, in the United States, giving birth to the concept of an intentionless "risky" poison.[4]

LEGAL QUAGMIRE

In the winter of 2004, a group of Vietnamese who claimed to have been poisoned by Agent Orange brought a class action lawsuit to Judge Weinstein's court in Brooklyn.[5] The defendants were a cast of familiar players in controversies over agrochemical risks and included high-profile multinational corporations such as Dow Chemical and Monsanto. The plaintiffs claimed that they suffered from the toxic effects of Agent Orange and other chemicals that the defendants manufactured for the US military during the Vietnam War and that the defendants were guilty of "aiding and abetting" violations of international law that banned poison in war.[6]

VAVA v. Dow et al. attracted much attention from peace activists, Vietnam veterans, scientists, and international legal scholars around the world. While different people were interested in different aspects of this case, legal scholars were particularly interested in the use of the US Alien Tort Statute (1789), which was one of the key legal instruments under which the lawsuit was filed (Sebok 2005). The Alien Tort Statute provides that US courts have "jurisdiction of any civil action by an alien for a tort only, committed in violation of the law of nations or a treaty of the United States" (cited in Bederman 2001, 107). Although the law was passed in 1789, it lay largely dormant until the landmark case of *Filártiga v. Peña-Irala* in 1980, in which the defendant was brought to US courts to be tried for the torture and murder of Joelito Filártiga, while the defendant was the inspector general of police in Asunción, Paraguay. Since then, the Alien Tort Statute has been invoked in a number of human rights cases (including *Sosa v. Alvarez and Doe v. UNOCAL*) in which grievances were filed in US courts by foreigners over violations of international humanitarian laws by US corporations that took place outside the United States. In recent years, Corporate America, whose business practices in developing countries sometimes border on human rights violations, has paid a large amount of attention to these cases (Sebok 2005, 2).[7] The outcome of *VAVA v. Dow et al*. was closely followed as it was thought to have a bearing on future cases involving the Alien Tort Statute.

VAVA v. Dow et al. was also, in a sense, a new case dealing with an old issue for US courts, which had had experience with similar lawsuits filed by Americans who also claimed to have been injured by Agent Orange.

Toward the end of the 1970s, the word began to spread that the veterans were suffering from health problems due to their exposure to herbicides during the war (Wilcox 1989). At that time, the Veterans' Administration had no special provision for Agent Orange-related illnesses (Institute of Medicine 1994). It was also around this time that the toxic effects of dioxins became internationally publicized through toxic disasters at Seveso, Italy; Love Canal, New York; and Times Beach, Missouri (Allen 2004). Scientists had also come to a better understanding of various pathogenic processes associated with dioxin exposure, such as carcinogenic and teratogenic effects (Van Miller et al. 1977; Kociba et al. 1978), and the mechanisms through which dioxin caused these toxic effects in animal cells (Poland and Glover 1973).

In 1978 several veterans began to bring their individual cases to court. Over the next four years, the lawsuits (which took the chemical companies as the defendants) grew in scale, involving over 600 separate lawsuits and 15,000 litigants (Novey 1988). These cases were consolidated into one class action lawsuit in 1983 under Judge Jack B. Weinstein in the Eastern District of New York (the same judge who would preside over the Vietnamese case two decades later). The veterans' Agent Orange litigation enjoyed an enormous public visibility, and in 1984, the veterans reached a $180 million settlement with the chemical manufacturers (Schuck 1986).[8]

Despite their ostensible success, many veterans were left disgruntled by this outcome. "To many of the veterans," a legal scholar, Peter Schuck (1986, 255), wrote, "the case was a morality play performed on a stage—the court." But the technical aspect of the case contradicted the intuitive understanding of the veterans, who were left with the impression that the law was "profoundly mystifying, alien, and unjust" (256). For one thing, the settlement was made without the defendants' admission of guilt. Furthermore, the central question about whether Agent Orange actually caused the veterans' illnesses remained unsettled (Weinstein 2009). In fact, in the opinion of Weinstein, the issue was ever murkier in 1984.

Following the settlement of the class action suit in 1984, the veterans who had opted out of the class action suit began to bring individual suits. During this second wave of veterans' Agent Orange litigation, Judge Weinstein dismissed all cases on the ground that the claims were covered by the class action suit and that these new plaintiffs failed to submit "acceptable evidence of causation" (Novey 1988). Twenty years later, Weinstein (2009) was quite ready to dismiss the Vietnamese case, as well as more recent cases filed by the Americans, on the same ground of lack of

evidence of causation, if it got that far.

The Vietnamese Agent Orange litigation was beset with many difficulties from the beginning. Shortly after the Vietnamese filed their lawsuit, the veterans' cases, often referred to as the Isaacson and Stephenson cases, were dismissed under the government contractor defense. It was inevitable that this decision would affect the Vietnamese class action case.[9]

VAVA v. Dow et al. was dismissed in March 2005. As expected, the principal argument in favor of the defendants was the government contractor defense. This gave the manufacturers of the chemicals virtual immunity by exempting them from legal liability for the consequences of Agent Orange, *except*—and the statute came with a clause, reiterated by Judge Weinstein—in cases where violations of international laws were recognized.[10]

Having dismissed the plaintiffs' domestic tort (product liability) claims under the government contractor defense, Weinstein moved on to examine their international law claims. The list of international laws that the plaintiffs accused the defendants of violating included: torture, war crimes, crimes against humanity, environmental law violations, and even genocide. Weinstein rejected all of these claims. Citing fifteenth-century "father of toxicology" Paracelsus's dictum that dose differentiates a poison from a remedy (Weinstein 2005, 59), the judge argued that whether or not Agent Orange was a poison hinged on the "*design and degree*" (in other words, the "intention and dosage").[11] Thus, while dioxin, which is a *contaminant* of Agent Orange, is a poison, Weinstein argued, Agent Orange, which contained on average 10 parts per million of dioxin, is not a poison. And insofar as Agent Orange is not a poison, its use in Vietnam did not violate the customary international law prohibiting the use of poisons in war. At least in part, therefore, this litigation was a contestation over the binary categorical identity of the *chemical substance* as poison or not and the subjective intentions that defined it. The question was: Whose intention and whose design determines the nature of a substance as a poison? And what does "intention" mean here?

In legal discourse, "intention" has a meaning somewhat different from the ordinary use of the term. *Mens rea*, or guilty mind, for lawyers lies on a continuum from *intent, knowledg*e, and *recklessness* to *negligence* (Khanna 1999, 357). A desired consequence with full awareness of the situation is *intended*. *Knowledge* entails awareness of practical certainty that a certain consequence will follow an act. *Recklessness* is a conscious disregarding of a substantial and unjustifiable risk. And *negligence* is a failure to

exercise due care. While *mens rea* normally refers to the subjective state of mind rather than an objective condition, in the absence of a confession, one must infer a subjective state of mind from the objective situation. On one extreme of the continuum, when the expected outcome is thought to be "objectively" inevitable, it is inferred to be desired and thus *intended*.

Ordinarily, negligence, for example, provides a case for product liability tort. But where, on this continuum, does mens rea of the manufacturers of Agent Orange fall? Agent Orange may have caused serious health consequences. Yet, ostensibly at least, the *purpose* of its use by the US military was to defoliate forests and destroy enemy crops rather than to harm human beings. Insofar as the military was concerned, therefore, the toxic health effects of Agent Orange were *unintended* collateral effects, and if the issue is about the unintended side effects of herbicides, it does not fall under the purview of international law.

After the dismissal of *VAVA v. Dow et al*. in 2005, the plaintiffs' subsequent appeals to the Court of Appeals for the Second Circuit and the Supreme Court of the United States were trapped in increasingly tortuous and convoluted arguments concerning international laws and the intentionality of the manufacturers. In the district court, Judge Weinstein made it clear that the criminality of an act of the magnitude of "crimes against humanity" must be accompanied by intentionality of the actors. As far as he was concerned, exposing the plaintiffs to a chemical substance that may have *inadvertently* posed health risks and injury was the result of negligence or recklessness; it was not the same as intentional poisoning. Thus, in the *writ certiorari* submitted to the US Supreme Court, the plaintiffs' attorney largely skirted around the question of intention of the US military, stating that they "nowhere allege that the *government* intended to harm human beings through its use of Agent Orange,"[12] and entreated the court to "*infer an intent* to poison" by the defendants' knowledge of this "very potent poison [dioxin]" in Agent Orange, which was to be "sprayed over vast populated areas."[13] Even if the toxic effects of Agent Orange suffered by the plaintiffs were an "unintended consequence" of spraying herbicides designed to defoliate forests as far as the US military was concerned, they argued, "at least insofar as the chemical company respondents are concerned," it was *not* an unintended consequence. Ultimately, the issue came down to the question of knowledge *not* so much of the *etiology* (cause) of the illnesses plaintiffs complained of, but a categorical question about whether Agent Orange was a poison or not, which, the plaintiffs' attorneys argued can be inferred

from the manufacturers' knowledge of the toxicity of Agent Orange at the time of procurement.

WHAT WAS AGENT ORANGE?

Agent Orange has been called a defoliant, an herbicide, an "anti-plant" agent, a toxic chemical, and even a chemical weapon. In the 1950s, the component chemicals of Agent Orange—2,4-D and 2,4,5-T—were hailed as new and promising weed killers. The newspaper ads from that period describe them as "The Weed-Killing Miracle: New and Improved Wee-Done"[14] and as "The Modern Methods"[15] in the "War on Weeds."[16] But by 1971, when its use in Vietnam as a military herbicide was finally terminated, this mixture of "miracle herbicides" had fallen from grace. It was given the sinister name "Agent Orange" (Newton and Young 2006, 40), which was immediately associated with "fetal malformation."[17] It was accused of being a chemical weapon,[18] an agent of "ecocide,"[19] and a weapon comparable to the atomic bombs dropped on Hiroshima and Nagasaki.[20] How did these seemingly benign and beneficial herbicides used in agriculture and forestry become such terrible, wicked, evil things in the span of merely two decades? The answer to this question lies not with the changing nature of the chemicals themselves, nor with the changing manner of their use (although the scale of use of these chemicals in Vietnam was enormous). It was the changing context of scientific knowledge and moral values that led to this change in the ontology of the chemicals that had come to be known as Agent Orange.

Agent Orange was not *meant* ("*designed*" in Judge Weinstein's language) for *all* the evils it was later accused of committing. In this sense, it was unlike any other weapon of mass destruction. From its birth, the atomic bomb, for example, was meant to be a lethal weapon, designed to cause indiscriminate destruction. The birth and "maturation" of Agent Orange, on the other hand, was not so straightforward. Born in 1941 in the laboratory of an industrial chemist, Robert Pokorny (1941), it was developed further in the Chemical and Biological Warfare Division of the US Army during World War II, mainly for the use in the Pacific front (Cecil 1986). In postwar America, the component chemicals of Agent Orange, 2,4-D and 2,4,5-T, became popular herbicides used in agriculture and forestry. Agent Orange was a military adaptation of these commercial herbicides.

This does not mean that there were no signs of their toxic side effects

in the early years. The signs of human toxicity of 2,4-D and 2,4,5-T were available to the chemical companies from the 1940s. Some of the earliest evidence came from the clinical cases of factory workers. In 1949, for example, the workers at Monsanto were exposed to the chemical in an accident involving a runaway reaction at the factory producing 2,4,5-trichlorophenol (precursor of 2,4,5-T). The workers initially complained of a burning sensation in the eyes, nose, and throat. Subsequent symptoms included nausea, vertigo, headache, vomiting, disturbed sleep, nervousness, loss of libido, impotence, high cholesterol, raised blood fat levels, unexplainable pain, and extreme fatigue, *plus* pustules and pigmentation of the skin. This latter skin condition was called "chloracne" (Hay 1982, 98).

Chloracne was no ordinary acne. A doctor at Diamond Alkali chemical plant, where an outbreak of chloracne occurred among workers in 1954, commented on "how disfiguring this disease is and what a social disability it is."[21] In 1956 a physician working with a patient from Boehringer Company in West Germany discovered that chloracne was caused by a contaminant of 2,4,5-T, called tetrachlorodibenzo-p-dioxin (Hay 1982). Scientists there subsequently developed a method to reduce dioxin impurity in 2,4,5-T, and this knowledge was shared with Dow Chemical in 1957. Thus, by the time Dow began to produce 2,4,5-T for the US military in the 1960s, its scientists had known for some time about the problem of chloracne and dioxin contamination of 2,4,5-T, as well as the technique to reduce this contamination (which they shared with other US manufacturers in 1965).[22] Was this not evidence enough to conclude that the manufacturers knew how toxic 2,4,5-T was?

One of the points of contention in Agent Orange litigation was the discrepancy between what the manufacturers knew and what the government knew. Here, the American plaintiffs' counsel and the Vietnamese plaintiffs' counsel differed as well. The attorney for the American plaintiffs focused on how the manufacturers had concealed much of the information on the toxicity of 2,4,5-T from the government and the military. They thought it important that the US government may not have "selected 2,4,5-T at all if the manufacturers [had] told them about the [dioxin] contamination."[23] If the government had unwittingly purchased a defective herbicide, the government contractor defense would not protect the defendants.

The Vietnamese plaintiffs, on the other hand, characterized the chemicals as military herbicides, equivalent to chemical weapons. If, as

Dr. James R. Clary of the Chemical Weapons Branch of the Air Force testified, the military knew that "the 'military' formulation [of 2,4,5-T] had a higher dioxin concentration [than the commercial version]" but accepted it "because the material was to be used on the 'enemy,'"[24] then it would be possible to characterize Agent Orange as a poison weapon (rather than just one version of a commercial herbicide). In their arguments, the veterans' acquitted the government thereby placing all blame on the manufacturers. The Vietnamese plaintiffs, on the other hand, accused the manufacturers of *complicity* with the US military, the principal agent in committing the war crimes.

However, one issue that did not get elaborated in these litigations was the fact that there was considerable difference between the symptoms and diseases associated with Agent Orange and the toxic effects of 2,4,5-T as it was known before the war. If one compares symptoms reported in association with 2,4,5-T before and after the Vietnam War, *chloracne*, *porphyria cutanea tarda* (skin symptoms), and *peripheral neuropathy* were the only conditions that persisted in the post-Vietnam War literature on Agent Orange (see Hay 1982). Many of the symptoms observed in cases of occupational exposure in the production process (such as "nausea, vertigo, headache, vomiting, and unexplainable pain and extreme fatigue") were acute conditions or could be mistaken for psychosomatic effects. Reports of these acute conditions virtually disappear from the scientific literature on the effects of Agent Orange in the postwar era (Institute of Medicine 1994). In fact, in a brief submitted to the court in *VAVA v. Dow et al*., the US government admitted that it knew about dangers of these chemicals as much as (or more than) the manufacturers did, although this knowledge was limited to "chloracne and certain forms of liver damage."[25] More serious diseases such as cancers and birth defects were suspected only after Agent Orange came in use in Vietnam.

In 1969 the understanding of Agent Orange shifted radically when evidence of teratogenic effects of 2,4,5-T on mice reached the public. This information was discovered in a general pesticide screening that was commissioned by the National Cancer Institute in 1964 (Advisory Committee 1971). In June 1966, preliminary results had already indicated the teratogenic nature of 2,4,5-T. However, the result was not delivered to the National Cancer Institute for two more years. It took a further year of bureaucratic dawdling before the information was leaked to a public advocacy group known as Nader's Raider's, who succeeded in putting

pressure on the White House to release the information in October 1969 (Wade 1971).

Within a month, it was reported in the United States that in the summer of 1969 "several South Vietnamese newspapers [had] printed photographs and stories about deformed South Vietnamese babies" (Nelson 1969). People now began to talk about Agent Orange, which was suddenly "discovered" to be contaminated with "one of the most poisonous substances ever created," dioxin.[26] Its primary crime was remembered to be its terrible effect on human health. Previously, when what was evil about Agent Orange was the *military* use of an herbicide, it was the *scale* of its use that mattered. Once malformation of fetuses was noted, however, the *nature* of the chemical itself—and by proxy, the chemical manufacturers—became the object of criticism.[27]

NEW PARADIGMS OF POISON

"But why," as Bryce Nelson (1969, 979), a reporter for the journal Science, wondered at the time, "were these herbicides allowed to be widely used in Vietnam before scientific studies on animals had been performed?" In 1969 it appeared as though there had been a complete oversight in testing these chemicals for safety. Why had this happened?

An easy answer is that before the 1960s, teratological testing of pregnant animals was not part of the routine procedure for product-safety testing. The government agencies had established no guidelines for it, and few scientists even considered doing it (Wilson and Warkany 1985, 293). But this is only part of the answer. In fact, ever since 2,4-D and 2,4,5-T were invented, scientists had been conducting their own safety tests. The question is: What kind of tests? Here are some examples of the toxicological tests performed before the 1960s:

- In the 1940s, Ezra Kraus at Fort Detrick's Chemical and Biological Warfare Division experimented with 2,4-D o*n himself*, ingesting 0.5 grams of the substance per day for three weeks. He found no ill effects (Butler 2005, 539).
- In 1946, Nancy Bucher, a medical researcher at Harvard, looked at the effects of 2,4-D on mice with sarcoma and found no effects (Butler 2005, 539).[28]
- In 1953, scientists at Dow Chemical tested 2,4,5-T on dogs. They found that 2,4,5-T was lethal for dogs at the dosage of 20 mg per kg

of body weight per day, but that at lower dosage the animals did not develop "significant lesions in the liver or other organs" (Butler 2005, 540).

- In the mid-1950s, Karl Schultz at Boehringer Company performed rabbit ear tests on the precursor of 2,4,5-T, 2,4,5-trichlorophenol. He found that a pure sample of trichlorophenol did not cause inflammation in rabbit ears, but the sample contaminated with dioxin did. He concluded that it was dioxin that caused the skin condition called chloracne in humans. Schultz also *tested the sample on his left arm* and found that it induced the same skin lesions as was seen in factory workers exposed to 2,4,5-T (Butler 2005, 540).

Note that except for Schultz's experiment these tests came to the conclusion that 2,4-D and 2,4,5-T were safe (at least at the dosage factory workers and farmers were exposed to). Whether these assessments were made in good faith or not, one can observe how crudely and carelessly these tests were designed and conducted. For instance, none of these studies tested for long-term health effects and reproductive effects, which they were later suspected to have. Kraus and Schultz were even reckless enough to test the chemicals on themselves.

This lack of concern for latent health effects was not entirely surprising, given that until the mid-1960s, long-term effects of low-dosage exposure to these chemicals in relation to cancer, for example, were not included in product-safety testing. Also, before 1960 "the *concept of* searching for embryotoxic/fetotoxic effects after *in utero* exposure and the application of any adverse findings to estimating human teratological risk simply had not evolved" (Wilson and Warkany 1985, 293). Ironically the critical events that altered the awareness of both scientists and the public occurred right around the time the US military began to use herbicides in Vietnam.

The year 1962 was an eventful one. Besides the beginning of the US herbicide program, Operation Ranch Hand, it was also the year that the thalidomide crisis exploded in countries in Europe and Japan. This crisis involving iatrogenically induced birth defects caused by the morning sickness drug, thalidomide, soon became proverbial. "Must we wait for definite proof of an abnormal birth before we are prepared to act? Have we learned nothing from the thalidomide tragedy?" asked Arthur Galston (1970), a scientist advocating a ban on 2,4,5-T in 1970. The thalidomide crisis had become a premonition for future disasters involving poorly tested chemicals.

The thalidomide crisis also played its part in altering the field of toxicology in the 1960s. Decades later, teratologists James Wilson and Josef Warkany (1985) recalled that it was this disaster that gave momentum to the teratologists who were beginning to organize themselves into its own discipline. Teratology, or the study of congenital malformation, was a relatively old discipline. By the eighteenth century, for example, teratology was a flourishing field in France, where scientists typically created multiheaded hydras by making incisions on the bodies of microorganisms or tried to create new species by changing their environment (Dally 1998). It was literally a *terato* (monster)-*logy* (study): a study of monster breeding. In these earlier days, most of the experiments were conducted on invertebrates; it was not until the 1930s that teratologists began to study mammalian fetuses. Even then, the focus was on *producing* malformation rather than protecting populations from teratogenic agents.

Chemical mutagenesis treaded a similar path. Scientists began to study the mutagenic effects of chemical substances in the post-World War II era (Frickel 2004). Scientists such as Charlotte Auerbach, a geneticist at Edinburgh, tested substances like mustard gas for mutagenic effects, but these studies on chemical mutagens soon went out of favor in her field. Geneticists were interested in studying *mutations* for basic research on genetics, and radiation, which could produce mutation at a more controlled rate, was a more convenient tool for research than the chemicals, whose ability to produce mutation was unpredictable. It was only toward the end of the 1960s that some geneticists began to express concern about the various synthetic chemicals and their effects on the genetics of the human population. They called these chemicals, "environmental mutagens."

The Environmental Mutagenesis Society was established in 1969, and the First International Conference on Environmental Mutagenesis was held in 1973 (Bendix 1974, 188), which indicates the importance of these decades in the development of the idea of chemical mutagenesis. Participants at the first environmental mutagenesis conference expressed astonishment that so many new chemicals were introduced to the market without proper testing for "mutagenicity, carcinogenicity, or teratogenicity" (188).

In the 1970s this new discourse on toxic substance was also entangled with a prophetic discourse of "epidemiologic transition" (Omran 1971). Throughout the 1960s, there was a gradual increase in awareness among public health experts that the diseases of foremost concern were no longer infectious diseases but "degenerative and man-made diseases" such as

cancers and autoimmune diseases (516). These man-made diseases were often attributed to new synthetic chemicals.

It was also in 1962 that Rachel Carson published *Silent Spring*, which soon became a national sensation in the United States. Carson brought together previously scattered knowledge in ecology, chemistry, medical science, and politics into a comprehensible form that discussed invisible pollution that threatened the "livable" environment, and gave agency to this impending ecological catastrophe. Chemicals such as DDT, benzenes, and dioxins, now known to be ubiquitous in the environment (Christie and Tansy 2004), were linked with the nation's growing preoccupation with cancer and birth defects. The common characteristics shown by these environmental poisons were that they were everywhere and persistent; they accumulated in the food chain; and their toxic effects could become manifest years after exposure.

In the 1970s, an air of "doom and gloom" pervaded the discourse of environmentalism.[29] The chemical industry was releasing thousands of "unwanted and even unidentified substances" each year (Abelson 1970, 495). Like merchandise streaming out of automated factories, these chemicals defied attempts to locate the intention. Operating beneath the threshold of detection, insidiously but surely, these new poisons were spreading; and since the exposure to these poisons did not necessarily result in any harmful effects but rather they were merely associated with an elevated *statistical rate* of certain diseases *in a population*, the experts call these poisons "risk factors." These "statistical" poisons rewrote the definition of "poison" in a radical way, giving rise to new concerns, sensibilities, and responsibilities.

Unlike the moral condemnation of poisoning of the previous age (Price 1995; Whorton 1974), the statistical risk posed by poison of our age led to a bifurcated response. Risk-benefit comparisons involve a comparison of incommensurable factors (Beck 1992). In the case of Agent Orange in the 1960s, this comparison was between the chance of immediate survival of American soldiers (ostensibly increased by the use of Agent Orange in defoliation) versus the long-term hazards of the chemicals on the health and environment of the Vietnamese people. Once the war in Vietnam was over, the struggle over 2,4,5-T was brought home to the United States in the form of a regulatory war over domestic commercial herbicides. The question became the economic benefit of using 2,4,5-T (for the chemical industry and for agriculture and forestry) versus its effects on long-term health and survival of the Americans.

The ontological question about Agent Orange was firmly wedged within this new discourse of environmental risk. With the emergence of this new paradigm of poison, the moral order and the legal structure dealing with accountability for suspected damages also had to change. Judge Weinstein (2009), for example, argued that "mass toxic tort," which seeks to deliver justice based on general risk and negligence (rather than particular causation and intention), was one of the legal traditions that developed within this context (Jasanoff 1987; Schuck 1986). In these toxic tort cases, court decisions effectively function as retroactive risk regulations, in which "preventive effects depend on potential injurers extracting appropriate signals from what the courts do and modify[ing] their behavior" (Galanter 1994, 135). International laws, however, still seems to work with the older notion of poison.

CONCLUSION

In this article, I argued that the characterization of Agent Orange as a poison, which was crucial for the plaintiffs' international law claim, was not as self-evident before the 1960s as it may seem now. This was because in the 1960s, a new paradigm of poison emerged, which rendered the toxic effects of Agent Orange visible. Poisons were no longer defined by an intention to do harm. Nor did they turn into remedies when applied below a certain dosage, as the classical toxicological dictum held. This transition happened in the 1960s, just as the US military was spraying Vietnamese forests with Agent Orange. This timing, I believe, was important for thinking about the litigation. The plaintiffs in *VAVA v. Dow et al*. invoked the international law that operated with the pre-1960s paradigm of poison, defined by the intention behind its design or those who deployed it. But the poison they complained about was only a *risk factor*, which came to dominate public discourse in the 1970s.

On March 2, 2009, the Vietnamese plaintiffs' five-year long quest for justice in the US courts came to an end when the US Supreme Court declined to review the lower court's decision. It was not altogether an unexpected outcome. One Vietnamese involved in the litigation stoically accepted the difficulty in finding justice in the US courts and contented himself that, at least, the litigation drew the world's attention to the problem in Vietnam and put pressure of the US government to deal with the problem of dioxin in Vietnam through other means.[30]

In the past few years, Monsanto, the world's largest producer of

genetically modified seeds (and also one of the defendants in the Agent Orange trial), has been gearing up to expand its business in selling seeds to Vietnam.[31] So far, the company has evaded its responsibility to compensate the victims of Agent Orange in Vietnam. Nevertheless, the leadership of the Socialist Republic of Vietnam seems quite willing to allow Monsanto to "return" to Vietnam. As with many state-led projects in Vietnam, opposition to this move is quickly silenced. On a positive note, the presence of Monsanto in the country may enable the victims of Agent Orange in Vietnam to sue the company under Vietnam's own legal system.

NOTES

[1] V*ietnamese Assoc. for Victims of Agent Orange/Dioxin v. Dow Chem. Co*., MDL No. 381, 04-CV-400 (EDNY).

[2] See, for example, Moto 2008.

[3] "A Non-Lawyer's Guide to Judge Weinstein's Agent Orange Decision," Warlegacies.org, accessed August 15, 2012, http://www.ffrd.org/Lawsuit/AO%20decision%20analysis.htm.

[4] In this article I use legal documents, scientific articles, and news reports on the toxicity of Agent Orange, as well as secondary literature on epidemiology, international law, and the history of the use of Agent Orange in Vietnam.

[5] *VAVA v. Dow et al*. (Original Complaint).

[6] *VAVA v. Dow et al*.(Amended Class Action Complaint).

[7] The ATS does not give courts a "mandate to seek out and define new and debatable violations of the law of nations." *VAVA v. Dow et al*. (2006): Brief for Defendants Appellees: 29 (05-1593-cv).

[8] With the high interest rates at the time, this amount increased to about $330 million by the time it was distributed to 291,000 veterans who filed their claims before the cut-off date in 1994. (*Stephensons v. Dow et al*. Prod. Liability Litig. 05-1760-cv [Second Circuit Court, brief for plaintiff appellee], 2007.)

[9] The government contractor defense is one element of the sovereign "discretionary-function" exception to the Federal Tort Claims Act (Cohen 2007). It gives protection to government and military contractors such as Dow and Monsanto by extending sovereign immunity under domestic tort law. For details, see Rakowsky (2005).

[10] The Nuremberg trials, in which the manufacturers of the chemical gas Zyklon B were found guilty of aiding the Nazi Final Solution by providing the chemicals used in gas chambers, offered historical precedence (Weinstein 2005).

[11] *Vietnamese Assoc. for Victims of Agent Orange/Dioxin v. Dow Chem. Co*., MDL No. 381, 04-CV-400 (EDNY, March 10, 2005) (Amended Memorandum, Order and Judgment), 50 (my italics in text).

[12] *VAVA v. Dow et al*., Appeal Decision, 27(my italics in text).

[13] In Re: *VAVA v. Dow et al.* (Petition for writ of certiorari to the Supreme Court of United States, 2008), 12–13.

[14] "The Weed-Killing Miracle: New and Improved Wee-Done," *New York Times,* May 27, 1951.

[15] "Modern Methods of Removing Unwanted Trees," *New York Times*, March 29,

1953.

[16] "War on Weeds," *New York Times*, February 8, 1953.

[17] "Scientists Assail Vietnam Method," *New York Time*s, August 29, 1971.

[18] "Soviet Note Assails US 'Poison Gas'; Embassy Rejects It," *New York Times*, March 27, 1965; "'Crime,' Peking Declares," *New York Times,* March 24, 1965; "Hanoi Charges US Tests Chemicals in South Vietnam," *New York Times*, August 23, 1965.

[19] "On the Planet Polluto," *New York Times*, April 25, 1971.

[20] "Hanoi Sees Birth Defects," *New York Times*, December 30, 1970.

[21] Cited in *VAVA v. Dow et al.* (United States Court of Appeals, September 30, 2005, brief for plaintiff apellants), 28.

[22] Dow memo (DOW 747096) June 24, 1965. AOWG listserv, Dow1-120896. "Report on the Chloracne Problem Meeting March 24, 1965," dated March 29, 1965, accessed March 2011, http://www.bluewaternavy.org/WhoKnew/Exhibit%2016.pdf.

[23] *Stephensons v. Dow et al*. (In re "Agent Orange" Prod. Liability Litig. 05-1760-cv) 2007 Second Circuit Court (reply brief for plaintiff appellants), 90.

[24] *VAVA v. Dow. 05-1953-cv* (brief for plaintiff appellant), 23.

[25] In Re: *VAVA v. Dow Supp. MDL No. 381, 04-CV-400* (EDNY); In Re: VAVA v. Dow et al, 05-1953-CV (Brief of the United States as Amicus Curea in Support of Defendants), 7.

[26] "Professor Arthur William Galston: The Plant Physiologist," Times (London), July 10, 2008, accessed July 22, 2011, http://www.timesonline.co.uk/tol/comment/obituaries/article4302745.ece.

[27] Stockholm Conference (United Nations Conference on the Human Environment), the first UN conference on the international environment, took place in 1972. On international laws of warfare, the Protocol I of the Geneva Convention and Environment Modification Treaty were promulgated in 1977, prohibiting the means of warfare intended or expected to cause long-term and severe damage to the environment.

[28] According to "Nancy Bucher: Biography, American Society for Cell Biology," accessed March 23, 2011, www.ascb.org/files/ profiles/Nancy_bucher.pdf, her study originally meant to examine the effects of 2,4-D in *reducing* sarcoma, not whether it could cause sarcoma.

[29] "On the Planet Polluto," *New York Times*, April 25, 1971.

[30] Anonymous personal communication.

[31] "Agent Orange maker Monsanto back in Vietnam: Scornful of Chemical's Killer Legacy, Some Vietnamese Resist Corporation's Return," Global Post, February 7, 2012, accessed August 17, 2012, http://www.globalpost.com/dispatches/globalpost-blogs/southeast-asia/agent-orange-maker-monsanto-returns-vietnam.

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