Allocating Pollution

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Law can often be used to reduce or even eliminate the harm from pollution by manipulating "exposure allocation," or how pollution is allocated across a target population. Opportunities for exposure allocation arise whenever the relationship between exposure to a pollutant and harm is nonlinear, as is the case for many pollutants. For these pollutants, exposure allocation presents the potential for reducing the harm from pollution even when it is not possible to reduce either the total amount of pollution emitted or the total amount of exposure. After identifying the conditions under which changing exposure allocations can improve health and save lives, this Article identifies legal strategies for managing exposure allocation to minimize the harm caused by pollution.

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INTRODUCTION

Carbon monoxide—an odorless, tasteless gas—is the leading cause of deadly poisoning in the industrialized world.[1] Unintentional carbon monoxide exposure hospitalizes thousands of people in the United States each year and kills hundreds.[2]

To prevent unintentional poisoning, the Consumer Product Safety Commission (CPSC) recommends that people install carbon monoxide detectors in their homes,[3] and the Environmental Protection Agency (EPA) regulates carbon monoxide as a criteria pollutant under the Clean Air Act.[4]

At the same time, the air in most American homes contains a detectable quantity of carbon monoxide—between 0.5 and 5 parts

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1 Stanley T. Omaye, *Metabolic Modulation of Carbon Monoxide Toxicity*, 180 Toxicology 139, 139 (2002).

2 See National Environmental Public Health Tracking Network (NEPHTN), *Carbon Monoxide Poisoning* (Centers for Disease Control and Prevention Apr 17, 2012), online at http://ephtracking.cdc.gov/showCarbonMonoxideLanding.action (visited Sept 23, 2012). The NEPHTN maintains updated statistics. See id. As of this writing, the Centers for Disease Control and Prevention (CDC) was reporting about 450 yearly cases of non-fire–related, deadly exposures to carbon monoxide. See id. Carbon monoxide is also used in more than two thousand suicides each year. See NEPHTN, *Carbon Monoxide Poisoning: Questions and Answers* 1 (CDC July 2006), online at http://www.cdc.gov/co/pdfs/faqs.pdf (visited Sept 23, 2012).

3 See CPSC, Press Release, *CPSC Recommends Carbon Monoxide Alarm for Every Home* (Jan 18, 2001), online at http://www.cpsc.gov/cpscpub/prerel/prhtml01/01069.html (visited Sept 23, 2012).

4 See 40 CFR § 50.8.

per million (ppm).[5] Readers of this Article have almost certainly inhaled carbon monoxide today and may be inhaling carbon monoxide right now.

Why are any of us still alive? And why aren't CPSC-approved carbon monoxide detectors going off constantly? Because carbon monoxide causes no measurable harm in the quantities to which we are typically exposed.[6] In fact, our own bodies produce carbon monoxide naturally and use it as a vital neurotransmitter and anti-inflammatory.[7] So while exposures to large amounts of carbon monoxide—such as can come from a broken appliance, a poorly ventilated wood stove, or automobile exhaust[8]—can be and are deadly, exposures to small amounts are harmless and, in very tiny quantities, might even be helpful.

Carbon monoxide is by no means unique in this. Toxicology, or the study of poisons, is based on the idea that "the dose makes the poison,"[9] and it has developed sophisticated techniques for quantifying just this kind of complex relationship between exposure to a dangerous substance and likely response.[10] For many pollutants[11]—like carbon monoxide—the relationship between exposure and harm is not linear;

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5 See EPA, *An Introduction to Indoor Air Quality (IAQ): Carbon Monoxide (CO)* (Apr 10, 2012), online at http://www.epa.gov/iaq/co.html (visited Sept 23, 2012).

6 The National Ambient Air Quality Standards (NAAQS) for carbon monoxide, which are set at levels "requisite to protect the public health" and "allow[] an adequate margin of safety," are 9 ppm for an eight-hour average and 35 ppm for a one-hour average concentration. 42 USC § 7409; 40 CFR § 50.8(a)(1)–(2). These are "not to be exceeded more than once per year." 40 CFR § 50.8(a)(1)–(2). The latter (one-hour) standard appears to have been set by reference to findings by the National Institute for Occupational Safety and Health (NIOSH), which identified 35 ppm as the lowest dose at which some measurable harm to human health occurs. See Occupational Safety & Health Administration (OSHA), *Occupational Safety and Health Guideline for Carbon Monoxide*, online at http://www.osha.gov/SLTC/healthguidelines/carbonmonoxide/recognition.html (visited Sept 23, 2012).

7 See Omaye, 180 Toxicology at 144–45 (cited in note 1).

8 See EPA, *Carbon Monoxide* (cited in note 5).

9 This is the modern iteration of an idea forwarded by Paracelsus, a Renaissance scientist. See Donald G. MacGregor, Paul Slovic, and Torbjorn Malmfors, *"How Exposed is Exposed Enough?" Lay Inferences About Chemical Exposure*, 19 Risk Analysis 649, 650 n 4 (1999). For further discussion, see Part I.C.

10 For an overview of toxicology methods and an introduction to the relationship between dose and response, see Casarett and Doull's classic toxicology reference. See generally Curtis D. Klaassen, ed, *Casarett & Doull's Toxicology: The Basic Science of Poisons* (McGraw-Hill 7th ed 2008); Curtis D. Klaassen and John B. Watkins III, eds, *Casarett & Doull's Essentials of Toxicology* (McGraw-Hill 2d ed 2010) (providing a distillation of the full Casarett and Doull reference).

11 By "pollutants," I mean units of anything that leads to "pollution." For a discussion of the concept of pollution, see Part I.

harm does not increase proportionally with exposure, and at low levels there may be no harm at all, or even some benefit.[12]

This Article argues that this quality of pollutants—that exposure and harm are not synonymous—creates opportunities for law and for pollution policy: opportunities to save lives and to improve human health, even where reducing the total amount of pollution is impossible or impractical. These opportunities are available through what I call "exposure allocation": the practice of allocating exposure to pollution to minimize the harm that the pollution causes and/or to maximize its beneficial effects. Because pollutants have different characteristics—and particularly because they vary in how much exposure causes increased harm—the same amount of pollution can cause significantly more or less harm depending upon how pollution exposure is allocated across the population. Good pollution policy seeks to reduce the harm caused by pollution. Exposure allocation often determines the harm caused by pollution, so good pollution policy must address exposure allocation.

The Article proceeds in five parts. Part I explores the relationship between pollution exposure and harm through three lenses: by looking at how people typically think of pollution and the social constructs around the concept; by exploring how regulatory agencies approach pollution regulation; and by summarizing the way that toxicologists have addressed the relationship, focusing particularly on the development of the dose-response curve.

Part II presents the concept of exposure allocation. Five common types of dose-response relationships are examined and I show that, for all but one type, exposure allocation is a key determinant of how much harm is caused by a set amount of pollution.

Part III addresses the question of how law can be used to affect exposure allocation. In the past, analyses of law's distributional effect on environmental goods have focused on its potential to create inequities between groups. This Part argues that this same power can be used for good as well, to shift exposure allocations, thereby saving lives and improving human health. It does so by identifying techniques, including siting, management of trading regimes, and strategic slippage and enforcement allocation, which can shift pollution exposures across space and time.

Part IV identifies places where exposure allocation can be incorporated into existing mechanisms for risk regulation. It focuses on

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12 See Michael A. Gallo, *History and Scope of Toxicology*, in Klaassen and Watkins, eds, *Essentials of Toxicology* 1, 1–3 (cited in note 10).

possibilities for integrating exposure allocation into modern risk analysis and cost-benefit analysis (CBA) and provides suggestions for legislative policy shifts.

Part V addresses remaining implications, puzzles, and limitations of exposure allocation as an approach to reducing harm from pollution and gestures at how exposure allocation can be applied to broadly-conceived examples of "pollution"—like hate speech or pornography—which fall outside the traditional ambit of environmental law.

In addressing pollution policy, the majority of this Article uses the term "pollution" in a way that will be familiar to readers, and that captures how the term is often treated in environmental statutes: to refer to chemical and biological substances that appear to cause some measurable harm to human health at some level of exposure.[13] This definition is adopted for two reasons: because it is an important category of pollution, and because it keeps the illustrations in Part II of the Article relatively simple. That said, this definition of pollution is by no means inevitable, and as we shall see in Part V, the ideas presented in the Article can be used to address wider conceptions of pollution as well.

I. REGULATING POLLUTION

A. What Is Pollution? The Social Meaning of Pollution and the "Pollution Heuristic"

We are exposed to pollution every day—in the air we breathe, the water we drink, and the food we eat—but this is something we are forced to tolerate, not something we seek out. No one likes to think about pesticide residue coating the apple they eat at lunch; no one enjoys pondering the arsenic content of the tap water used to make their coffee. No one eats more fish because they hear that radiation levels have increased in the wake of nuclear disaster; no one

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[13] This is essentially an effects-based approach to defining pollution, but one that ends up being remarkably comprehensive since—as we shall see—everything is harmful to human health if a person is exposed to a large enough quantity. For a discussion of the drawbacks of various approaches to pollution definition, including comprehensive approaches (which treat everything as a potential pollutant), effects-based approaches (which focus on a substance's effects), and listing approaches (which list either pollutants or nonpollutants), see John Copeland Nagle, *The Idea of Pollution*, 43 UC Davis L Rev 1, 29–41 (2009). Different environmental statutes take different approaches to defining pollution. For a comprehensive discussion, see id at 29–33.

breathes more deeply because they know that carbon monoxide concentrations are particularly high in their office.

If anything, the opposite is true. People respond to news about increased pollution by buying organic apples, water filters, Atlantic-farmed fish, and carbon monoxide detectors, and by lobbying federal and state lawmakers to increase pollution protections and to decrease the total amount of pollution. Pollution's unpopularity is not new.[14] Throughout history and across cultures, the concept of pollution has been used to express supreme aversion and normative condemnation: "Pollution, as opposed to purity, disturbs equilibrium, destroys or confuses desirable boundaries and states, and engenders destructive natural forces or conditions."[15] Observing this phenomenon, anthropologists have characterized responses to pollution as a recoiling from the unclean.[16] What counts as "unclean" is informed by underlying cultural values: modern Americans typically use

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14 For a fascinating and thorough interdisciplinary discussion of the concept of "pollution," see Nagle, 43 UC Davis L Rev at 5–14 (cited in note 13) (describing the history of concepts of pollution and analyzing the social and legal meaning of the word).

15 R.S. Khare, *Pollution and Purity*, in Alan Barnard and Jonathan Spencer, eds, *Encyclopedia of Social and Cultural Anthropology* 437, 437 (Routledge 1996). The entry goes on to list widely divergent societies—including the Western Pueblo Hopi, the Samoan, and the Bedouin—that all share a similar underlying view of pollution, and to give an overview of the development of the concepts of pollution and purity in sociology over the last one hundred years. Early sociology was much involved with tracing this dichotomy between pollution and purity. Early sociologists who worked on this issue included William Robertson Smith, James Frazer, and Émile Durkheim. Early analyses focused primarily on religious concepts of pollution. See William Robertson Smith, *Lectures on the Religion of the Semites* 142–44 (A&C Black 1889) (distinguishing holiness and purity from "magical" uncleanness or pollution); J.G. Frazer, *The Golden Bough: A Study in Comparative Religion* 171 (Macmillan 1890) (developing a theory of "primitive societies" and relationships between "magic" and pollution); Émile Durkheim, *Elementary Forms of the Religious Life* 310 (Oxford 2001) (Carol Cosman, trans) (originally published 1912) (examining multiple "primitive societies" and determining that each relied on distinctions between the sacred and the profane); Franz Steiner, *Taboo* 141–47 (Cohen & West 1956) (chronicling the remarkable intercultural diversity of rules about pollution and taboo). A culmination of this work came in sociologist Mary Douglas's *Purity and Danger*, where she argued that concepts of pollution and taboo are basically intertwined with Durkheim's distinction between the "sacred" and the "profane," or between order and disorder. Mary Douglas, *Purity and Danger: An Analysis of Concepts of Pollution and Taboo* 14–16 (Penguin 1966). For an application of Douglas's theory of pollution to legal constructions of pollution, see Nagle, 43 UC Davis L Rev at 72–78 (cited in note 13) (applying Douglas's approach to air and water pollution and to "cultural pollution" like violent video games and pornography).

16 See, for example, Douglas, *Purity and Danger* at 73–89 (cited in note 15) (comparing American concerns about hygiene with concerns about spiritual and ritual pollution in non-Western societies). See also Mary Douglas and Aaron Wildavsky, *Risk and Culture: An Essay on the Selection of Technical and Environmental Dangers* 186–98 (Berkeley 1982) (building on Douglas's earlier work to argue that modern conceptions of pollution are cultural constructs, and that pollution is "risky" in American culture—as in any culture—because it is embedded with highly tailored conceptions of what counts as risky).

"pollution" to refer to "the introduction of harmful substances or products into the environment,"[17] but it was not always so;[18] other cultures and historical Americans used the same term to discuss many other "unclean" things, including spiritual pollution,[19] culturally inappropriate food preparation,[20] and contamination through the "pollution" of bloodlines.[21]

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17 Stuart Berg Flexner and Leonore Crary Hauck, eds, *Random House Unabridged Dictionary* 1114 (2d ed 1993) (defining pollution as "the act of polluting or the state of being polluted," which leaves room for alternative definitions). Aaron Wildavsky and Mary Douglas explain this modern American focus on environmental pollution as a function of what they see as a culturally important narrative about "abuse of technology." See Douglas and Wildavsky, *Risk and Culture* at 2 (cited in note 16). See also Nagle, 43 UC Davis L Rev at 16–23 (cited in note 13) (chronicling additional uses of "pollution" in modern legal contexts).

18 For historical accounts of the development of the modern approach to pollution, see Peter Thorsheim, *Inventing Pollution: Coal, Smoke, and Culture in Britain Since 1800* 1–9 (Ohio 2006) (chronicling evolving conceptions of air pollution); Adam W. Rome, *Coming to Terms with Pollution: The Language of Environmental Reform, 1865–1915*, 1 Envir Hist 6, 6 (1996) (arguing that modern constructions of pollution as environmental postdate the Civil War, before which pollution was used as a concept referring to the "violation, perversion, or corruption of moral standards"); Nagle, 43 UC Davis L Rev at 7–16 (cited in note 13) (summarizing significant additional historical, legal, and sociological work done on the modern American conception of pollution as involving human-caused degradation of the environment and comparing modern and historical treatments of pollution). The first federal statute referring to pollution was the Oil Pollution Act of 1924, Pub L No 68-238, 43 Stat 604.

19 In rural India, for example, access rights to drinking water are often allocated according to caste, so that higher-caste individuals will not be at risk of spiritual pollution from water that has contacted people from lower castes. See Nandita Singh, *Water Management Traditions in Rural India: Valuing the Unvalued*, in Harsh Bhargava and Deepak Kumar, eds, *Rural Transformation: Socio-Economic Issues* 111–16 (ICFAI 2006):

> Water is believed to be a medium that transmits pollution when in contact with a person who himself is in a 'state of pollution.' Hence, the upper and lower castes, are expected to maintain distinctness of water sources as the lower castes . . . are believed to have the potential of transmitting pollution by sharing water sources.

For a more general discussion of the concept of pollution in caste systems, see generally Louis Dumont, *Homo Hierarchicus: The Caste System and Its Implications* (Chicago 1980) (Mark Sainsbury, Louis Dumont, and Basia Gulati, trans) (arguing that the primary structuralist dichotomy in the Hindu caste system is ritual purity and ritual pollution).

20 Romani law, for example, incorporates many rules meant to address the risk of cultural pollution (or "*marime*"), a number of which revolve around food preparation. See Walter Otto Weyrauch and Maureen Anne Bell, *Autonomous Lawmaking: The Case of the "Gypsies,"* 103 Yale L J 323, 342–51 (1993) (explaining that, among other things, a state of *marime* can result from eating food prepared in a culturally inappropriate way, as where it was cooked by a pregnant woman; served on dishes used by a non-Romani; or touched by someone's shadow, particularly if that person was non-Romani).

21 For a discussion of the conceptions of racial "pollution," see Bill Ezzell, *Laws of Racial Identification and Racial Purity in Nazi Germany and the United States: Did Jim Crow Write the Laws that Spawned the Holocaust?*, 30 S U L Rev 1, 5 n 20 (2002) (explaining that Nazi law prohibited sexual relations "between Jews and non-Jews on the grounds that such activities constituted 'racial pollution'"). For an even more recent example of what is arguably a view based on a concept of racial "pollution," see Michele Goodwin, *The Body Market: Race*

In psychological terms, once something is identified as unclean, the response to it becomes "affective": sudden, instantaneous, and emotionally laden.[22] Affective responses are automatic and can be quite powerful.[23] They can even cause measurable physical responses.[24] Feelings of disgust are an obvious example. In one famous experiment, experimenters offered participants a free glass of orange juice. The catch was that participants watched the experimenters dip a carefully sterilized cockroach into the juice just prior to being handed the glass. Perhaps unsurprisingly, most participants did not want to drink the juice.[25] We might also speculate that many of them felt little desire for a glass of orange juice for some time afterward.[26]

One characteristic of disgust is that it tends to be all or nothing: something is either repulsive or it is not. This creates a distinctive lack of scalability in response: a glass of juice is just as disgusting if one-fourth of a sterilized cockroach has been dipped into it as if the whole bug were submerged. In the context of toxic substances, this materializes as a general insensitivity to the quantity of substance to which people are exposed: people care far more about whether they have been exposed to pollution at all than the amount of that exposure.[27]

Politics and Private Ordering, 49 Ariz L Rev 599, 604–08 (2007), discussing Richard Titmuss, *The Gift Relationship: From Human Blood to Social Policy* 151–52 (Pantheon 1971) (arguing that paying blood donors would lead to overwhelming numbers of "negro" donors, who would "pollute" the blood supply).

22 See Paul Slovic, et al, *The Affect Heuristic*, in Thomas Gilovich, Dale Griffin, and Daniel Kahneman, eds, *Heuristics and Biases: The Psychology of Intuitive Judgment* 397, 397–420 (Cambridge 2002) (describing the affect heuristic as the tendency to make decisions based on immediate, intuitive emotional reactions).

23 See id.

24 See Paul Rozin, Jonathan Haidt, and Rick McCauley, *Disgust*, in Michael Lewis, Jeannette M. Haviland-Jones, and Lisa Feldman Barrett, eds, *Handbook of Emotions* 757, 767–69 (Guilford 3d ed 2008) (finding that feelings of moral repulsion or disgust are measurable in the brain's neurochemistry).

25 See id at 760. For a readable summary of findings in the psychology of disgust that focuses on Professor Paul Rozin's cockroach experiment and other experiments, see Michael D. Lemonick, *The Ewww Factor*, Time 51–52 (June 4, 2007).

26 A feeling of disgust is easily transferred to items associated with the object of disgust. See Andrea C. Morales and Gavan J. Fitzsimons, *Product Contagion: Changing Consumer Evaluations through Physical Contact with "Disgusting" Products*, 44 J Marketing Rsrch 272, 272 (2007) (exploring the limits of touch transference, and finding that consumers' values of goods decreased significantly when those goods came into contact with new, wrapped products that are typically associated with "disgusting" uses, like toilet paper or kitty litter).

27 For an excellent and informative comparison of laypeople and toxicologists' approaches to toxic substances and a presentation of evidence that laypeople act as "intuitive toxicologists" when faced with dangerous substances, see Nancy Kraus, Torbjörn Malmfors, and Paul Slovic, *Intuitive Toxicology: Expert and Lay Judgments of Chemical Risks*, in Paul Slovic, *The Perception of Risk* 285, 290–91 (Earthscan 2001) (finding that, in comparison with

We might easily tell an evolutionary story about this holistic aversion to dangerous substances;[28] a gatherer who instinctively recoils when faced with poisonous berries that once made her violently ill may do better at the game of natural selection than the gatherer who sees the same berries and decides to give them another go, or to only eat half as many this time around.

Whether the evolutionary account is explanatory or not, however, we should note that because response to pollution is often affective and emotional, it is not subject to systematic interrogation.[29] In this way it shares much in common with many other heuristics, or rules of thumb that function as simplified strategies for making decisions.[30]

Using heuristics to make decisions is quick and cheap—relatively few resources are needed to make a decision.[31] But heuristics can also lead to "misfire": to overapplication to situations for which they are ill-suited or simply wrong.[32] This is not to say that the use of heuristics is irrational or unworthy of respect[33]—much of the

toxicologists, laypeople are far more likely to agree with the statement that "[t]he fact of exposure to a pesticide is the critical concern, rather than the amount of exposure").

28 Jonathan Haidt tells a similar narrative about the evolution of disgust. See Jonathan Haidt, *The Moral Emotions*, in Richard J. Davidson, Klaus R. Scherer, and H. Hill Goldsmith, eds, *Handbook of Affective Sciences* 852, 857 (Oxford 2003) (describing disgust as an emotion with an evolutionary basis, and describing disgust as "a more generalized guardian of the mouth").

29 See Cass R. Sunstein, *The Arithmetic of Arsenic*, 90 Georgetown L J 2255, 2261–63 (2002) (warning that policies based on "intuitive toxicology" may lead to misfire, and arguing that CBA provides a disciplining mechanism).

30 See, for example, Slovic, et al, *The Affect Heuristic* at 419–20 (cited in note 22). Heuristic or affective processing is sometimes described as an example of one of two kinds of processes people use to make decisions. See generally Shelly Chaiken and Yaacow Trope, eds, *Dual-Process Theories in Social Psychology* (Guilford 1999); Steven A. Sloman, *The Empirical Case for Two Systems of Reasoning*, 119 Psych Bull 3 (1996). Recent advances in neuroscience suggest that these processes may have a biological component. See Colin Camerer, George Loewenstein, and Drazen Prelec, *Neuroeconomics: How Neuroscience Can Inform Economics*, 43 J Econ Lit 9, 14 (2005) (summarizing findings that different parts of the brain are associated with affective or cognitive processing). For a readable summary of recent social science findings on decision making, see generally Daniel Kahneman, *Thinking, Fast and Slow* (Farrar, Straus and Giroux 2011).

31 See Daniel Kahneman and Shane Frederick, *Representativeness Revisited: Attribute Substitution in Intuitive Judgment*, in Gilovich, Griffin, and Kahneman, eds, *Heuristics and Biases* 49, 51 (cited in note 22). See generally Cass R. Sunstein, Book Review, *Hazardous Heuristics*, 70 U Chi L Rev 751 (2003) (contextualizing *Heuristics and Biases* in the context of various treatments of heuristics in the legal literature).

32 See, for example, Sunstein, Book Review, 70 U Chi L Rev at 755 (cited in note 31).

33 Scholars argue about how best to treat decision making based on biases and, more particularly, about whether heuristic-based decision making is best treated as a cheap strategy for decision making or as value laden. For a helpful overview of different approaches, see Dan M. Kahan, et al, Book Review, *Fear of Democracy: A Cultural Evaluation of Sunstein on Risk*, 119

time, the risk of misfire may not be so great that it is worthwhile for an individual to invest in a tailored decision-making strategy over a heuristic.[34]

There is danger, however, in applying heuristic thinking to policy contexts where large numbers of people are affected by each decision. For these decisions, error costs typically increase far faster than do the decision costs of adopting tailored strategies.[35]

Harv L Rev 1071, 1072–73 (2006) (arguing, in response to Sunstein, that the experts themselves suffer from heuristic biases subject to overapplication, so excluding lay people will not improve risk regulation). For a defense of the practical use of heuristics in everyday life, see Gerd Gigerenzer, Peter M. Todd, and the ABC Research Group, *Simple Heuristics That Make Us Smart* 3–21 (Oxford 1999). For a presentation (and critique) of a rule-utilitarian defense of the use of heuristics in moral realms, see Sunstein, Book Review, 70 U Chi L Rev at 776–82 (cited in note 31); Cass R. Sunstein, *Moral Heuristics*, 28 Behav & Brain Sci 531, 534–40 (2005).

[34] More formally, let us consider the hyperrational individual faced with a decision about what to do with a dangerous substance. Imagine that she must decide between two decision strategies.

The first decision option (D_1) is to create a tailored strategy for dealing with the substance, based on all available data and considering a number of different approaches. This decision strategy will lead to an outcome where the individual gets a certain payoff from adopting the tailored strategy (O_1). This strategy has high decision costs; it requires significant time and resources.

The second decision option (D_2) is to rely on a heuristic to deal with the substance. Doing so will also give the individual some payoff (O_2). In comparison to a tailored strategy, the application of this heuristic strategy has low decision costs; it will be easy, cheap, and will not require the individual to invest many resources at all, such that the cost of $D_2 \leq D_1$.

When will it be worth it to this individual to adopt a heuristic strategy over a tailored strategy? When the marginal cost of adopting the tailored strategy outweighs the marginal benefit of the increased decision quality from adopting that strategy; when $(D_1 - D_2) > (O_1 - O_2)$. Conversely, when will it be worthwhile for an individual to adopt a tailored strategy over a heuristic strategy? When the marginal benefit of adopting the tailored strategy outweighs the marginal cost; when $(D_1 - D_2) < (O_1 - O_2)$.

For many decision contexts—particularly those having to do with exposure to the thousands of substances we encounter every day—this calculus would reasonably lead an individual to adopt a heuristic strategy because the payoff from increased accuracy is simply not that great.

[35] More formally, let us imagine that the benefit of a policy will accrue to a number of people (P). In that case, the marginal benefit of adopting the tailored strategy will outweigh the marginal cost when $(D_1 - D_2) < P(O_1 - O_2)$. The right side of the equation will grow in proportion with the number of people affected, so as more people are affected, there will be a greater and greater benefit to adopting the tailored strategy.

One reasonable objection would be that a policy decision might increase in complexity as we have to aggregate across increasing numbers of affected people. In that case, we really ought to modify the tailored decision cost (D_1) with some measure of increased decision complexity from aggregation (C), so that when $(CD_1 - D_2) < P(O_1 - O_2)$, we should adopt the tailored decision strategy, and when $(CD_1 - D_2) > P(O_1 - O_2)$ we should adopt the heuristic strategy. Is this more likely to support the adoption of a heuristic strategy or a tailored one? It will depend upon whether we think that complexity is likely to increase proportionally with the size of the affected population. When policies affect large populations—as is frequently the case with environmental regulations—this is unlikely to be the case: decisions as to permissible

What does this mean in the context of pollution policy? It means that when the health of many people is at stake, it is dangerous to use a heuristic approach to pollution policy, even if most of the people at risk use a pollution heuristic in their everyday lives. For policy decisions that aggregate potential harm, we need deliberate decision-making tools that give real weight to the suffering that each affected person might experience.

B. Quantitative Risk Analysis and the Traditional Bifurcation between Assessment and Management

In our everyday lives, most of us are "intuitive toxicologists," recoiling from pollution and avoiding it wherever possible.[36] But while this sort of intuitive recoil from pollution arguably underlies some early environmental regulations,[37] it is not how most risks from pollution are now regulated. Rather, modern risk regulation begins with quantitative risk assessment, a systematized approach to analyzing and quantifying the type, magnitude, and probability of harm that might be caused by exposure to a particular environmental hazard.[38] It has developed into a highly technical process with a formalized set of analytical steps.

Modern quantitative risk assessment was developed in response to two important events in the early 1980s. The first was the Supreme Court's decision in *Industrial Union Department, AFL–CIO v American Petroleum Institute*[39] ("The Benzene Case"), where it struck down regulations promulgated by the Occupational Safety and Health Agency (OSHA) for failure to demonstrate the existence of a significant risk to human health from occupational exposure to

levels of arsenic in the drinking water, for example, seem unlikely to increase 300-million-fold in complexity, even though they might easily affect 300 million people.

36 Sunstein, 90 Georgetown L J at 2262–63 (cited in note 29).

37 See, for example, the 1958 Delaney Clause, which banned all carcinogens from the food supply, regardless of dose. 21 USC § 348(c)(3)(A) (applying the following restriction to food additives: "[N]o additive shall be deemed to be safe if it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal"). See also 21 USC § 379e(b)(5)(B) (applying an identical ban to color additives); 21 USC § 360b (applying the ban to animal drug residues).

38 See EPA, *An Examination of EPA Risk Assessment Principles and Practices* 2 (Mar 2004), online at http://www.epa.gov/osa/pdfs/ratf-final.pdf (visited Sept 23, 2012) (defining risk assessment as a "process in which information is analyzed to determine if an environmental hazard might cause harm to exposed persons and ecosystems"). See also Arden Rowell, *Risk Assessment*, in Ian Spellerberg, ed, *Berkshire Encyclopedia of Sustainability: Measurements, Indicators and Research Methods for Sustainability* 308, 308–12 (Berkshire 2012).

39 448 US 607 (1980).

benzene, which was a known carcinogen.[40] This decision was a shock to regulators, particularly since the regulatory analysis done by OSHA was one of the more technical and formalized of its time. Regulators were left scrambling to find new ways to ensure that their regulations were based on verifiably "significant" risks.

The second key event was what was perceived as the increasing politicization of EPA under Director Anne Gorsuch. Gorsuch was appointed by President Ronald Reagan in 1981 and implemented a series of policies to shrink the size of the agency and to lessen environmental burdens on business.[41] These policies were extremely controversial, and Gorsuch was forced to resign in 1983, amid a congressional inquest and after having been cited for contempt of Congress.[42]

William Ruckelshaus, who had been the founding director of EPA in 1970, was asked to step in after Gorsuch's resignation. Ruckelshaus saw quantitative risk assessment as a tool for combating the kind of overt politicization of the regulatory process that had taken place under Gorsuch. Accordingly, he advocated for sharp and clearly operationalized distinctions between what he saw as the scientific risk *assessment* process and the more political decision making inherent in decisions about risk management.[43] Ruckelshaus was aware that the risk assessment process unavoidably involved some

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40 Id at 661–62 (plurality). The decision was based on a reading of the Occupational Safety and Health Act of 1970 (OSH Act), Pub L No 91-956, 84 Stat 1590. The Act defined a health and safety standard as a standard "reasonably necessary and appropriate to provide safe or healthful employment." OSH Act § 3(8), 84 Stat at 1591. It directed the Secretary of Labor to "set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity." OSH Act § 6(b)(5), 84 Stat at 1594. On the basis of this language, the plurality (in an opinion written by Justice John Paul Stevens) wrote that "[a]lthough the Agency has no duty to calculate the exact probability of harm, it does have an obligation to find that a significant risk is present before it characterizes a place of employment as 'unsafe.'" *The Benzene Case*, 448 US at 655 (plurality).

41 See Phil Wisman, *EPA History (1975–1985)*, online at http://www.epa.gov/aboutepa/history/topics/epa/15b.html (visited Sept 23, 2012). See also Anne M. Gorsuch, *Views from the Former Administrators* (EPA Journal Nov 1985), online at http://www.epa.gov/aboutepa/history/topics/epa/15e.html (visited Sept 23, 2012).

42 The inquest involved the $1.6 billion Superfund and allegations that EPA had mishandled the funds. Congress asked Gorsuch to deliver related files and she refused. See Wisman, *EPA History* (cited in note 41). For a brief discussion of Gorsuch's legacy, see Douglas Martin, *Anne Gorsuch Burford, 62, Reagan E.P.A. Chief, Dies*, NY Times C13 (July 22, 2004).

43 See William D. Ruckelshaus, *Risk, Science and Democracy*, 1 Issues in Sci & Tech 19, 31–35 (1985). Ruckelshaus was the first Administrator of EPA from 1970 to 1973 and served again from 1983 to 1985.

policy decisions, such as who the targeted population would be.[44] But he saw significant value in teasing apart the two processes, and this bifurcated approach was foundational to the development of modern risk analysis.

The bifurcated approach to risk assessment and risk management was firmly established in the National Academy of Science's 1983 *Risk Assessment in the Federal Government: Managing the Process*, more commonly called the "Red Book" for the original color of its cover.[45] This document became the basic source book for quantitative risk assessment for federal regulations,[46] and is still widely relied upon,[47] despite the recent issuance of the National Research Council's (NRC) *Science and Decisions: Advancing Risk Assessment*, known as the "Silver Book," in late 2008.[48]

The initial edition of the *Red Book* was focused particularly on human health risks from carcinogens. Accordingly, it adopted a set

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44 Most famously, Ruckelshaus compared the process of risk assessment to a captured spy: "We should remember that risk assessment data can be like a captured spy: if you torture it long enough, it will tell you anything you want to know." William D. Ruckelshaus, *Risk in a Free Society*, 4 Risk Analysis 157, 157–58 (1984).

45 See National Research Council (NRC) Committee on the Institutional Means for Assessment of Risks to Public Health, *Risk Assessment in the Federal Government: Managing the Process* ("Red Book") ix, 48–49 (National Academy 1983).

46 See Lorenz R. Rhomberg, *A Survey of Methods for Chemical Health Risk Assessment among Federal Regulatory Agencies*, 3 Human & Ecological Risk Assessment 1029, 1080 (1997).

47 See EPA, *Risk Assessment Principles and Practices* at 4 (cited in note 38) (describing the *Red Book* and explaining that "EPA has integrated the principles of risk assessment from this groundbreaking report into its practices to this day"). As part of integrating the *Red Book* processes into its regulatory decision making, EPA published *Risk Assessment and Management*, which gives more detail about how each step of the risk assessment process should be handled. EPA, *Risk Assessment and Management: Framework for Decision Making* 13–14 (EPA 1984). In following years, the National Academy of Sciences refined its principles still further with a series of supplementary reports. See generally NRC Committee on Pesticides in the Diets of Infants and Children, *Pesticides in the Diets of Infants and Children* (National Academy 1993); NRC Committee on Risk Assessment of Hazardous Air Pollutants, *Science and Judgment in Risk Assessment* ("Blue Book") (National Academy 1994) (responding to a congressional request to review the risk assessment methods used by EPA); NRC Committee on Risk Characterization, *Understanding Risk: Informing Decisions in a Democratic Society* (National Academy 1996) (Paul C. Stern and Harvey V. Fienberg, eds).

48 See NRC Committee on Improving Risk Analysis Approaches Used by the US EPA, *Science and Decisions: Advancing Risk Assessment* ("Silver Book") (National Academies 2008). The *Silver Book* presents a number of critiques of *Red Book* processes. Among other things, it directs risk managers to explicitly identify potential policy outcomes that assessors should analyze. As of this writing, regulators are still struggling to find ways to shift to *Silver Book* processes. See Bernard D. Goldstein, *EPA at 40: Reflections on the Office of Research and Development*, 21 Duke Envir L & Pol F 295, 307–08 (2011). For a discussion of how the recommendations of this Article can be implemented along with *Silver Book* processes, see notes 214–20 and accompanying text.

of methodologies borrowed from toxicology. These methodologies continue to provide the basis for modern quantitative risk assessment, although they have been somewhat modified over the years to apply to other types of health risks and risks to the environment as well as to humans.[49]

As EPA and other federal agencies interpret the *Red Book*, there are four processes necessary for quantitative risk assessment: hazard identification,[50] dose-response assessment,[51] exposure assessment,[52] and risk characterization.[53] Completing these steps allows the

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49 See EPA, *Risk Assessment Principles and Practices* at 2 (cited in note 38). See also Rowell, *Risk Assessment* at 308–11 (cited in note 38).

50 Hazard identification is an assessment of whether an environmental stressor has the potential to be hazardous to human health. Particular attention is paid to toxicodynamics (the effect the substance has on the human body) and toxicokinetics (how the body absorbs, distributes, metabolizes, and eliminates the substance). Although statistically controlled clinical studies on humans are considered the gold standard for linking a stressor to adverse health impacts, these are often not available because of human subjects concerns about testing dangerous stressors (like poisons) on people. Accordingly, environmental risk assessors must often extrapolate from two kinds of second-best data: epidemiological studies, which look at statistical evaluations of human populations to see if there is an apparent association between exposure to the stressor and adverse health effects; and animal studies, which test the health impacts of environmental stressors on animals. See NRC, *Red Book* at 3 (cited in note 45). See also Rowell, *Risk Assessment* at 309 (cited in note 38).

51 Dose-response assessment, also called hazard characterization, seeks to establish the "dose" of a stressor that causes measurable harm to human health, and the way in which changing a dose will affect the likelihood or magnitude of that harm. With most (but not all) stressors, the adverse effects of the stressor increase with the dose. Assessors try to draw a "dose-response curve" to represent just how quickly adverse effects worsen. See id. For substantially more discussion of the dose-response relationship, see Part II.

52 Exposure assessment is the process of determining the magnitude, frequency, and duration with which humans are exposed to an environmental stressor. Because exposure to stressors often varies widely across the population, a challenge in exposure assessment is determining the population whose exposure will be measured. Typically assessors try to describe the size, nature, and types of humans exposed to the stressor, as well as the different exposure pathways (the course a stressor takes from its source to the person being exposed) and exposure routes (the means of entry into the body) through which people can be exposed. Assessors attempt to quantify exposure using three different approaches: point of contact measurement, scenario evaluation, and reconstruction. Assessors also attempt to adjust for variable exposure across individuals. See NRC, *Red Book* at 3 (cited in note 45). See also Rowell, *Risk Assessment* at 310 (cited in note 38). For a very useful discussion of the current state of exposure assessment in the context of the risks posed by Hazardous Air Pollutants (HAPs), see Thomas O. McGarity, *Hazardous Air Pollutants, Migrating Hot Spots, and the Prospect of Data-Driven Regulation of Complex Industrial Complexes*, 86 Tex L Rev 1445, 1448–53 (2008) (outlining and evaluating actual exposure assessment practices at EPA and at the Texas Commission on Environmental Quality). Note that exposure *assessment*—a process for assessing quantities and sources of exposure—is necessary to but not the same as exposure *allocation*, which, as we shall see, looks at whether alternative allocations of exposure would lead to less aggregate harm. See Part II.

53 Risk characterization is meant to be an integration of the other three processes, so it must happen after they are complete. To characterize the risk, assessors set the dose incurred

risk assessor—at least in theory—to identify potential health effects of the substance (from hazard identification), how the risk of those effects changes with the dose (from dose-response assessment), and how and how much a target population is likely to be exposed to the substance (from exposure assessment). And all of this information is then integrated into a single risk characterization.[54]

Under *Red Book* methodology, all of these steps are meant to be completed before risk management procedures begin. Regulators then implement one or many risk management frameworks to determine which, how, and how much risks will be regulated against. Often these risk-management decisions are driven by the environmental statute being enforced.[55] The Federal Insecticide, Fungicide, and Rodenticide Act,[56] for example, allows pesticides to be marketed only if they "will not generally cause unreasonable adverse effects on the environment."[57] The Clean Air Act,[58] in contrast, requires that EPA set National Ambient Air Quality Standards (NAAQS) for specified criteria pollutants at levels that "allowing an adequate margin of safety, are requisite to protect the public health."[59] And the Safe Drinking Water Act[60] requires a two-part procedure, whereby EPA first sets a maximum contaminant-level goal "at the level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety,"[61] and then promulgates the national primary drinking water regulations

by the target population against the hazard and dose-response data available for the substance. This characterization typically includes a detailed explanation of the nature of the hazard, its seriousness, and how likely it is to occur. NRC, *Red Book* at 3 (cited in note 45).

54 For the human health risk assessments on which EPA routinely relies, see EPA, *Integrated Risk Information System (IRIS)* (Sept 13, 2012), online at http://www.epa.gov/iris (visited Sept 23, 2012).

55 For a nice introductory overview of risk-management frameworks used in various environmental statutes, see Richard L. Revesz, *Environmental Law and Policy* ch II.2 at 90–93 (Thomson West 2008).

56 Pub L No 80-104, 61 Stat 163 (1947), codified at 7 USC § 136 et seq.

57 7 USC § 136a(c)(5)(D). "Unreasonable adverse effects" are those that pose an "unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits." 7 USC § 136(bb).

58 Pub L No 88-206, 77 Stat 392 (1963), codified at 42 USC § 7401 et seq.

59 42 USC § 7409(b)(1). Different portions of the Clean Air Act require different risk management decisions. See, for example, 42 USC § 7412(f)(2)(A) (requiring that HAPs be controlled through technology-based standards with a negligible-risk backstop).

60 Pub L No 93-523, 88 Stat 1661 (1974), codified at 42 USC § 300f et seq.

61 42 USC § 300g-1(b)(4)(A).

"as close to the maximum contaminant level goal as is feasible," "taking cost into consideration."[62]

Agencies also undertake risk-management decisions in the context of executive orders, which provide additional guidance on how to trade off different regulatory goals. Since the 1980s, agency interpretation of environmental statutes has been performed through the lens of a series of orders that centralize regulatory review under the Office of Management and Budget (OMB), and which have imposed a requirement that major regulations[63] undergo cost-benefit analysis (CBA) prior to promulgation.[64] Since the effect of this requirement is that CBA informs all environmental regulations except where expressly prohibited by statute, CBA is an important risk-management tool for managing environmental risks.[65]

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62 42 USC § 300g-1(b)(4)(D). More specifically, "feasible" is "feasible with the use of the best technology, treatment techniques and other means . . . available (taking cost into consideration)." 42 USC § 300g-1(b)(4)(D).

63 "Major regulations" are defined as regulations with an annual effect on the economy of $100 million or more. See Executive Order 12291, 3 CFR 127 (1982) (promulgating a new rules requirement that government regulation be cost-benefit justified and maximize net benefits to society).

64 The first such order, Executive Order 12291, was issued by President Reagan a few months after he took office. It directed that "to the extent permitted by law," "[r]egulatory action shall not be undertaken unless the potential benefits to society for the regulation outweigh the potential costs to society," and ordered that "[a]mong alternative approaches to any given regulatory objective, the alternative involving the least net cost to society shall be chosen." 3 CFR 127, 128 (1982). This Order remained in effect through President George H.W. Bush's presidency. President Bill Clinton replaced that order with Executive Order 12866, which continued to require CBA, but applied a slightly softer requirement that "in choosing among alternative regulatory approaches, agencies should select those approaches that maximize net benefits (including potential economic, environmental, public health and safety, and other advantages; distributive impacts; and equity) unless a statute requires another regulatory approach." Executive Order 12866, 3 CFR 638 (1994). President George W. Bush kept the requirement of CBA and centralized control of regulatory review still further by revising Executive Order 12866 with Executive Order 13422, which added a requirement that agencies identify a "market failure" before regulating, allowed the Office of Information and Regulatory Affairs (OIRA) to review and edit agency guidance documents (including risk assessment materials), and created Regulatory Policy Review Officers, which were political appointments placed in agencies. Executive Order 13422, 3 CFR 191 (2008) (amending Executive Order 12866). President Barack Obama revoked Executive Order 13422 within weeks of taking office and issued Executive Order 13563. See Executive Order 13497, 3 CFR 318 (2010); Executive Order 13563, 76 Fed Reg 3821, 3821 (2011) (incorporating the cost-benefit requirements of Clinton-era Executive Order 12866 and adding additional requirements for public participation and retrospective analysis of existing rules). Executive Order 13563 requires, among other things, that agencies "propose or adopt a regulation only upon a reasoned determination that its benefits justify its costs (recognizing that some benefits and costs are difficult to quantify)." 76 Fed Reg at 3821 (cited in note 64).

65 For a more detailed discussion of agencies'—and particularly EPA's—cost-benefit practices, see Arden Rowell, *The Cost of Time: Haphazard Discounting and the Undervaluation of*

How are distributional fairness issues addressed in risk regulation? Despite repeated calls for a clear process of distributional analysis, implementation remains haphazard, and no dominant approach has emerged.[66] Under Executive Order 12866, which was the primary order governing regulatory analysis for many years, distributional impacts were incorporated into regulatory analyses only with a breezy exhortation to count "distributive impacts" and "equity" in with other "benefits."[67] The Clinton-era Executive Order 12898 on environmental justice was a bit more specific:

> To the greatest extent practicable and permitted by law, . . . each Federal agency shall make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations in the United States.[68]

But while this required that agencies make "environmental justice" part of their "mission," it provided no guidance as to how disproportionate effects were to be either identified or addressed. Even EPA, which is typically the most methodologically sophisticated of the environmental agencies, struggled to operationalize this directive.[69]

The Obama administration's response to this critique has been to include a separate provision in Executive Order 13563 explicitly

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Regulatory Benefits, 85 Notre Dame L Rev 1505, 1517–33 (2010) (describing agency cost-benefit practices).

66 See Matthew D. Adler, *Risk Equity: A New Proposal*, 32 Harv Envir L Rev 1, 2–5 (2008) (noting that, in stark contrast to risk assessment and CBA, "[n]o clear paradigm for equity analysis has yet emerged in [current] practice" and presenting a potential paradigm for operationalizing equity analyses).

67 3 CFR 638, 638–40 (1994). As Professor Matthew Adler notes in *Risk Equity*, this is a peculiar exhortation, given that "the net-benefits-maximization test of traditional cost-benefit analysis is insensitive to distributional considerations." Adler, 32 Harv Envir L Rev at 2 (cited in note 66). For EPA's attempt at incorporating distributional analysis of some kind into its analysis of benefits, see EPA, *Guidelines for Preparing Economic Analyses* 139–74 (Sept 2000), online at http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0228C-07.pdf/$file/EE-0228C-07.pdf (visited Sept 23, 2012).

68 Executive Order 12898, 3 CFR 859, 859 (1995).

69 See Adler, 32 Harv Envir L Rev at 2–4 (cited in note 66) (arguing that, despite the fact that EPA has written numerous guidelines directing its agents to consider equity, they nonetheless continue to rely primarily on CBA). For a deep attempt to combine a distributional analysis with welfarism, see Matthew D. Adler, *Well-Being and Fair Distribution: Beyond Cost-Benefit Analysis* 114–53 (Oxford 2012) (surveying inequality metrics and concluding that they are all inferior to a social welfare function).

permitting distributional concerns—and other difficult-to-quantify values—to be "considered": "Where appropriate and permitted by law, each agency may consider (and discuss qualitatively) values that are difficult or impossible to quantify, including equity, human dignity, fairness, and distributive impacts."[70] In comparison to the requirement that agencies perform quantitative CBA,[71] this exhortation is strikingly vague, and agencies have yet to determine the best way to operationalize it.

In sum, then, risk assessment of environmental risks has commonly been used to inform risk management, which usually involves CBA. And then at some separate point, regulators consider qualitative values like distributive impacts and equity. Perhaps in part because of the historical distinction between risk assessment and risk management, these relationships have been unidirectional: distributional analyses have not been used to inform CBAs, and CBAs and other risk-management techniques are not used to inform risk assessments.[72] In the following Section, I will show that this is a mistake: that a unified understanding of assessment, management, and the allocation of pollution can provide real and important benefits in the form of better health and of lives saved.

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70 76 Fed Reg at 3821 (cited in note 64). Compare the language of President Obama's Executive Order 13563 with President Clinton's Executive Order 12898. See 3 CFR 859, 859 (1995) ("[E]ach Federal agency shall make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse . . . effects . . . on minority populations.").

71 76 Fed Reg at 3821 (cited in note 64):

> As stated in [Executive Order 12866] and to the extent permitted by law, each agency must, among other things: (1) propose or adopt a regulation only upon a reasoned determination that its benefits justify its costs (recognizing that some benefits and costs are difficult to quantify); (2) tailor its regulations to impose the least burden on society, consistent with obtaining regulatory objectives, taking into account, among other things, and to the extent practicable, the costs of cumulative regulations; (3) select, in choosing among alternative regulatory approaches, those approaches that maximize net benefits (including potential economic, environmental, public health and safety, and other advantages; distributive impacts; and equity); (4) to the extent feasible, specify performance objectives, rather than specifying the behavior or manner of compliance that regulated entities must adopt; and (5) identify and assess available alternatives to direct regulation, including providing economic incentives to encourage the desired behavior, such as user fees or marketable permits, or providing information upon which choices can be made by the public.

72 Although this appears to be changing with the *Silver Book*'s recent suggestions. See NRC, *Silver Book* at 242–55 (cited in note 48) (arguing that "the questions that risk assessments need to address must be raised before risk assessment is conducted" in order to ensure its utility for risk management and developing a framework for the inclusion of risk-management concerns into the risk assessment process).

C. A Brief History of Poisons and of the Development of the Dose-Response Relationship

People have been aware of poisonous substances for as long as we have records of knowledge:[73] the Ebers Papyrus, which is one of the oldest known writings, lists a number of recognized poisons, including aconite, lead, and antimony;[74] the book of Job speaks of poison arrows;[75] and Socrates, like other condemned Greek prisoners, was purposefully poisoned with hemlock.[76] Laws to regulate deliberate poisoning date back to Sulla's *Lex Cornelia*, which prohibited poisoning (by arsenic and other means) in Roman times.[77]

Yet even in antiquity, some suspected that dangerous substances could also be helpful to health: consider the classic story of King Mithridates, who ingested a potpourri of poisons every day to make himself resistant to assassination attempts.[78] The potentially complex relationship between the dose of a substance and its effect was first summarized by Paracelsus, a Renaissance physician and alchemist.[79] On the basis of experiments, he contended that "all substances are poisons; there is none which is not a poison. [Only the] dose differentiates [a] poison from a remedy."[80] This relationship—the dose-response relationship—became foundational for modern toxicology.[81]

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73 For an excellent short history of toxicology, on which this historical discussion depends, see Gallo, *History and Scope of Toxicology* at 3–10 (cited in note 12).

74 Id at 3.

75 Job 6:4 (King James Version) (speaking of poison arrows that "drinketh up my spirit").

76 Plato's *Phaedo* gives his famous account of Socrates's last hours. See generally Plato, *Phaedo* (Cambridge 1875) (E.M. Cope, trans).

77 The *Lex Cornelia* was issued in 82 BC. After Sulla's time the law also came to be used to prosecute careless druggists. See Gallo, *History and Scope of Toxicology* at 4 (cited in note 12).

78 Readers might be familiar with Mithridates IV of Pontus through the final stanza of A.E. Housman's 1886 poem "Terence, This is Stupid Stuff":

> There was a king reigned in the East:/ There, when kings will sit to feast,/ They get their fill before they think/ With poisoned meat and poisoned drink./ He gathered all that springs to birth/ From the many-venomed earth;/ First a little, thence to more,/ He sampled all her killing store;/ And easy, smiling, seasoned sound,/ Sate the king when healths went round./ They put arsenic in his meat/ And stared aghast to watch him eat;/ They poured strychnine in his cup/ And shook to see him drink it up:/ They shook, they stared as white's their shirt:/ Them it was their poison hurt./ —I tell the tale that I heard told./ Mithridates, he died old.

A.E. Housman, *A Shropshire Lad* 91, 94 (John Lane 1917). For a fascinating history of Mithridates that attempts to disentangle the legends surrounding him, see generally Adrienne Mayor, *The Poison King: The Life and Legend of Mithridates* (Princeton 2010).

79 See Gallo, *History and Scope of Toxicology* at 5 (cited in note 12) (referring to Paracelsus as having "formulated many revolutionary views that remain an integral part of the structure of toxicology, pharmacology, and therapeutics today").

80 Id at 4. See also Walter Pagel, *Paracelsus: An Introduction to Philosophical Medicine in the Era of the Renaissance* 363 (Karger 2d ed 1982) (applying Paracelsus's famous words—

Early studies of dose-response relationships focused on contexts with very large exposure levels, as these were the contexts where searching for a relationship was particularly fruitful:[82] targeted relationships included those between mining activities and "miners' sickness"[83] and between exposure to soot and cancer.[84]

As scientists and experimenters developed new substances over subsequent decades, the dose-response relationship was used in increasingly sophisticated ways. "Patent" medicines were analyzed;[85] the health effects of anesthetics and disinfectants were quantified.[86] Discovery of radioactivity and "vital amines," or vitamins, led to the development of large-scale, multiple-animal studies that were used to determine whether these new discoveries were helpful or harmful.[87] Paracelsus would not have been surprised to hear that researchers found many of these substances to be both helpful *and* harmful—that small amounts of radiation, and of many vitamins, ap-

"Alle Ding sind Gift und nichts ohn' Gift; allein die Dosis macht, daß ein Ding kein Gift ist"—to argue that "poisons can be remedies of high power when administered in a non-lethal form"). Paracelsus's maxim is often translated more simply as "the dose makes the poison." Erica Beecher-Monas, *The Heuristics of Intellectual Due Process: A Primer for Triers of Science*, 75 NYU L Rev 1563, 1641 n 444 (2000).

81 See Gallo, *History and Scope of Toxicology* at 5 (cited in note 12) (calling the relationship a "bulwark of toxicology," and citing Paracelsus's work as formulative). Paracelsus is frequently cited as the father of toxicology for his role in articulating the dose-response relationship. See, for example, id at 4–5. That said, many toxicologists now believe—as we shall see in Part II—that Paracelsus's maxim is not strictly true, because there appear to be "nonthreshold" substances that are dangerous in all quantities. For a criticism of the careless use of Paracelsus's maxim in judicial decisions, see Beecher-Monas, 75 NYU L Rev at 1615 n 283 (cited in note 80).

82 See, for example, Bernardino Ramazzini, *Diseases of Workers* 15 (Chicago 1940) (Wilmer Cave Wright, trans).

83 "Miners' sickness" probably referred to black lung, but may have meant lung cancer. See Paracelsus, *On the Miners' Sickness and Other Diseases of Miners*, in Henry E. Sigerist, ed, *Four Treatises of Theophrastus von Hohenheim, Called Paracelsus* 61–73 (Johns Hopkins 1941) (C. Lilian Temkin, et al, trans) (theorizing, in 1567, that the origin of "miners' sickness" was exposure to chemicals found within mines).

84 See Gallo, *History and Scope of Toxicology* at 5 (cited in note 12) (noting that, in 1775, Percival Pott identified exposure to soot as the cause of cancer in chimney sweeps).

85 So-called patent medicines proliferated in the nineteenth century, and new methods of distillation and purification meant that peddlers could now offer more concentrated forms of their wares. People died as a result, and this helped spur passage of legislation meant to prevent adulteration and encourage labeling. See Pure Food and Drug Act of 1906, Pub L No 59-384, ch 3915, 34 Stat 768.

86 The human health effects of disinfectants remain controversial. Note that EPA regulates disinfectants as pollutants. See, for example, National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule, 71 Fed Reg 388 (2006).

87 See Gallo, *History and Scope of Toxicology* at 7 (cited in note 12).

peared to be useful, and sometimes even vital, whereas excessive amounts could kill.[88]

Thus toxicologists came to chronicle more and more substances and, in doing so, found that different substances exhibit very different relationships between dose (or exposure) and harm, and that some substances that are dangerous in large quantities are actually helpful in smaller doses.[89] In time, toxicologists began creating graphical representations of these varying relationships by drawing dose-response curves.[90]

D. How Agencies Use Dose-Response Data

In the 1980s, as regulatory agencies began to adopt quantitative methods for analyzing environmental risks,[91] EPA (and other agencies charged with environmental mandates[92]) began to incorporate toxicological data into their quantitative risk assessments and thus

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88 See Mark P. Mattson and Edward J. Calabrese, *Hormesis: What It Is and Why It Matters*, in Mark P. Mattson and Edward J. Calabrese, eds, *Hormesis: A Revolution in Biology, Toxicology, and Medicine* 1, 2 (Humana 2010). William Fletcher is typically credited as the first to discover that illness could result from vitamin deficiencies. See Kenneth J. Carpenter, *Beriberi, White Rice, and Vitamin B: A Disease, a Cause, and a Cure* 74–75, 77 (Berkeley 2000) (describing Fletcher's experiment on insane asylum inmates, which found that the nervous system illness beriberi occurred only in subjects who ate polished, as opposed to unpolished, rice; we now know that beriberi is caused by a deficiency of vitamin B1). The term vitamin, originally "vitamine," was coined by Polish scientist Casimir Funk as a combination of the Latin term for life and "amine," which he mistakenly used to refer to the nicotinic acid he isolated from rice husks. See Irwin W. Sherman, *The Power of Plagues* 367 (ASM 2006).

89 See Mattson and Calabrese, *Hormesis* at 1, 6–7 (cited in note 88).

90 See, for example, id at 3 fig 2 (providing dose-response curves for two chemicals: glutamate and carbon monoxide). See also Part II.

91 For a discussion of the development of modern quantitative risk assessment, see Part I.B.

92 Federal agencies that play a significant role in pollution control regulation include the Occupational Safety and Health Administration (OSHA), which creates and also enforces laws to manage workplace safety from pollution and other sources; the US Department of Agriculture (USDA), which handles a number of environmental and natural resource issues as part of its mandate to manage US agriculture; the Food and Drug Administration (FDA), which manages pollution exposures in food, drugs, and cosmetics; the Department of the Interior (DOI), which is responsible for managing pollution control on public lands; the Department of Transportation (DOT), which manages the transport of hazardous materials; the Department of Energy (DOE), which administers the National Energy Conservation Policy Act; the Nuclear Regulatory Commission, which manages atomic material; the US Army Corps of Engineers (USACE), which manages the permit program for dredge and fill activities under the Clean Water Act; the Department of Housing and Urban Development (HUD), which handles housing and siting issues, including management of contaminated and potentially contaminated "brownfields"; National Institute for Occupational Safety and Health (NIOSH), which creates recommended standards for workplace safety and worker health; and the Council on Environmental Quality (CEQ), which coordinates federal environmental policy. State environmental agencies and departments of health also play key roles in pollution control.

into their decision making. Nowadays EPA maintains an Integrated Risk Information System (IRIS), which provides dose-response data on hundreds of regulated substances.[93]

How do environmental agencies use dose-response data? Use of this data is typically focused on determining safe and/or tolerable levels of exposure—levels at which there are no, or limited, observed harmful effects.[94] These threshold determinations are then used to inform regulatory decisions that require standards to be set at zero-risk levels, for example, the setting of a Maximum Contaminant Level Goal (MCLG) for a given pollutant under the Safe Drinking Water Act,[95] and to inform technological standards.[96] Another example of the use of the toxicological data would be in setting the reference dose (RfD),[97] which is "[g]enerally used in EPA's noncancer health

93 As of this writing, IRIS included dose-response data on over 550 substances. See EPA, *IRIS* (cited in note 54). This is only a tiny fraction of the total substances in the world, but it still represents a significant compendium of dose-response data.

94 For example, through calculating a substance's No Observed Adverse Effect Level (NOAEL) or its Lowest Observed Adverse Effect Level (LOAEL).

95 Pub L No 93-523, 88 Stat 1660 (1974), codified as amended at 42 USC § 300f.

96 For example, the Maximum Contaminant Level (MCL) is set by reference to the MCLG. See 42 USC § 300g-1(b)(4)(B).

97 The reference dose (RfD) is

> [a]n estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used.

IRIS Glossary/Acronyms & Abbreviations (EPA July 26, 2011), online at http://www.epa.gov/iris/help_gloss.htm#r (visited Sept 23, 2012). In addition to the NOAEL or LOAEL, the RfD incorporates two measures of uncertainty: the Standard Uncertainty Factor (UF), which measures the uncertainty in sensitivity by using extrapolations from animal studies and from short-term studies, and the Modifying Factor (MF), which is a catch-all subjective uncertainty adjustment. More formally, RfD = NOAEL / (UF * MF). See *Terminology Services--Vocabulary Catalog List Detail Report: Reference Dose* 1.3.2.3 (EPA Sept 23, 2012), online at http://ofmpub.epa.gov/reports/rwservlet?termepa&report=glossaryDetailReport.jsp&desformat=HTMLCSS&p_xml_data=http%3A%2F%2Fofmpub.epa.gov%3A80%2Fsor_internet%2FterminologyExport%2F%3Ftype%3DglossaryDetail%26key%3DXF5SEGY6b2Zgercq76EeK195D8EYl3W6fdvWBih%252Fjck%253D%26p_xml_schema%3Dhttp%3A%2F%2Fofmpub.epa.gov%2Fsor_internet%2Fxml%2FterminologyReport.xsd&pagestream=yes&desname=TerminologyServicesReport.htmlcss&p_criteria=%20Search%20Terms%20%28Contains%29%3A%20reference%20dose%20 (visited Sept 23, 2012). EPA uses similar measures of health effects that are based on toxicological data for other forms of exposure as well; the reference concentration (RfC), for example, provides an estimate of a safe dose of inhaled substance. See *Terminology Services: Reference Dose* (cited in note 97).

assessments,"[98] for example, for pesticides that lead to endocrine disruption.[99]

While use of toxicological data is deeply embedded in EPA's risk analyses, the data is used to inform the determination of single levels, like RfDs and reference concentrations (RfCs), which are meant to act as thresholds for regulation. This is a valuable use of the data, but the IRIS glossary, which contains over 150 terms relevant to EPA's Integrated Risk Assessment System, does not have a single term addressing the relationship between the amount of harm a substance causes and the way it is allocated across the population. In the next Part, I will show that this is a dangerous mistake.

II. EXPOSURE ALLOCATION

If exposure to a pollutant determines the harm (or benefit) caused by that pollutant, what determines exposure? Or put another way, what kinds of policy interventions will tend to decrease the harm from pollution exposure?

One seemingly obvious method for reducing the harm from pollution exposure is to *reduce* pollution exposure, either by pollution reduction (by reducing the total amount of pollution emitted into the world)[100] or by exposure reduction (reducing the total amount of pollution to which individuals are exposed).[101] These approaches have

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98 See *Terminology Services: Reference Dose* (cited in note 97).

99 See *Endocrine Disruptor Screening Program (EDSP)* (EPA July 11, 2012), online at http://www.epa.gov/endo (visited Sept 23, 2012).

100 This would include approaches that seek to limit or eliminate pollution emissions. Many of the major provisions in many of the major environmental statutes employ this strategy, including the National Pollutant Discharge Elimination System (NPDES) under the Clean Water Act § 402, 86 Stat at 880–83; the NAAQS, the powerhouse of the Clean Air Act, established by the Clean Air Amendments of 1970 § 109, Pub L No 91-604, 84 Stat 1676, 1679–80, codified at 42 USC § 7409; the regulation of HAPs, under which EPA must establish emission standards that require the maximum degree of reduction in emissions of HAPs per the Clean Air Act Amendments of 1990 § 112, Pub L No 101-549, 104 Stat 2399, 2539, codified at 42 USC § 7412(d)(2); the MCL and MCLG standards set under the Safe Drinking Water Act § 1401, 88 Stat at 1661; and the Pollution Prevention Act of 1990, Pub L No 101-508, 104 Stat 1388, codified at 42 USC § 13101 et seq.

101 This would include approaches that seek to limit or eliminate how much pollution people are exposed to. It would include, for example, the portions of the Resource Conservation and Recovery Act, Pub L No 94-580, 90 Stat 2795 (1976), codified at 42 USC § 6901 et seq, which regulate hazardous waste transportation, treatment, storage, and disposal; the Emergency Planning and Community Right-to-Know Act of 1986, Pub L No 99-499, 100 Stat 1733, codified at 42 USC § 11001 et seq, insofar as it seeks to ensure that communities would know if they were being exposed to emergency pollution exposures so that they could take mitigating or avoidance actions; and the OSH Act § 6, 84 Stat at 1593–97, insofar as it seeks to limit worker exposure.

intuitive affective appeal and undergird the vast majority of current approaches to pollution policy.

But while pollution reduction and exposure reduction are powerful tools, they are not the only possible mechanisms for reducing the harm from pollution. This Article argues that another way to reduce the harm from pollution is to focus not on the aggregate *amount* of pollution exposure, but on the way pollution exposure is distributed across a population: exposure *allocation*. Because different substances exhibit different dose-response relationships and because for most of these relationships the harm caused by pollution exposure varies depending upon concentration, so long as it is possible to change who is exposed to pollution, it will sometimes be possible to exchange high-harm exposures for low-harm exposures.[102] This means that, for many pollutants, it will be possible to *reduce the harm from pollution without reducing the total amount of pollution emitted.*

The key to building exposure allocation policies that can reduce the harm from pollution is to match allocation strategies with the specific characteristics of the pollutants being managed. Dose-response relationships vary across substances, and different dose-response relationships implicate different exposure allocation strategies. The remainder of this Part goes through illustrations of five types of dose-response relationships that represent five common types of pollutants.[103] Each of these dose-response relationships has different implications for a pollution policy that seeks to reduce the harm from pollution.

A. The Simplest Story: Linear Nonthreshold Dose-Response Curves

Let us begin with the simplest form of dose-response relationship—and the *only* dose-response relationship for which exposure

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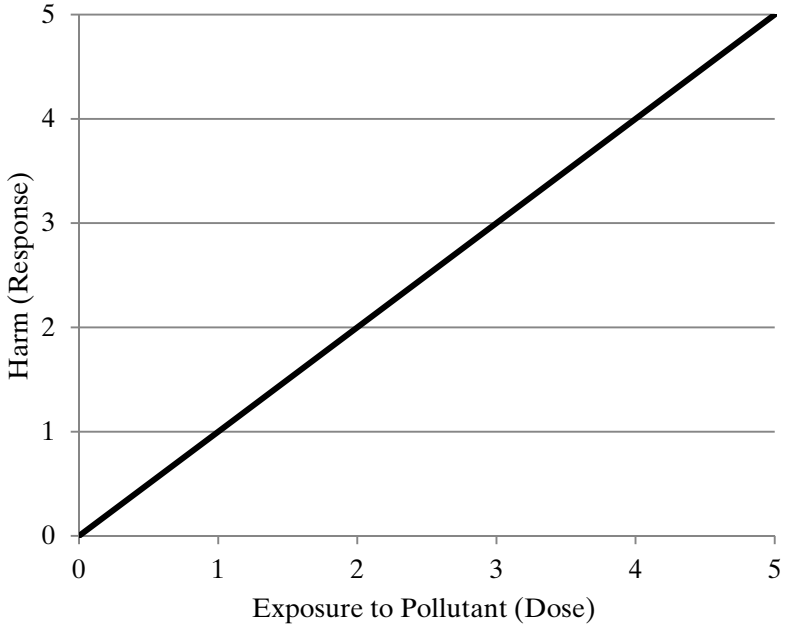
102 Note that in many contexts there may still be distributional, or fairness, objections to shifting pollution exposure from one person to another. Analyses of exposure allocation can help inform distributional analysis, because exposure allocation will frequently determine how much harm is caused by a set amount of pollution. In asking whether the people who bear that harm can fairly be asked to do so, distributional analysis is complementary to exposure allocation analysis. But the two analyses are logically separable, since exposure allocation tells us *how much* harm is caused by a given allocation of pollution exposure across a population, and a distributional analysis tells us whether a given allocation of pollution exposure across a population meets an acceptable standard of fairness. See Part IV (discussing fairness concerns).

103 The dose-response relationships were selected based on their prevalence in the toxicology literature and on their use by EPA.

allocation will prove to be irrelevant to the harm caused by pollution exposure.

Imagine a substance where response (in this case, harm) increases proportionally with dose (or the amount of pollutant to which the organism is exposed).[104] For this substance, the relationship between dose and harm is 1:1, so that for every one unit of pollution distributed, there will be one unit of harm. If we were to display the dose-response relationship for this type of substance as a line, it would look something like this:

EXAMPLE 1. LINEAR NONTHRESHOLD DOSE-RESPONSE CURVE



104 See Daniel L. Eaton and Steven G. Gilbert, *Principles of Toxicology*, in Klaassen, ed, *Toxicology: The Basic Science of Poisons* 11, 19–26 (cited in note 10). EPA operates on the default assumption that carcinogens exhibit this type of dose-response curve. See EPA, *Guidelines for Carcinogen Risk Assessment* 1-12 to -15 (Mar 2005), online at http://www.epa.gov/raf/publications/pdfs/CANCER_GUIDELINES_FINAL_3-25-05.pdf (visited Sept 23, 2012). For a criticism of this approach, see NRC, *Silver Book* at 127–34 (cited in note 48). The assumption of a linear nonthreshold dose-response relationship has been successfully challenged for some suspected carcinogens. See, for example, *Chlorine Chemistry Council v EPA*, 206 F3d 1286, 1290 (DC Cir 2000) (overturning EPA's decision to treat chloroform, a suspected carcinogen, as if it exhibited a linear nonthreshold dose-response curve).

Imagine now that we need to distribute some set amount of this pollutant—in this case, three units—across three persons: Tom, Dick, and Larry. And for now, let us imagine that allocation of the pollution is wholly within our power as policy makers.[105]

We might imagine a number of different exposure allocations, but for this very specific type of substance—for substances where there is harm at any measurable quantity of exposure, and all additional exposure quantities result in proportional increases in harm—there will be the same total amount of harm caused regardless of who is left holding the bag.

To see this, start by imagining that we adopt what I will call a "spreading" strategy, where we spread the pollution exposure equally across the target population. Under this approach, with three units of pollution to allocate and three persons, we would end up exposing each person to one unit of pollution exposure. Each person would be harmed one unit of harm, for a total harm of three ($1 + 1 + 1 = 3$).

In contrast, we could adopt what I will call a "bunching" strategy, where we bunch the pollution exposure on a subset of the target population. Under this approach, we would choose one unlucky person to be exposed to all three units of pollution. The exposed person would be harmed three, for a total harm of three ($3 + 0 + 0 = 3$).[106]

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105 This is a simplifying assumption. There are obviously practical and important constraints on policy makers' ability to parcel out pollution exposure. That said, I argue in Part III that there are opportunities for policy makers to affect—even if not definitively determine—exposure allocation.

106 Of course there are a number of other potential allocation strategies as well. For example, we could imagine a "mixed" strategy, where we allocate the pollution exposure as between Dick and Larry, and let Tom off the hook entirely. But regardless of the allocation strategy, for this type of pollutant, three units of pollution exposure will result in three units of harm.

TABLE 1. HARM BY EXPOSURE ALLOCATION STRATEGY FOR IDEALIZED LINEAR NONTHRESHOLD POLLUTANT

	Spread		Bunch	
	Dose	*Harm*	*Dose*	*Harm*
Tom	1	1	0	0
Dick	1	1	0	0
Larry	1	1	3	3
Total Harm		**3**		**3**

Both of these allocation strategies result in the same amount of total harm caused. And in fact, for this kind of substance, every possible allocation strategy will result in the same amount of harm. Reallocating the pollution—through legal or other means—will therefore do little except pass the buck: we can only decrease the amount of harm caused by decreasing the amount of pollution and/or the total amount of pollution exposure to be allocated.[107] Of course, for political or personal reasons, we might still prefer one allocation to another—Tom and Dick, for example, have an interest in promoting a bunching strategy over a spreading strategy, so long as they can ensure that Larry is the one holding the short end of the stick. If Larry is otherwise disadvantaged, we as policy makers might choose not to burden him with additional harm because we think it would be distributionally unfair.[108] Or if one of them is relatively politically powerful, he might be able to successfully lobby to ensure that one of the others bears the brunt of the exposure.[109] Any of these outcomes may be more or less desirable for normative, political, or moral reasons, but none of the possible exposure allocations would lead to a

107 See James K. Hammitt, *Economic Implications of Hormesis*, 23 Hum & Experimental Toxicology 267, 276 (2004) (noting that "[u]nder the linear no-threshold model, the total health effects within a population depend solely on the change in total exposure," and comparing this to the harm implications of hormetic substances).

108 For a careful argument that fairness requires consideration of existing welfare, see Adler, *Well-Being and Fair Distribution* at 314–38 (cited in note 69).

109 Which is to say that interest groups and power politics might play an important role in determining who ends up holding the short end of the stick. For an introduction to political choice and interest group politics, see generally Daniel A. Farber and Philip P. Frickey, *Law and Public Choice: A Critical Introduction* (Chicago 1991).

different amount of aggregate harm so, at least on harm reduction grounds, no outcome is preferable to another.

If all substances exhibited exactly this relationship between dose and response—where harm occurs at all levels of exposure and increases proportionally with each incremental dose—policy makers could determine the harm caused by pollution exposure without determining how that pollution exposure was allocated across the population. And any policies that led to one final exposure allocation over another would have to be debated based upon grounds other than harm reduction, such as whether the allocation was fair. As we shall see, however, there are many other types of relationships between dose and response, and for *all* of these other relationships, exposure allocation is a key determinant of the amount of harm caused by a set amount of pollution.

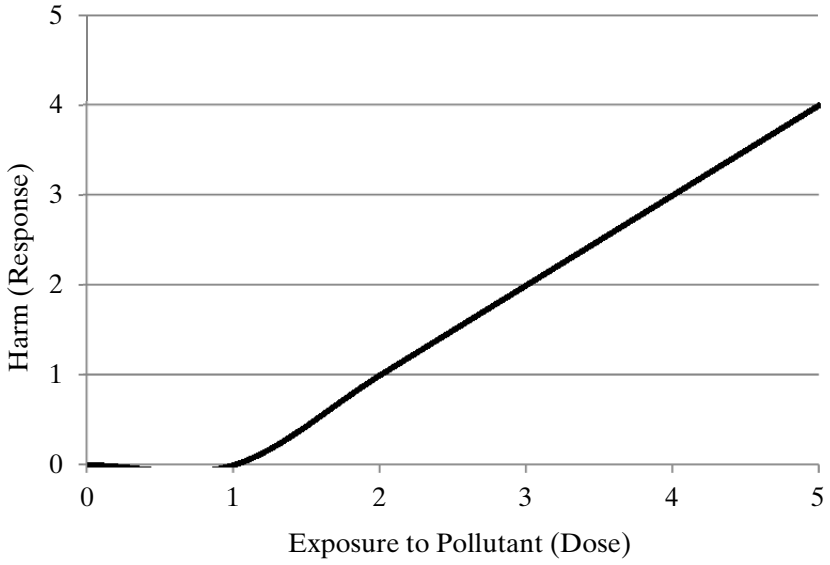
B. Threshold Pollutants: The Possibility of Harmless Pollution

Now let us consider a slightly different substance, of the form sometimes referred to as a "threshold" pollutant. For these substances, there are detectable quantities of exposure that cause no detectable harm.[110] And as we shall see, for these substances—as with all substances that do not exhibit the proportional dose-response relationship of Example 1—the exposure allocation of the substance will be a key determinant of the total amount of harm caused by any given quantity of the substance.

Since threshold pollutants have some dose at which they cause no measurable harm, we can imagine an idealized dose-response curve that would look something like this:

110 See Eaton and Gilbert, *Principles of Toxicology* at 13 (cited in note 104).

EXAMPLE 2. THRESHOLD DOSE-RESPONSE CURVE



This dose-response curve is much like the one portrayed in Example 1, except that there is no measurable harm at one unit of pollution.

EPA currently assumes a threshold model for noncarcinogenic pollutants,[111] and for some suspected carcinogens.[112] In many circumstances, EPA has used the existence of threshold pollutants to justify adoption of lower minimum and higher maximum exposure standards, reasoning that health-protective, environmental regulations do not require industry, for example, to decrease the exposures it causes past the point of no harm.[113] This response has led to a politicization of threshold pollutants, with proponents for industry arguing that EPA should change its current baseline assumption that pollutants have no safe threshold of exposure, so that more pollutants will be regulated under the more permissive threshold standards, and environmentalists arguing the opposite.

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111 See NRC, *Silver Book* at 128 (cited in note 48).

112 Although EPA has a default assumption that the dose-response curves for carcinogens are linear and nonthreshold, this assumption has been overborne for a few substances. See *Chlorine Chemistry Council*, 206 F3d at 1290 (overturning EPA's decision to set a maximum contaminant level goal for chloroform, a suspected carcinogen, at zero).

113 See, for example, Revisions to the Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health, 65 Fed Reg 66444, 66446 (2000) (describing how EPA sets MCLGs above zero for drinking water contaminants, except where there is strong evidence of carcinogenicity).

What has been lost in this debate, however, is that the difference between Example 1 and Example 2 creates a win-win opportunity to keep pollution emissions the same while ensuring that *no one is harmed by the pollution*.

Suppose we wish to distribute three units of this threshold pollutant between Tom, Dick, and Larry. We could choose to implement a spreading strategy, such that each person is exposed to a small quantity of the pollutant. If each person is exposed to one unit of the pollutant, they will each fall within the safe threshold of exposure and be unharmed. As a result, exposure to the three units of pollution would cause zero harm (0 + 0 + 0 = 0).

In contrast, imagine that we failed to see that a spreading strategy would be helpful here, and we adopted a policy that led to the opposite exposure allocation: to bunching the pollutant, such that a small number of people would be exposed to large amounts of it. Keeping the number of people and the amount of pollutant constant, this would mean that one of the people exposed would receive three units of exposure to the pollutants and harm of two (0 + 0 + 2 = 2)—two harm more than would have resulted from the same amount of pollution with a different exposure allocation. This means that poor exposure allocation will cause *more harm with the same amount of pollution*—what I will call "supplemental harm."

TABLE 2. HARM BY EXPOSURE ALLOCATION STRATEGY FOR IDEALIZED THRESHOLD POLLUTANT

	Spread		Bunch	
	Dose	*Harm*	*Dose*	*Harm*
Tom	1	0	0	0
Dick	1	0	0	0
Larry	1	0	3	2
Total Harm		**0**		**2**

In other words, for threshold pollutants, adopting an exposure allocation strategy of spreading—of exposing many people to small amounts of the pollutant—can decrease the quantity of aggregate harm caused.[114] And this is possible *without* reducing the amount of

[114] For substances that implicate spreading strategies, the "solution to pollution" really could—at least in theory—be "dilution."

pollution: without changing the quantity of pollution emitted or the quantity of pollution to which people are exposed. Furthermore, because there is a safe threshold, where the quantities of pollution to be distributed are small enough, there will even be the possibility of spreading the pollution to the point of harmlessness.[115] At the same time, a careless or foolish exposure allocation may well lead to needless suffering by creating supplemental harm.

C. Hormetic Dose-Response Curves: Converting Harm into Benefit

Now let us consider a third type of substance: the "hormetic," or biphasic, substance.[116] These substances have the capacity either to help or to hurt human health: in small quantities they provide benefits, but as the dose of any of these substances increases, at some point they become harmful—even deadly.[117]

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[115] Insofar as the spreading of the pollution is treated as costless and the quantities of the pollutant are small enough to keep all exposures subthreshold, this is a Pareto-optimal improvement: no one is harmed, and some (perhaps many) are helped. For a discussion of Pareto optimality, see Richard A. Posner, *Economic Analysis of Law* 12–13 (Aspen 7th ed 2007) (establishing unanimity among affected parties as the criterion of a Pareto-superior transaction). Note that a pure spreading strategy, such as the ones I discuss here, spreads exposure to the level at which each person is exposed to the same amount of pollution. We could also imagine "mixed" strategies that seek only to spread below the safe threshold and otherwise allow some heterogeneity of exposure.

[116] Compared to other dose-response relationships, a significant (although still limited) amount of scholarship by legal academics has addressed the phenomenon of hormesis. Most of this scholarship came in the early 2000s, as there was growing attention in the toxicology community to the possibility that hormetic substances—once thought to be relatively rare—might actually be common or even the norm. See, for example, Edward J. Calabrese and Linda A. Baldwin, *Hormesis: The Dose Response Revolution*, 43 Ann Rev Pharmacology & Toxicology 175, 176 (2003). For a provocative and important analysis of the legal implications that would come from shifting from an assumption that most substances exhibit linear nonthreshold dose-response relationships (in line with Example 1) to the default assumption that substances are hormetic, see F.B. Cross, *Legal Implications of Hormesis*, 20 Hum & Experimental Toxicology 122, 124–27 (2001), and responses at 20 Hum & Experimental Toxicology 129, 129–68. See also Hammitt, 23 Hum & Experimental Toxicology at 267–77 (cited in note 107), and responses at 23 Hum & Experimental Toxicology 279, 279–305; Cass R. Sunstein, *Risk and Reason: Safety, Law, and the Environment* (Cambridge 2002) (discussing other dose-response shapes and the assumptions used in regulating them); Sunstein, 90 Georgetown L J at 2279–83 (cited in note 29); Jonathan B. Wiener, *Hormesis, Hotspots and Emissions Trading*, 23 Hum & Experimental Toxicology 289, 289–300 (2004) (discussing the policy implications of hormetic dose-response curves); Cass R. Sunstein, *Precautions against What? The Availability Heuristic and Cross-Cultural Risk Perception*, 57 Ala L Rev 75, 80–87 (2005) (warning that making linear assumptions when hormetic assumptions should be made can cause harm).

[117] The individual characterization of the toxicology of many substances remains controversial, as does the answer to the question of whether hormetic dose-response relationships are exceptional. At least some toxicologists argue that most substances are hormetic. See *Special Issue on Hormesis*, 3 Am J Pharmacology & Toxicology 1, 1–192 (2008); Mattson and Calabrese,

This may at first seem counterintuitive, but consider the familiar idea of an overdose. A prescription medication, taken daily in the specified amount, is expected to provide a benefit. That is the reason for the prescription. But the same substance ingested too freely may cause grave illness, even death.

Many vitamins also exhibit hormetic dose-response relationships. We need vitamin D to grow and maintain bones, but too much can cause hypercalcemia, which is an excess of calcium in the blood; this can cause damage to a number of vital systems, including the kidneys.[118] Vitamin A can cause liver damage and can harm the cardiovascular system, but without it we cannot maintain our bodily organs.[119] Iodine, which is a deadly poison, is routinely added to table salt worldwide.[120] Selenium, iron, chromium, and zinc are all essential to human health, even as they are toxic and even deadly in larger quantities.[121] Other hormetic stressors include (arguably) sunshine, mercury, arsenic, heat, pesticides, carbon monoxide, exercise, food, and water.[122]

This Article is not concerned with establishing that any particular substance is hormetic or with establishing any particular frequency of the hormetic relationship.[123] The point is simply that there are

Hormesis at 1–14 (cited in note 88). Nevertheless, the prevailing view appears to be that, although some substances—such as vitamins—clearly exhibit hormetic dose-response curves, it is too early to conclude that most substances are hormetic. See Jocelyn Kaiser, *Sipping from a Poisoned Chalice*, 302 Sci 376, 376–79 (2003); Deborah Axelrod, et al, *"Hormesis"—An Inappropriate Extrapolation from the Specific to the Universal*, 10 Intl J Occupational & Envir Health 335, 335–39 (2004).

118 For a discussion of various hormetic substances, see generally Mattson and Calabrese, *Hormesis* (cited in note 88). Note that the United States routinely supplements milk with vitamin D. See Mona S. Calvo, Susan J. Whiting, and Curtis N. Barton, *Vitamin D Fortification in the United States and Canada: Current Status and Data Needs*, 80 Am J Clin Nutr 1710S, 1711S–13S (Supp 2004).

119 Mattson and Calabrese, *Hormesis* at 1–2 (cited in note 88).

120 Iodine deficiency is the world's most prevalent cause of brain damage. See World Health Organization, *Micronutrient Deficiencies: Iodine Deficiency Disorders*, online at http://www.who.int/nutrition/topics/idd/en (visited Sept 23, 2012). Iodine deficiency can also lead to goiter, an enlargement of the thyroid. See Roy D. McClure, *Goiter Prophylaxis with Iodized Salt*, 82 Science 370, 370 (1935).

121 Selenium is a trace element that is necessary for the maintenance of selenoproteins, blood proteins that are necessary to survival; too much leads to selenium poisoning and, potentially, death. Iron is necessary for red blood cells, but too much can damage bodily tissue. See Mattson and Calabrese, *Hormesis* at 2 (cited in note 88).

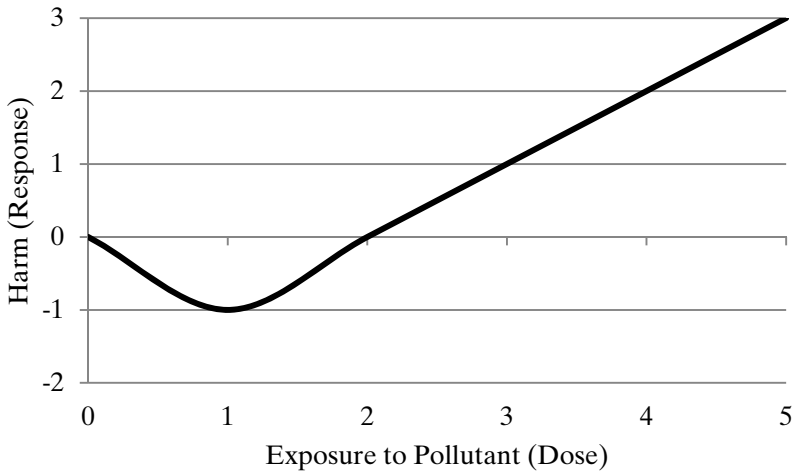
122 See generally id.

123 Note that the particular scientific subfield used to categorize a substance may matter to whether a substance is categorized as hormetic or not. For a discussion of how different uses of different sciences can affect environmental law, see Eric Biber, *Which Science? Whose Science? How Scientific Disciplines Can Shape Environmental Law*, 79 U Chi L Rev 471, 512–44

such substances in sufficient amount that they matter for purposes of legal policy, and that once substances are identified as hormetic, there is an opportunity for win-win arbitrage such that harms from pollution are converted into benefits.[124]

To see this, consider an idealized hormetic dose-response curve:[125]

EXAMPLE 3. HORMETIC DOSE-RESPONSE CURVE



For a substance with this type of dose-response curve, at one unit of pollution we would expect to observe *negative* harm (that is, some benefit); at two units, no apparent effects; and at three units, one unit of harm. For these substances, the allocation decision will not

(2012) (explaining how different scientific disciplines can come to different conclusions about the same sets of facts).

124 In other words, redistributing pollutants with hormetic dose-response curves has the potential of creating Pareto-optimal improvements. See Yew-Kwang Ng, *Welfare Economics: Towards a More Complete Analysis* 26–37 (Palgrave Macmillan 2004); Posner, *Economic Analysis of Law* at 12 (cited in note 115); Hammitt, 23 Hum & Experimental Toxicology at 274 (cited in note 107). See also Cross, 20 Hum & Experimental Toxicology at 126–27 (cited in note 116).

125 Note that this is one possible hormetic curve. Vital nutrients, such as iodine and vitamin D, will exhibit a curve such that there is actually measurable harm at zero exposure—that is, where there is a deficiency. See Calvo, Whiting, and Barton, 80 Am J Clin Nutr at 1711S (cited in note 118). The same reasoning applies to this type of hormetic curve: to reduce aggregate harm, policy makers should seek to spread where incremental harm is increasing and to bunch where incremental harm is decreasing.

only determine who is harmed; it may also determine *whether there is harm or benefit*.

Imagine again that we must allocate three units of pollution among Tom, Dick, and Larry. If we adopt a spreading strategy by allocating one unit to each person, each of those people would gain a benefit of one, for a total *benefit* of three ($-1 + -1 + -1 = -3$).

In contrast, if we were to bunch all three units on unlucky Larry, he would accrue a harm of one for an aggregate *harm* of one ($0 + 0 + 1 = 1$). Adoption of a bunching strategy would therefore lead to supplemental harm and would, moreover, convert an aggregate benefit into an aggregate harm.

TABLE 3. HARM BY EXPOSURE ALLOCATION STRATEGY FOR IDEALIZED HORMETIC POLLUTANT

	Spread		Bunch	
	Dose	*Harm*	*Dose*	*Harm*
Tom	1	−1	0	0
Dick	1	−1	0	0
Larry	1	−1	3	1
Total Harm		**−3**		**1**

For hormetic substances, therefore, reallocation has the potential to create win-win situations that reduce harm and confer measurable benefits. Furthermore, in contrast to the general intuitive feeling that it is always better to reduce pollution exposure, for hormetic substances increasing pollution (emission and exposure) has the potential to increase aggregate benefits.[126]

D. Curvilinear Dose-Response Curves: Opportunities for Decreasing Aggregate Harm

Now let us consider dose-response relationships for substances for which the curve is curvilinear rather than linear.[127] For these substances, as we shall see, the allocation of the pollution can have an

126 For further discussion of hormetic substances, see generally Cross, 20 Hum & Experimental Toxicology 122 (cited in note 116); Hammitt, 23 Hum & Experimental Toxicology 267 (cited in note 107); Wiener, 23 Hum & Experimental Toxicology 289 (cited in note 116).

127 See Eaton and Gilbert, *Principles of Toxicology* at 9–14 (cited in note 104).

enormous effect on aggregate levels of harm.[128] But, unless the substance also exhibits a safe threshold, there is not necessarily any opportunity to adopt a Pareto-optimal allocative strategy: *someone* will be harmed by the pollution, and policy makers will have to make interpersonal comparisons to determine who that should be and how to weigh the number of people affected against the amount of harm caused.[129] That said, we can still use the shape of the dose response curve to identify exposure allocation strategies that will decrease aggregate harm.

1. Supralinear curves: opportunities for spreading.

Imagine a substance for which any amount of exposure causes harm, and where each additional unit of exposure causes twice the harm of the previous unit, such as can happen with many cumulative toxins.[130] So at one unit of pollution we would expect one harm; at two units, three harm; at three units, seven harm; and so on.

An idealized dose-response curve for this substance might look like this:

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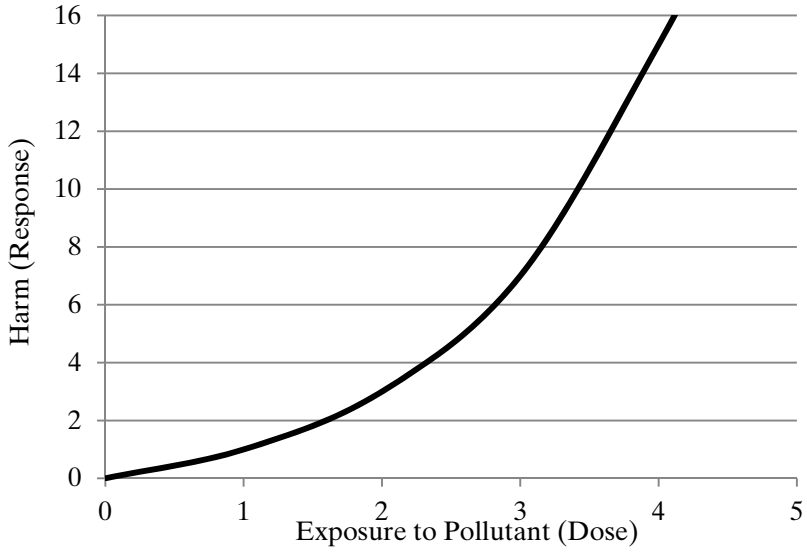
128 Two articles have noted the impact of curvilinear dose-response curves in the context of economic trading regimes. See Jonathan Remy Nash and Richard L. Revesz, *Markets and Geography: Designing Marketable Permit Schemes to Control Local and Regional Pollutants*, 28 Ecol L Q 569, 578–79 (2001) (discussing the social welfare implications of curvilinear dose-response curves, and concluding that "[i]f the public policy objective were to maximize social welfare, the optimal distribution of pollution concentrations in the region would depend on the shape of the damage function—the function linking the pollutant's concentrations to its adverse effects"); Wiener, 23 Hum & Experimental Toxicology at 291–93 (cited in note 116) (discussing the policy implications of supralinear and sublinear dose-response curves on bunching and draining through emissions trading programs and comparing them to linear nonthreshold and hormetic substances).

129 Or in other words, redistributing pollutants with curvilinear dose-response curves has the possibility of creating Kaldor-Hicks improvements in efficiency, such that the winners from a distributional regime could in theory compensate the losers. See J.R. Hicks, *The Foundations of Welfare Economics*, 49 Econ J 696, 712 (1939); Nicholas Kaldor, *Welfare Propositions in Economics and Interpersonal Comparisons of Utility*, 49 Econ J 549, 550 (1939). For an overview of Kaldor-Hicks efficiency (sometimes called potential Pareto efficiency), see Ng, *Welfare Economics* at 26–37 (cited in note 124). For policy applications of Kaldor-Hicks efficiency, see Posner, *Economic Analysis of Law* at 15–16 (cited in note 115).

130 Or in other words, where the marginal harm doubles for every unit increase in dose. For discussion of substances with these kinds of curves, see Elaine M. Faustman and Gilbert S. Omenn, *Risk Assessment*, in Klaassen and Watkins, eds, *Essentials of Toxicology* 47, 51 fig 4-3 (cited in note 10).

EXAMPLE 4. SUPRALINEAR DOSE-RESPONSE CURVE



Now let us consider our potential exposure allocation strategies. Since there is no safe threshold with this type of substance, it is inescapable that—so long as we are not able to reduce either the amount of pollution or the amount of pollution exposure—at least one of our three test subjects will be harmed. But we still have a choice as to how exposures will be allocated across our population of Tom, Dick, and Larry. If we spread the pollution across our three subjects, then each would be exposed to one unit of pollution and each would only sustain one unit of harm, for a total of three units of harm (1 + 1 + 1 = 3).

What if we chose to bunch the exposure on unlucky Larry? In that case, Tom and Dick are unharmed, but because higher concentrations of this substance cause significant additional harm, Larry would be harmed seven (0 + 0 + 7 = 7). This means that, with the same amount of pollution exposure and the same amount of pollution, a bunching strategy would cause more than twice the aggregate harm of a spreading strategy.

TABLE 4. HARM BY EXPOSURE ALLOCATION STRATEGY FOR IDEALIZED SUPRALINEAR POLLUTANT

	Spread		Bunch	
	Dose	*Harm*	*Dose*	*Harm*
Tom	1	1	0	0
Dick	1	1	0	0
Larry	1	1	3	7
Total Harm		**3**		**7**

For substances with supralinear dose-response curves, then, adoption of a spreading strategy will lead to significantly less aggregate harm than adoption of a bunching strategy and, conversely, adopting a bunching strategy would lead to supplemental harm (in this case, a supplemental harm of four ($7 - 3 = 4$), which is more than the total harm that would be caused under a spreading approach).[131] Or more generally speaking, wherever a dose-response curve for a substance has an increasing slope—where incremental harm increases with additional exposure—the harm from pollution can be decreased via spreading.

2. Sublinear curves: opportunities for bunching.

Thus far, all of the curves we have considered have implied that spreading strategies are superior[132]—or at least not inferior[133]—to bunching strategies. But that is not inevitably the case. To see this, let us consider a substance that exhibits a dose-response curve where harm increases with each increased unit of exposure at smaller doses but then levels out in larger quantities. This type of relationship can

131 For an application of the policy implications of supralinear dose-response curves in the context of an economic trading regime, see Nash and Revesz, 28 Ecol L Q at 579 (cited in note 128); Wiener, 23 Hum & Experimental Toxicology at 291 (cited in note 116):

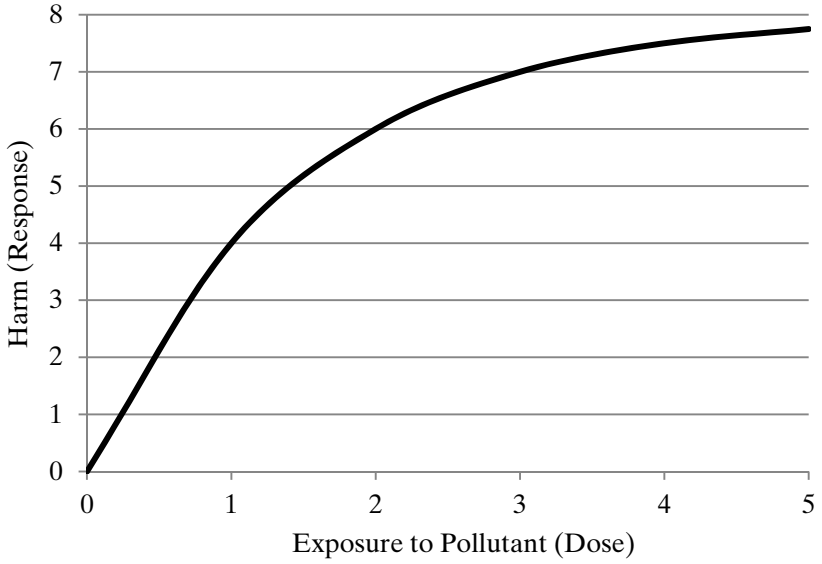
> Under a supralinear dose-response function, which is monotonically increasing (not hormetic) but has increasing marginal harms (each added unit of exposure causes more harm than the prior unit), and with constant marginal exposure from emissions, bunching would yield rising harms, but draining would yield declining harms, compared to a uniform distribution of emissions.

132 As with threshold, hormetic, and supralinear dose-response curves. See Examples 2–4.

133 As with linear nonthreshold substances. See Example 1.

occur with pollutants (like silica or asbestos) that tend to act like pathogens:

EXAMPLE 5. SUBLINEAR DOSE-RESPONSE CURVE



Now we can see that there is a significant difference in the harm caused by our two different idealized exposure allocation strategies, but that for these substances, *bunching* creates less aggregate harm.

If we choose to spread the pollution equally, each person exposed to one unit of pollution will accrue four harm, for a total of twelve aggregate harm (4 + 4 + 4 = 12).

In contrast, a bunching strategy will lead to one person being exposed to relatively significant harm: seven harm under the terms of this curve. But the other two persons would be exposed to no harm at all, which means that the aggregate harm from this policy is seven (7 + 0 + 0 = 7), almost half the *aggregate* harm caused by an exposure allocation strategy based on spreading.

TABLE 5. HARM BY EXPOSURE ALLOCATION STRATEGY FOR IDEALIZED SUBLINEAR POLLUTANT

	Spread		Bunch	
	Dose	*Harm*	*Dose*	*Harm*
Tom	1	4	0	0
Dick	1	4	0	0
Larry	1	4	3	7
Total Harm		**12**		**7**

This means that, for this substance, a bunching strategy causes less aggregate harm, and adoption of a spreading strategy will lead to significant supplemental harm (in this case five (12 − 7 = 5)). And with larger amounts of pollution, or a slope that decreases even more quickly, the impact of exposure allocation strategy on aggregate harm will be even greater.

From a harm-reduction standpoint, then, bunching strategies are superior to spreading strategies wherever a substance exhibits a sublinear dose-response curve.[134] Spreading strategies for these substances should only be adopted when countervailing policy concerns—such as distributional equity, fairness, or cost—are sufficiently strong to outweigh the supplemental harm caused.

3. Composite dose-response curves: a call for tailored strategies.

Not all dose-response curves will fall into only one of the categories above. In fact, many are likely to exhibit what are known as "sigmoidal" curves, which start off slow, accelerate rapidly (as in Example 4), and then level off again (as in Example 5). For composite curves, regulators should recognize that they can reduce the total

134 Professor Jonathan Wiener makes this point in the context of emissions trading. See Wiener, 23 Hum & Experimental Toxicology at 292 (cited in note 116):

> If the dose-response function is sublinear, it flattens out and has declining marginal harms with increasing exposure. In this case bunching (from buying allowances) and draining (from selling allowances) would have the converse orientation to the supralinear case. In the sublinear case, trading would be beneficial on net if buying (bunching) and selling (draining) diverge from the average, so that bunching occurs where the dose-response curve is rising but flattening out, and draining occurs where the dose-response curve is falling more steeply.

harm from a set amount of pollution by spreading pollution exposure when the slope of the dose-response curve is increasing and bunching when the slope of the dose-response curve is decreasing.

III. MANAGING EXPOSURE ALLOCATION WITH LEGAL TOOLS

The previous Part established that, for many pollutants, exposure allocation determines how much harm is caused by pollution (and sometimes whether there is any harm at all). But this observation is helpful for policy purposes and can only lead to an exposure allocation strategy if it is possible to purposefully manipulate the final exposure allocation.

This Part argues that it is possible to change exposure allocations and to do so using familiar legal tools. In fact, environmental justice scholars have been worried for decades about the impact of law on the distribution of environmental harms.[135] While their focus has been on the impact of the law on *who* is affected by distributional policies, and the fairness of those distributions, we can use the same tools to allocate exposure to minimize aggregate harm (or maximize aggregate benefit) from pollution exposure.

The key is to match legal instruments with the toxicology of the pollutants they seek to regulate. We can reduce harm from pollution by adopting legal tools that tend to spread a pollutant whenever higher concentrations of that substance tend to lead to higher marginal harm for the same amount of pollution exposure. This means that we should adopt spreading strategies for curves that correspond to Examples 2, 3, and 4 above: for portions of dose-response curves that are below a threshold and for supralinear curves (that is, curves with an increasing slope).

In contrast, when a curve (or a portion of a curve) corresponds to Example 5 above—when it exhibits a sublinear curve or a decreasing slope such that increasing amounts of exposure are causing less and less additional harm—we can reduce aggregate harm from a set

135 See, for example, Robert D. Bullard, *Introduction*, in Robert D. Bullard, ed, *The Quest for Environmental Justice: Human Rights and the Politics of Pollution* 1, 4 (Sierra Club 2005) (summarizing environmental justice findings over the last few decades); Robert D. Bullard, *Environmental Justice in the Twenty-First Century*, in Bullard, ed, *Quest for Environmental Justice* 19, 30–31 (cited in note 135) (criticizing EPA and other legal decision makers on the basis of findings that low-income groups and people of color are exposed to higher levels of pollution than the rest of the nation). For additional discussion of the environmental justice literature, particularly in regard to siting decisions, see notes 141–46 and accompanying text.

amount of pollution by adopting legal tools that tend to bunch the pollution.[136]

And finally, where a curve is nonthreshold and linear such that it corresponds with Example 1 above, exposure allocation is irrelevant to the amount of harm caused:[137] it will not matter to aggregate harm levels how a set amount of pollution is allocated across the population. That is because, for linear nonthreshold dose-response relationships, a single unit of exposure will always cause the same amount of harm. For these types of substances, any harm reduction will have to come from using the other two mechanisms for reducing the harm from pollution: pollution reduction and/or exposure reduction.[138]

With the exception of hormetic dose-response relationships,[139] vanishingly little has been written on the matching of legal instruments to the toxicology of individual pollutants.[140] Where relevant, I have utilized suggestions from the hormesis literature in the more generalized discussion below, which outlines legal tools with identifiable

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136 There may be serious political choice and fairness concerns with this strategy. For a discussion of these concerns, see Part V. For now, the point is merely that it is possible to reduce aggregate harm by bunching where a dose-response curve is sublinear.

137 See Hammitt, 23 Hum & Experimental Toxicology at 267–70 (cited in note 107); Wiener, 23 Hum & Experimental Toxicology at 291 (cited in note 116).

138 See notes 100–01 and accompanying text.

139 For several very thoughtful analyses of the hormetic dose-response relationship, see Wiener, 23 Hum & Experimental Toxicology at 289 (cited in note 116) (focusing on the relationship between hormesis and instrument choice, and arguing that it is possible to structure emissions trading to be useful even for hormetic substances); Hammitt, 23 Hum & Experimental Toxicology at 276 (cited in note 107) (discussing the policy implications of hormetic dose-response relationships and arguing that hormesis poses problems for emissions trading); Cross, 20 Hum & Experimental Toxicology at 122 (cited in note 116) (arguing that, in comparison to linear dose-response relationships, hormetic relationships sometimes justify more stringent regulations and sometimes justify less stringent regulations), and responses at 20 Hum & Experimental Toxicology 129, 129–68.

140 What analysis is available is tantalizingly imbedded in the discussion of the implications of hormesis. See J.B. Wiener, *Hormesis and the Radical Moderation of Law*, 20 Hum & Experimental Toxicology 162, 162–64 (2001) (arguing—in a single paragraph—that inferences about the hormetic relationship "support a more general proposition: the health benefits of any reduction in exposure will vary directly with the slope of the dose-response curve in the relevant range of exposures. This proposition is important whether one accepts the notion of hormesis or not"), responding to Cross, 20 Hum & Experimental Toxicology at 122 (cited in note 116) (supplying "a tentative and preliminary examination of how existing legal structures can be deployed to acknowledge the reality of hormetic effects and how those structures might best be altered to deal with recognition of hormesis" and providing a model of hormetic effects in the context of legal regulation). See also Wiener, 23 Hum & Experimental Toxicology at 290–93 (cited in note 116) (discussing the viability of an emissions trading regime for welfare-maximizing allocation of hormetic pollutants).

impacts on exposure allocation and recommends the toxicological relationships those tools complement.

A. Siting

A key example of a legal tool for affecting exposure allocation is the one on which the greatest amount of environmental justice work has been done: siting.[141] Much of the early work in environmental justice was focused on the observation that areas with large, poor, and African American communities were highly correlated with the presence of locally undesirable land uses (LULUs) like waste dumps.[142] One prominent Government Accountability Office (GAO) study, for example, found that three-fourths of hazardous waste landfill sites studied through eight southeastern states were located in poor communities of color;[143] as a result, pollutants were concentrated in these areas as well. Initially many commentators assumed that this was the result of racial animus, but empirical work in the 1990s challenged that view.[144] That empirical work has since been

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141 See, for example, Alice Kaswan, *Environmental Justice: Bridging the Gap Between Environmental Laws and "Justice,"* 47 Am U L Rev 221, 237–51 (1997) (discussing siting as an example of a legal effect on distribution); Omar Saleem, *Overcoming Environmental Discrimination: The Need for a Disparate Impact Test and Improved Notice Requirements in Facility Siting Decisions*, 19 Colum J Envir L 211, 222–36 (1994).

142 See, for example, Robert D. Bullard, *Solid Waste Sites and the Black Houston Community*, 53 Sociological Inquiry 273, 285–86 (1983); Government Accountability Office (GAO), *Siting of Hazardous Waste Landfills and their Correlation with Racial and Economic Status of Surrounding Communities*, GAO-B-211461, 1–2 (June 1, 1983), online at http://www.gao.gov/assets/150/140159.pdf (visited Sept 23, 2012).

143 GAO, *Siting of Hazardous Waste Landfills* at 2–4 (cited in note 142). Another influential study, done by the United Church of Christ Commission for Racial Justice in 1987, determined that race was the most significant factor in predicting the likelihood of living near a hazardous waste site. Commission for Racial Justice, *Toxic Wastes and Race in the United States: A National Report on Racial and Socio-Economic Characteristics of Communities with Hazardous Waste Sites* 15–22 (United Church of Christ 1987), online at http://www.ucc.org/about-us/archives/pdfs/toxwrace87.pdf (visited Sept 23, 2012).

144 See, for example, Vicki Been and Francis Gupta, *Coming to the Nuisance or Going to the Barrios? A Longitudinal Analysis of Environmental Justice Claims*, 24 Ecol L Q 1, 9 (1997) (finding no evidence that LULUs were disproportionately sited by wealth of the area, but finding that there was disproportionate siting in Hispanic communities); Vicki Been, *Locally Undesirable Land Uses in Minority Neighborhoods: Disproportionate Siting or Market Dynamics?*, 103 Yale L J 1383, 1383–92 (1994) (arguing that postdecision market dynamics mean that poor and disadvantaged people are disproportionately likely to move *into* an area with a LULU, which indicates that a finding of distributional inequity is not necessarily evidence of bad intent); Vicki Been, *What's Fairness Got to Do with It? Environmental Justice and the Siting of Locally Undesirable Land Uses*, 78 Cornell L Rev 1001, 1009–15 (1993).

challenged as well,[145] and the debate continues as to what proportion of the existing distribution of undesirable uses—and therefore of pollution—is a function of legal decision making and what proportion results entirely from market forces.

Even these commentators agree, however, that law has the power to affect the siting of undesirable—that is, polluting—land uses.[146] And it can do so in numerous ways, including zoning,[147] private and public nuisance law,[148] and granting or denying permits.[149]

How can siting be used to bunch or spread exposures? Basically, by bunching or spreading the sites from which pollution is emitted. Note, however, that bunching or spreading sites is not necessarily the same as bunching or spreading exposure allocation: the degree to which site bunching or site spreading will result in *exposure* bunching or *exposure* spreading will be a function of exposure assessment—which tells us the various vectors from which exposures come[150]—as well as the dispersal characteristics of the particular pollutant in question.[151]

Here it may be helpful to consider the relationship between an analysis of exposure allocation and an analysis of the distributional and environmental justice implications of a particular allocation. While the mechanisms affecting these analyses are the same, the

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145 See, for example, Robert D. Bullard, *The Legacy of American Apartheid and Environmental Racism*, 9 St John's J Legal Commen 445, 460–66 (1994) (challenging Been's methodology and contending that disparities existed when siting decisions were made).

146 See, for example, Been and Gupta, 24 Ecol L Q at 3–9 (cited in note 144).

147 See Charles P. Lord, *Environmental Justice Law and the Challenges Facing Urban Communities*, 14 Va Envir L J 721, 729 (1995) (arguing that even facially neutral zoning ordinances can exacerbate existing concentrations of undesirable land uses, because existing uses are often incorporated into the analysis of whether something is a permitted use).

148 See Andrew Jackson Heimert, *Keeping Pigs out of Parlors: Using Nuisance Law to Affect the Location of Pollution*, 27 Envir L 403, 406 (1997); Emily Sangi, Note, *The Gap-Filling Role of Nuisance in Interstate Air Pollution*, 38 Ecol L Q 479, 481–84, 522–26 (2011).

149 For a list of examples of permit denials on distributional grounds, see Kaswan, 47 Am U L Rev at 250 n 133 (cited in note 141) (noting that an executive order requiring distributional considerations had prevented siting of a uranium enrichment facility, sewage treatment facility, and other projects). Note that many permit programs are administered by federal agencies, including the NPDES permit under § 402 of the Clean Water Act, which is administered by EPA, and the dredge-and-fill permit system administered by the USACE under § 404 of the Clean Water Act. See note 92.

150 Exposure assessment is one of the four processes regulators execute during a quantitative risk assessment. In performing exposure assessments, risk assessors seek to determine the magnitude, frequency, and duration with which humans are exposed to the pollutant in question. See notes 50–54 and accompanying text.

151 See, for example, James Salzman and J.B. Ruhl, *Currencies and the Commodification of Environmental Law*, 53 Stan L Rev 607, 628–29 (2000). See also Wiener, 23 Hum & Experimental Toxicology at 296–99 (cited in note 116) (discussing transference from emission to risk).

analyses themselves focus on different metrics: exposure allocation looks to aggregate harm, whereas environmental justice is concerned with fairness.

To see the distinction, consider how these analyses play out differently in the context of toxic waste disposal. Scholars have noted that modern waste disposal policies have the effect of cleaning up most waste disposal sites while putting additional pressure on the waste disposal sites left in business.[152] Environmental justice analyses of this phenomena have focused on the question of whether it is equitable to disproportionately harm the people who live in communities with waste disposal sites, who in turn are disproportionately minorities and disproportionately poor.[153]

Although it might interact with this analysis in important ways, an exposure allocation analysis of the siting of toxic waste dumps would look different. It would inquire into whether bunching strategies end up causing more aggregate harm to human health than spreading strategies, given the relationship between dose and harm revealed by the relevant dose-response curves. Insofar as the wastes being disposed of are threshold pollutants or exhibit supralinear dose-response curves, this bunching effect is perverse and is leading to unnecessary amounts of harm given the amount of pollutants being distributed. Insofar as the wastes exhibit sublinear dose-response curves, however, a bunching strategy might be a reasonable way to limit aggregate harm (although we might still have objections to the identity of who is being harmed, to the overall quantity of harm being allocated, or to the fairness of exposing a particular disadvantaged group to still greater hardship).[154] Without an analysis of exposure allocation, however, it is impossible to know whether spreading

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152 See, for example, Richard J. Lazarus, *The Meaning and Promotion of Environmental Justice*, 5 Md J Contemp Legal Issues 1, 4 (1994).

153 See Bullard, 9 St John's J Legal Commen at 451–55 (cited in note 145); Kaswan, 47 Am U L Rev at 269 (cited in note 141); Lazarus, 5 Md J Contemp Legal Issues at 4 (cited in note 152) (arguing that aggregation is likely to occur in minority communities due to the vestiges of de jure racism and the communities' lack of political influence).

154 Recall that allocating exposures to sublinear substances by bunching will tend to reduce aggregate harm even where it is not possible to reduce pollution or to reduce exposure. See Part II.D.2. This point is distinct from the reduction in harm that can come from using the traditional mechanisms for reducing environmental harm: pollution reduction (as through cleaning up sites) and exposure reduction (as through preventing people from living on or near contaminated sites). Consider W. Kip Viscusi and James T. Hamilton, *Cleaning Up Superfund*, 124 Pub Interest 52, 56–59 (1996) (criticizing EPA for overinvesting in cleanup of Superfund sites when other options—"such as deed restrictions and other institutional controls that prevent residential areas from being located on hazardous waste sites"—would be effective and cheaper).

or bunching strategies will cause less aggregate harm. To the extent that pollution policy seeks to limit harms to human health, it can use siting and other legal tools to affect exposure allocation and therefore to affect the quantity of harm caused by pollution exposure.

B. Using Pollution Standards to Allocate Pollution

Another option for managing exposure allocation is the classic risk-management technique of setting pollution standards, which forms the foundation of many of the early environmental statutes, including the Clean Air Act[155] and the Clean Water Act.[156] Command-and-control regulation is characterized by the setting of explicit standards, often by reference to available technology. Standards may be set by reference to ambient levels of pollution—National Ambient Air Quality Standards, for example, set a maximum concentration of pollutants in the ambient air[157]—or by reference to the quantities emitted by a particular polluter or class of polluters, as is the case with effluent standards under the Clean Water Act.[158]

Command-and-control statutes can affect exposure allocation in a number of ways, including the setting of standards to include or exclude certain emissions or outputs, the encouragement or requirement of technologies that tend to bunch or spread pollution exposure, and provision of discretion in the allocation of permits.

To see how these different methods might work, let us consider a particular statutory structure, in this case the way the National Ambient Air Quality Standards (NAAQS) are calculated and enforced by EPA under the Clean Air Act.[159] Implementation of the

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155 Pub L No 88-206, 77 Stat 392 (1963), codified at 42 USC § 7401 et seq.

156 Federal Water Pollution Control Act Amendments of 1972 ("Clean Water Act"), Pub L No 92-500, 86 Stat 816, codified at 33 USC § 1251 et seq.

157 See, for example, 40 CFR § 50.4 (establishing that emissions of sulfur dioxide may not exceed 0.4 ppm in a given twenty-four hours).

158 See, for example, 33 USC § 1342 (establishing the National Pollutant Discharge Elimination System (NPDES)).

159 Under the Clean Air Act, EPA is required to set NAAQS at levels "requisite to protect the public health," "allowing an adequate margin of safety," for six criteria air pollutants: carbon monoxide, lead, ozone, particulate matter (PM-10), sulfur dioxide, and nitrogen dioxide. 42 USC §§ 7407–09. These pollutants are commonly found throughout the country. For a summary of current trends in air quality across the nation, see EPA, *Air Trends* (July 24, 2012), online at http://www.epa.gov/airtrends (visited Sept 23, 2012). Another good candidate for the application of exposure allocation analysis is the treatment of Hazardous Air Pollutants (HAPs) under the Clean Air Act. See Clean Air Act § 108, 42 USC § 7408. HAPs can create "migrating toxic hot spots" that expose groups of people to very high levels of hazardous pollutants. See generally McGarity, 86 Tex L Rev 1445 (cited in note 52) (outlining the HAPs

NAAQS is primarily left to the states, which develop State Implementation Plans (SIPs) to identify strategies for meeting the standards. EPA then monitors the states to determine whether states' plans are sufficient, whether the states are implementing their plans, and whether they are meeting the national standards.[160]

This structure gives states significant discretion to determine how to implement the NAAQS. One way states could use their discretion is to determine preferred exposure allocations for criteria pollutants and to allocate emissions accordingly, either spreading or bunching as desired. And because states are responsible not only for the promulgation of their SIPs but also for their implementation, states have opportunities to make back-end adjustments in implementation as they see how their plans are playing out.[161]

The federal EPA can also play a role in exposure allocation through standard setting. One key federal concern under the Clean Air Act arises when air ignores state lines and pollution travels from one state to another. How should interstate pollution be counted for the purposes of determining whether a state has met its SIP?

This is a common question, and the typical answer is that a state is not penalized for failing to comply with NAAQS when EPA believes that the state would have complied but for out-of-state pollution.

On the flip side, the polluting state is not penalized either, creating an unfortunate set of incentives that essentially amounts to each state being incentivized to pollute, so long as the pollution is carried

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regime, explaining how hot spots can arise, and arguing that hot spots can be addressed through "data-driven" technologies).

Exposure allocation is particularly helpful to policy analyses of hot spot formation insofar as it can identify the substances for which hot spots are likely to cause supplemental harm (that is, substances with thresholds and substances with supralinear dose-response relationships—Examples 2 and 4 above—and hormetic substances where the harm threshold has been exceeded) and those for which hot spots are likely to cause less aggregate harm than if the same pollution were more spread across the exposed population (that is, substances exhibiting sublinear dose-response relationships—Example 5 above—and vital nutrients and other hormetic substances where deficiency may cause harm). For this reason, it is critical that policy makers seeking to address hot spots—of HAPs or of other pollutants—do an exposure allocation analysis. Otherwise, a "successful" amelioration of a hot spot could have the unintended and perverse consequence of actually causing *more* harm than if existing exposure allocations had been allowed to continue.

160 See 42 USC § 7410. See also 40 CFR § 50.1 et seq.

161 See Sidney A. Shapiro and Robert L. Glicksman, *Risk Regulation at Risk: Restoring a Pragmatic Approach* 158–77 (Stanford 2003) (arguing that back-end adjustments like this one play a central role in the NAAQS and other regulatory schemes because they allow for case-by-case adjustments). For a further discussion of slippage, see Part III.C.

(by air or by water—similar problems arise under the Clean Water Act) across state lines.

The point here is not to defend or even criticize this effect; it is merely to point out that a state's implementation of a statute like the Clean Air Act is likely to vary depending upon how compliance is calculated. If out-of-state pollution is not counted in a state's emissions, the state will be likely to site emitting facilities near the border of the state and to institute technological measures like tall stacks, which emit pollutants higher into the atmosphere so that they are caught by stronger winds and pulled farther away.[162] Thus technological means—like tall stacks—can also be used to "spread" pollution, just as shorter stacks will tend to lead to a more bunched exposure allocation.[163] In many contexts, individual states can choose to implement these technologies (or not) and, insofar as states are focused on harm created within their own borders, they might not always choose exposure allocation regimes that minimize aggregate harm. As in other contexts where states might cause negative externalities, one solution to this problem is to have federal involvement, in this case in exposure allocation strategy.[164]

Opportunities for states to externalize pollution are available across time as well as space.[165] Consider the case of wildfire policy. Fire policy in the western United States can be simplified into two basic choices: implement small, planned burns, which burn up the fuel of grass, bracken, and other ground detritus in small doses over long durations; or do not implement planned burns, in which case the fuel will build up until some future time, when it will be ignited by lightning, a careless cigarette butt, or an unquenched campfire.[166] The

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162 See Richard L. Revesz, *Federalism and Interstate Environmental Externalities*, 144 U Pa L Rev 2341, 2349–58 (1996).

163 See id at 2350–53.

164 See id.

165 Note that, because pollution exposure and harm are not synonymous, externalizing *pollution* is not necessarily harmful, either because not all pollution leads to exposure or because the pollution exposures created are below the safe threshold of exposure. In the context of nonthreshold pollutants, however, the issue is more fraught. If we assume that the same amount of exposure occurs regardless of allocation, then externalizing nonthreshold pollutants will lead to some level of harm, and the only question is how much harm will be created. In these contexts, we might want to think about intertemporal externalities differently than interstate externalities, as one important mechanism for managing potential interstate conflictnegotiation—is not available, or at least operates very differently, in the intertemporal context. See Cass R. Sunstein and Arden Rowell, *On Discounting Regulatory Benefits: Risk, Money, and Intergenerational Equity*, 74 U Chi L Rev 177, 194–96 (2007).

166 For more on fire policy in the western United States, see generally Karen M. Bradshaw and Dean Lueck, eds, *Wildfire Policy: Law and Economics Perspectives* (Resources for the Future 2012).

latter policy results in less frequent but more intense burns. Besides the potential for additional property loss as a result of these burns and the chance that people will lose their lives in the more intense fires, another difference is that airborne particulate matter levels vary widely between the two policies.[167] Particulate matter released in planned burns will be at a lower level and will disperse after a short period of time.[168] Particulate matter released in wildfires may skyrocket to extreme levels.[169]

States can choose their wildfire policy, which means that states have the power to choose either to spread particulate emissions through time by adopting planned burns or to have periodic bunching of particulate emissions by allowing occasional wildfires. But a state's choice of policy is not unconstrained: a recent analysis by Professor Kirsten Engel and Andrew Reeves shows that federal implementation of the NAAQS creates critical incentives for whether states adopt planned burn strategies or whether they opt to bear the risks of periodic wildfire.[170] Currently, particulate matter released from planned burns is included in the calculations for whether a state is meeting the NAAQS; particulate matter from wildfires is not.[171] As a result, states have an incentive (at least at the margins) to proscribe or otherwise disincentivize planned burns, with the result that wildfire incidence increases.[172] But EPA could just as easily exempt planned burns while including wildfires in the calculation, and states could just as easily incentivize planned burns.[173]

Should policy makers incentivize planned burns or should they encourage states to tolerate wildfires? The answer may depend upon

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167 See Kirsten Engel and Andrew Reeves, *When "Smoke Isn't Smoke": Missteps in Air Quality Regulation of Wildfire Smoke*, in Bradshaw and Lueck, eds, *Wildfire Policy* 127, 127–28 (cited in note 166).

168 Id.

169 See id. By one reckoning, wildfires are responsible for more emissions of fine particulate matter than all sources of fuel combustion combined, including the burning of coal, oil, natural gas, wood, and biomass both in homes and for industrial uses. See Kirsten H. Engel, *Anachronistic Pollution Policy: The Case of Wildfire Smoke Regulation* *16 & n 47 (Arizona Legal Studies Research Paper No 12-26, Aug 2012), online at http://papers.ssrn.com/sol3/papers.cfm?abstract_id=2131366 (visited Sept 23, 2012) (calculating wildfire emissions of fine particulate matter at 998,959 tons a year, in comparison to 804,519 tons from all sources of fuel combustion).

170 Id at 134–36.

171 Engel and Reeves, *When "Smoke Isn't Smoke"* at 134 (cited in note 167).

172 Id at 127.

173 Id at 136–40 (identifying alternative strategies for incentivizing or disincentivizing planned burns using NAAQS calculations).

a number of factors,[174] including how policy makers value future versus immediate harm and the discount rate that policy makers attach to future harm.[175] But one of those factors should be the different exposure allocations that result from adoption of the different strategies.

C. Affirmative Slippage and the Allocation of Enforcement Resources

Exposure allocation can also be strategically manipulated through purposeful allocation of enforcement resources, or what Professor Daniel Farber has called "affirmative slippage."[176] Affirmative slippage provides the opportunity for creative solutions to environmental problems.[177] One possible solution to suboptimal exposure allocation is to use affirmative slippage to effectively loosen standards, either to bunch pollution emission by providing affirmative slippage incentives to polluters in the same area or to spread pollution by spreading affirmative slippage incentives across a wider range, perhaps by instituting a relaxed set of standards for the first polluter in an area and using increasingly stringent enforcement to gradually disincentivize additional emissions as the incremental harm from those emissions increases.

A related approach is to calibrate expenditures on enforcement differently depending upon the incremental benefit of cleanup activity at a particular location. This kind of resource allocation already occurs within EPA in the context of the National Contingency Plan (NCP), which is prepared pursuant to the Comprehensive

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174 For a discussion of other important factors in wildfire policy, see generally Bradshaw and Lueck, eds, *Wildfire Policy* (cited in note 166).

175 See Karen M. Bradshaw, *Norms of Fire Suppression Among Public and Private Landowners*, in Bradshaw and Lueck, eds, *Wildfire Policy* 89, 100–02 (cited in note 166) (addressing the potential intergenerational effects). For a discussion of how the choice of valuation of the future affects final valuations both pre- and post-discounting, and the relationship between temporal valuation method and choice of discount rate, see Rowell, 85 Notre Dame L Rev at 1533–37 (cited in note 65) (identifying different methods for valuing future harms and arguing that different methods result in widely varying valuations).

176 Daniel A. Farber, *Taking Slippage Seriously: Noncompliance and Creative Compliance in Environmental Law*, 23 Harv Envir L Rev 297, 299, 325 (1999) (arguing that environmental law is characterized by pervasive slippage, where decision makers ignore violations of the law, and "affirmative" slippage, where "required standards are renegotiated rather than ignored"). As examples of affirmative slippage, Professor Daniel Farber identifies a number of contexts where EPA habitually "negotiate[s]" against the backdrop of existing standards, including the use of permits allowing the incidental "taking" of endangered species to enable renegotiation of requirements under the Endangered Species Act and EPA's increasing use of "Supplemental Environmental Project[s]" in lieu of statutorily required penalties. Id at 300–11.

177 See id at 320. Farber also recognizes that slippage has a dark side, in that it typically occurs "in the shadow of the law" instead of "in the light of public deliberation." Id at 319.

Environmental Response, Compensation, and Liability Act of 1980[178] (CERCLA).[179] The NCP is required to establish procedures and standards for responding to releases of "hazardous substances," and EPA determines level of "cleanup."[180] This obviously confers significant discretion on EPA in deciding how to allocate its resources. Of course, this discretion could be used poorly.[181] But as with other tools that allow for differential allocation, differential enforcement allocation could also be used to affect exposure allocation and to minimize aggregate harm by targeting those cleanup opportunities where each unit of enforcement resource will have the most bang for the buck; where the pre- and post-cleanup exposure levels result in the greatest change in predicted harm.[182] This approach also has the virtue of being easily tailored, so that it could be used to affect either spread or bunched exposure allocations. The key will be to match allocation of enforcement resources to exposure allocation.

D. Trading and Market-Based Tools

Trading and market-based tools for risk management rely on market mechanisms to allocate polluting uses and therefore pollution.[183] These regimes are increasingly central to environmental regulation in the United States; as one critical commentator puts it, "[t]hese days, the Environmental Protection Agency (EPA) rarely develops any pollution control program without including some form

178 Pub L No 96-510, 94 Stat 2767, codified at 42 USC § 9601 et seq.

179 See CERCLA § 105, 94 Stat at 2779–80, codified at 42 USC § 9605.

180 42 USC § 9605. See also 40 CFR § 300.2.

181 At least one environmental justice scholar has argued that agencies (including EPA) tend to wrongly allocate fewer cleanup resources to disadvantaged areas. See Saleem, 19 Colum J Envir L at 219 (cited in note 141) (referencing a case in Aspen, Colorado, where EPA spent millions fighting a dispute with middle-class residents, only to neglect minority cleanup sites).

182 Professors Kip Viscusi and James Hamilton have suggested similar kinds of arbitrage in the context of allocating resources to Superfund cleanups. See Viscusi and Hamilton, 124 Pub Interest at 53 (cited in note 154) ("In answering the question—how clean is clean?—EPA should follow three principles: assess risks accurately, determine the extent of the population exposed to the risk, and strive for an appropriate balance between benefits and costs."). The key additional point here is that any arbitrage should include an analysis of exposure allocation. Where the exposure allocations of the substance(s) being cleaned up are currently causing supplemental harm—as will frequently be the case where, for example, the dose-response relationship is supralinear—any reductions in pollution or exposure will tend to have a particularly large payoff in terms of harm reduction.

183 For a description of market mechanisms for regulation, see Robert W. Hahn and Gordon L. Hester, *Marketable Permits: Lessons for Theory and Practice*, 16 Ecol L Q 361, 364 (1989); Robert W. Hahn and Robert N. Stavins, *Incentive-Based Environmental Regulation: A New Era from an Old Idea?*, 18 Ecol L Q 1, 7–12 (1991).

of environmental trading within it."[184] While these programs can keep enforcement, compliance, and administrative costs down, they can also create "hot spots," where pollution exposure is bunched in comparison to nearby areas.[185]

The possibility of hot spots has led many scholars to sharply criticize the use of trading regimes.[186] Criticisms have come from two primary directions. The first is based on environmental justice objections: many environmental justice scholars argue that hot spots tend to be located in poorer areas and in areas populated by people of color, and they criticize this allocation as being distributionally unfair.[187]

The second criticism focuses on the fungibility of pollution, relying on the assumption that trading regimes do not work well where the goods being traded are not fungible.[188] In the context of hot spots, the fungibility criticism has focused particularly on the dispersal characteristics of the pollutants: whether they are likely to be localized or dispersed. As one scholar presents this view,

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184 David M. Driesen, *Trading and Its Limits*, 14 Penn St Envir L Rev 169, 169 (2006).

185 For a discussion of how trading programs can lead to hot spots, see Noga Morag-Levine, *The Problem of Pollution Hotspots: Pollution Markets, Coase, and Common Law*, 17 Cornell J L & Pub Pol 161, 163 (2007). See also James E. Krier, *Marketable Pollution Allowances*, 25 U Toledo L Rev 449, 452–54 (1994) (discussing the relationship between hot spots and pollution trading mechanisms); Wiener, 23 Hum & Experimental Toxicology at 291–94 (cited in note 116) (discussing the relationship between hormesis and hot spots). Note that hot spots can also be created (or remedied) by legal mechanisms other than trading programs, including siting decisions, interpretation of pollution standards, and allocation of enforcement resources. All of these mechanisms have the potential to encourage bunching, which means they can create hot spots.

186 See, for example, Morag-Levine, 17 Cornell J L & Pub Pol at 163 (cited in note 185). Proponents of trading regimes also recognize that these regimes can lead to hot spots. See, for example, Nash and Revesz, 28 Ecol L Q at 579–82 (cited in note 128) (arguing that trading programs may lead to concentration of emitters, leading to NAAQS violations despite the overall level of pollution remaining at the permissible level).

187 See, for example, Stephen M. Johnson, *Economics v. Equity: Do Market-Based Environmental Reforms Exacerbate Environmental Injustice?*, 56 Wash & Lee L Rev 111, 116–21 (1999) (summarizing environmental justice literature on hot spots); Lily N. Chinn, *Can the Market Be Fair and Efficient? An Environmental Justice Critique of Emissions Trading*, 26 Ecol L Q 80, 95–96 (1999); Nash and Revesz, 28 Ecol L Q at 580–82 (cited in note 128). Hot spots are often used in the classroom as examples of failures of environmental justice. See, for example, David M. Driesen, Robert W. Adler, and Kirsten H. Engel, *Environmental Law: A Conceptual and Pragmatic Approach* 313–16 (Wolters Kluwer 2d ed 2011) (describing the potential for trading regimes to lead to hot spots and noting the attendant environmental justice concerns).

188 For a highly influential analysis of fungibility in environmental markets, see Salzman and Ruhl, 53 Stan L Rev at 607 (cited in note 151). Professors James Salzman and J.B. Ruhl identify three ways in which a currency being traded on a market may lack fungibility: through nonfungibilities of space, type, and time. See id at 638–42.

> [w]here the pertinent threat is global—such as greenhouse gas emissions—the location at which reductions take place is of marginal importance. Where emissions are not locally fungible, however, the potential for pollution hotspots transforms emissions trading from a win-win situation into something closer to a zero-sum game.[189]

Are hot spots always undesirable and always equally undesirable? It is true that global pollutants—the rare air pollutants, like carbon dioxide, that tend to spread themselves automatically throughout the global atmosphere—cannot create hot spots in a meaningful sense because they enter a global pool regardless of where they are emitted.[190] And it is true that the fungibility of pollutants should be a key consideration in the determination of whether trading regimes are appropriate in any particular regulatory context.[191] But whether emissions are locally fungible depends upon the dose-response curve of the pollutant(s) involved. To see why, consider again Example 1 in Part II where we analyzed substances with linear nonthreshold dose-response curves. Recall that these substances are uniquely indifferent to exposure allocation: they cause the same amount of harm regardless of how they are allocated.[192] This means that these substances are highly fungible in an important sense: they will not tend to cause more or less harm as a function of their exposure allocation. It may be possible to create hot spots when substances of this type are concentrated on one area and that area has relatively higher exposure than other areas. And there may be important fairness concerns about how those hot spots are allocated across the population.[193] But objections to hot spots resulting from linear nonthreshold dose-response curves must be made purely on distributional fairness

189 Morag-Levine, 17 Cornell J L & Pub Pol at 163 (cited in note 185).

190 See Nash and Revesz, 28 Ecol L Q at 614–23 (cited in note 128) (discussing the impacts of regional versus local pollutants on the structuring of trading regimes). See also Wiener, 23 Hum & Experimental Toxicology at 293 (cited in note 116) (distinguishing between the effects of local emissions and the effects of hormesis and explaining that hot spots can only arise where a substance has localized impact).

191 For an elegant analysis along these lines, see Wiener, 23 Hum & Experimental Toxicology at 291–93 (cited in note 116).

192 Setting interpersonal variability aside. For a bit more on the implications of interpersonal variability, see Part V.

193 See Nash and Revesz, 28 Ecol L Q at 579–82 (cited in note 128) (noting that fairness concerns about whether the most vulnerable populations are most likely to accrue additional risk are "independent of the shape of the pollutant's damage function"); Wiener, 23 Hum & Experimental Toxicology at 295–96 (cited in note 116).

grounds because, insofar as the amount of harm they cause is concerned, these pollutants are perfectly fungible.[194]

Inasmuch as this eliminates a serious objection to trading regimes, this means that substances with linear nonthreshold dose-response relationships should be treated as particularly good candidates for management through trading mechanisms.

This is in sharp contrast to substances that exhibit any other dose-response relationship: for these substances, unrestricted trading regimes create not just the possibility of distributionally unfair hot spots but also the possibility of inadvertently causing *more harm with the same amount of pollution*—of causing supplemental harm. When trading regimes are applied to any of these substances, supplemental harm can result both from incidental bunching and from incidental spreading—meaning that trading regimes can lead to supplemental harm both when they create bunched hot spots and when they fail to create bunched hot spots.[195]

Although this point has been neglected in the legal literature, it has been noted in the toxicology literature.[196] One important article by Professor James Hammitt in a leading toxicology journal examined the implications of hormetic and linear nonthreshold dose-response relationship for the use of economic, incentive-based regulations and concluded that "[t]he environmental consequences of allowing firms to reallocate emission reductions depend on the substances involved."[197] As Hammitt explains it:

> Under the linear no-threshold model, the total health effects within a population depend solely on the change in total exposure. The marginal damage associated with a unit of exposure is identical, and so a system of tradable exposure permits or an exposure tax could be anticipated to reduce total control costs while providing the same total health benefit as a command-and-control system. An optimal system would set the exposure tax at a level equal to the marginal benefit of reduced exposure, or would set the total quantity of exposure permits at a level

194 See Hammitt, 23 Hum & Experimental Toxicology at 276 (cited in note 107).

195 See Wiener, 23 Hum & Experimental Toxicology at 291–93 (cited in note 116).

196 For examples of this point being recognized in toxicology literature, see Hammitt, 23 Hum & Experimental Toxicology at 276 (cited in note 107); Wiener, 23 Hum & Experimental Toxicology at 291–93 (cited in note 116).

197 Hammitt, 23 Hum & Experimental Toxicology at 267 (cited in note 107).

> such that the market-clearing price of permits was equal to the marginal benefit of reduced exposure.[198]

This is in contrast to substances with hormetic dose-response relationships, for which marginal damage per unit of exposure varies according to where the exposure falls along the dose-response curve: "The marginal harm associated with a unit of exposure is not constant but depends on whose exposure is altered."[199] This led Hammitt to conclude that "[u]nder the hormetic model, a simple economic-incentive mechanism having a single tax or single type of tradable permit would not be anticipated to work as well as under the linear model."[200]

How generalizable is Hammitt's critique of trading regimes? In a provocative reply to Hammitt's article, Professor Jonathan Wiener suggests that Hammitt's critique of trading regimes is both broader and narrower than Hammitt claims.[201] Wiener limits Hammitt's critique by identifying additional conditions where economic, incentive-based regulations would be undermined by hormetic dose-response curves: "[T]he necessary conditions involve not just hormesis but also local emissions effects, a level of protection set at or near the hormetic minimum-effects level, and a pattern of selling and buying by sources along the dose-response curve in a direction that poses a net increase in harm."[202] And he broadens Hammitt's critique by expanding the analysis to address supralinear and sublinear dose-response curves as well, noting the circumstances under which trading activities would tend to "bunch" or "drain" above or below the average level of emissions.[203] We can easily expand the analysis to apply to threshold pollutants as well; the point is simply that, whenever the relationship between exposure and harm deviates from simple proportionality, allowing exposure allocation to be determined by the market can lead to supplemental harm.

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198 Id at 276.

199 Id:

> Under the hormetic model, the total health effects of a reduction in population exposure depend on the distribution of changes in exposure levels among the population. If exposure reductions are concentrated among highly exposed individuals, total health benefits will be relatively large. If reductions are concentrated among individuals having low exposure, the health effects will be smaller and may be adverse.

200 Id.

201 See Wiener, 23 Hum & Experimental Toxicology at 300 (cited in note 116).

202 Id at 293, 300.

203 Id at 291–93. See also notes 128–29.

This analysis suggests that linear nonthreshold pollutants (and global pollutants of all types) are uniquely well suited to trading regimes because the amount of harm they cause is indifferent to where the exposure occurs. But while this is a bonus for the use of economic incentives in regulating these substances, it does not mean that economic incentives are useless as to other substances. As Hammitt notes, "[a] more complicated system, in which the tax or quantity of permits required per unit of exposure varies across subpopulations in proportion to the marginal benefits of reducing exposure could provide superior outcomes but would be substantially more complicated to develop."[204] Professors Jonathan Nash and Richard Revesz have discussed various methods for structuring trading regimes to effect different levels of exposure, and these methods offer additional mechanisms for fine-tuning exposure allocation.[205]

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In sum, then, law affects the allocation of pollutants through a variety of mechanisms, including siting decisions, allocation of slippage and enforcement resources, and implementation of economic incentives like trading regimes. Since the allocation of pollutants determines the harm those pollutants cause (and sometimes whether there is any harm at all), the choice of legal mechanism for exposure allocation determines how much harm is caused by pollution. That determination should be made mindfully and strategically.

IV. INCORPORATING EXPOSURE ALLOCATION INTO RISK REGULATION

How and where should exposure allocation be incorporated into decision making about environmental harms? Exposure allocation offers an alternative strategy for reducing the harm from pollution—a strategy that can be used either instead of or in addition to the familiar strategies of pollution reduction and exposure reduction. As such, it should be considered as a potential tool to further any policy seeking to reduce the harm from pollution.

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204 Hammitt, 23 Hum & Experimental Toxicology at 276 (cited in note 107).

205 Nash and Revesz, 28 Ecol L Q at 617 (cited in note 128) (arguing that there are two conditions for a viable market-based regulatory system when a pollutant's effects are nonlinear: "[T]he regulated pollutant must have the characteristic that only relatively large shifts in its emission locations affect the spatial distribution of the harm, or there must be a sufficiently large concentration of potential market participants").

Ignoring exposure allocation doesn't make it go away. As we have seen, existing environmental legal policies already affect exposure allocations in identifiable ways.[206] But currently they do so without regard to the total amount of harm created by whichever exposure allocation they effect. Incidental exposure allocation may periodically luck into good allocations. But it seems perverse to trust harm prevention to the vagaries of chance.[207]

Explicit consideration of exposure allocation would improve the transparency of the regulatory process and would encourage deliberation.[208] This may be helpful both because expressive participation in government can be thought of as a democratic good in itself and because increasing transparency and deliberation may increase decision quality.[209]

That said, there might be significant barriers to addressing exposure allocation in the public sphere. Not least of which is that exposure allocation involves highly technical analyses that require at least some understanding of basic toxicology—an understanding that conflicts with the intuitive "pollution heuristic" most people utilize when making decisions about dangerous substances.[210]

Since exposure allocations will often involve the allocation of harm, public choice concerns are also likely to arise.[211] Consider the exposure allocation of sublinear pollutants. Bunching may reduce aggregate harm, but who—or which group of people—ends up bearing the harm that does result? In many circumstances, it may be

206 For discussion and examples, see Part III.

207 But see Adam M. Samaha, *Randomization in Adjudication*, 51 Wm & Mary L Rev 1, 4–27 (2009) (summarizing objections to randomization as a decision-making tool, and providing a partial defense of the virtues of randomization as a decision-making tool, particularly where the decision maker must choose between equally strong claims). Where policy makers believe that potentially affected communities have equally strong claims to protection, a limited form of randomization might be one strategy for addressing the difficult question of who bears the harm.

208 See John Rawls, *A Theory of Justice* 16, 454 (Belknap 1971) (requiring transparency in a just society because it informs individuals about their choices in being governed); Mark Fenster, *The Opacity of Transparency*, 91 Iowa L Rev 885, 895–99 (2006) (arguing that transparency plays a critical role in a democratic society). For a discussion of the political effects of a lack of transparency, see Gia B. Lee, *Persuasion, Transparency, and Government Speech*, 56 Hastings L J 983, 1008–15 (2005).

209 See John Rawls, *The Idea of Public Reason Revisited*, 64 U Chi L Rev 765, 785–86 (1997); Cass R. Sunstein, *Democracy and the Problem of Free Speech* 244 (Free Press 1993).

210 For a discussion of intuitive toxicology and the pollution heuristic, see Part I.A.

211 See generally Farber and Frickey, *Law and Public Choice* (cited in note 109).

those groups who are already disempowered by the existing process, raising serious potential concerns of distributional fairness.[212]

These are difficult political and moral concerns related to the choice of exposure allocations and are worth significant attention. As a starting point, however, we should note that these difficult questions should be informed by technical analyses—analyses of how exposure allocation is likely to be affected by various legal policies and of the amount of harm that is likely to result from various exposure allocations. Without an understanding of how exposure allocation affects harm, we will not be able to meaningfully debate the virtues of competing allocations.

With that in mind, this Part identifies practical and technical opportunities for incorporating exposure allocation analysis into existing structures of risk regulation. It makes two concrete prescriptions: first, that risk assessment and risk management be unified insofar as they relate to exposure assessment; and second, that cost-benefit analyses address how exposure allocation affects the expected costs and benefits of a regulation.

A. Integrating Exposure Allocation into Risk Assessment and Risk Management

The most obvious analytical home for exposure allocation is within quantitative risk analysis, which includes both risk assessment and risk management processes. Risk analysts already perform the bulk of the analysis necessary for exposure assessments. They perform hazard characterizations, dose-response assessments, and exposure assessments as part of the quantitative risk assessment process, and they choose between regulatory instruments as part of risk management.[213] What is missing from current practice is the connection between the regulatory instruments chosen and the effects on aggregate harm caused, as militated by dose-response and exposure data. At the least this information should be incorporated into the final step of risk assessment—risk characterization—when assessors create a synthesized analysis of the risk being addressed.

Why have risk analysts not already begun to incorporate exposure assessment into their analyses? Part of the reason may be the traditional bifurcation between risk assessment and risk management, which would have made it difficult to identify—much less

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212 For the argument that power politics of this sort explain existing exposure allocations, see Bullard, *Environmental Justice in the Twenty-First Century* at 30–31 (cited in note 135).

213 See Part I.B.

elaborate on—the relationship between dose-response characteristics and instrument choice.

This bifurcation has been criticized on a number of grounds including by the NRC itself in the 2008 *Silver Book*.[214] As a result, agency risk-analysis practices are slowly shifting. As agencies increasingly try to integrate risk assessment and risk management—and particularly as they implement the *Silver Book*'s recommendation that risk managers identify the particular policies they are considering so that risk assessors can do targeted assessments of their impact[215]—agencies should also take the opportunity to incorporate exposure allocation into their risk analyses.

B. Integrating Exposure Allocation into Cost-Benefit Analysis

CBA has long been subject to the criticism that it is dangerous when used as the sole determinant of policy as it is insensitive to important distributional concerns, such as whether harms and benefits are distributed across a population in a fair or just way.[216]

This Article suggests that CBA is subject to a related, but distinct, objection, which is that current cost-benefit techniques do not account for exposure *allocation*: that separate from debates about distributional fairness, current CBAs fail to account for a key determinant of the amount of aggregate harm caused under the policy being analyzed. Because CBAs seek to measure the magnitude of costs and benefits and because exposure allocation is a determinant of the magnitude of harm caused by pollution, this critique cannot be deflected by suggesting that exposure allocation be handled as a separate analysis.[217]

To see why, consider how a CBA could account for exposure allocation. It would calculate costs and benefits of pollution control

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214 See NRC, *Silver Book* at 241–45 (cited in note 48).

215 See id at 240.

216 See Sunstein and Rowell, 74 U Chi L Rev at 198–208 (cited in note 165) (noting that CBA can create distributional inequity and arguing that that inequity should be handled through distributional analyses); Richard L. Revesz and Michael A. Livermore, *Retaking Rationality: How Cost-Benefit Analysis Can Better Protect the Environment and Our Health* 9–19 (Oxford 2008) (advocating for CBA as a general method for risk management but arguing that distributional analysis is needed as a supplement); Adler, 32 Harv Envir L Rev at 2 (cited in note 66) (noting that standard CBA "is insensitive to distributional considerations").

217 In fact, the only time that CBA—which is attempting to quantify the effects of a regulation—might reasonably ignore exposure allocation is where the policy affects only linear nonthreshold pollutants; that is, those pollutants that are indifferent to exposure allocation. When this is the case, the CBA should explicitly state that exposure allocation will be unaffected because the pollutant being affected is assumed to exhibit a linear nonthreshold dose-response relationship.

policies based on the exposure allocation those policies created. For any pollutants with curvilinear, hormetic, or threshold dose-response curves, assumed exposure allocation would be a key driver of the final determination of aggregate cost and benefit, as we saw in Part II.

These benefits and harms are contingent on the final allocation of the relevant pollutant. And as we have just seen, the final exposure allocation will be contingent upon the risk-management strategy used to implement the policy. In other words, CBA of pollution policy must know—or at least guess—the exposure allocation in order to do a systematic analysis of the expected costs and benefits of a regulation. This requires some iteration between risk-management options and CBA calculations.

Currently, agencies do not incorporate this sort of analysis into their CBAs.[218] Although agencies do distributional analyses (and indeed, are required to do so via executive order),[219] these analyses are disconnected from the CBAs.[220] Since analysts cannot avoid making assumptions about the level of harm that is done and since they are, in fact, routinely performing CBAs, they are making assumptions about the quantity of exposure—about, in fact, the exposure allocation. These assumptions should be made explicit.

The point here is not merely that CBA is unacceptable without an allocational analysis but that CBA necessarily imbeds a form of allocational analysis. In failing to identify the exposure allocation they are assuming, regulators are masking the impact of exposure allocation on aggregate harm levels and are also opaquely implementing policies affecting exposure allocation that ought to be debated. Future CBAs should be explicit about the exposure allocations they assume.

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218 Agencies do follow detailed guidelines in performing CBA. See, for example, Office of Management and Budget (OMB), Circular A-4, *Regulatory Analysis* (Sept 17, 2003) (available on Westlaw at 2003 WL 24011971) (presenting OMB's guidelines); EPA, *Guidelines for Preparing Economic Analyses* (Dec 17, 2010), online at http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0568-50.pdf/$file/EE-0568-50.pdf (visited Sept 23, 2012) (providing EPA's recently updated guidelines). Neither of these influential documents directs analysts to address exposure allocation.

219 At least insofar as the regulation is likely to affect minorities or the poor. See 3 CFR 859 (1995).

220 See notes 63–72 and accompanying text. For an example of other commentators proposing distributional analysis as a remedy for unfairness, see Revesz and Livermore, *Retaking Rationality* at 182 (cited in note 216):

> Distributional analysis is not an easy undertaking, but it is a necessary corollary to cost-benefit analyses. Cost-benefit analysis, on its own terms, excludes concern for the distribution of the benefits and burdens of regulations. This omission is acceptable only if a separate effort is undertaken to account for these effects.

V. LIMITATIONS, OBJECTIONS, AND GENERALIZABILITY

This Article has argued that misallocating pollution can lead to great harm—converting potential health benefits into supplemental harm and even into lives lost. It has also argued that current attempts to analyze harm caused by pollution are fundamentally incomplete because they do not incorporate the critical observations that law affects how pollutants are allocated across the population and that the way pollutants are allocated determines the quantity of harm they cause. To remedy this problem, this Article recommends that future analyses of pollution explicitly address exposure allocation, both within the internal agency-decision procedures in the form of unified risk analysis and adjusted CBAs, and within the forum of public debate in the form of explicit policies adopted to address exposure allocation.

But while exposure allocation can substantially improve the quality of decision making about environmental issues, it is subject to the same constraints that operate in other environmental contexts.

One important constraint is that exposure allocation can inform us about harm reduction, but harm reduction is not the only goal of environmental laws.[221] Exposure allocation is helpful only insofar as it informs aggregate harm. For other policy goals, we will need other policy tools. I have gestured at two of these tools: namely, pollution reduction and exposure reduction. This Article has been concerned with establishing exposure allocation as a potential and neglected approach to using law to reduce the harm from pollution. But a key question for policy makers will be when to use exposure allocation instead of, or in addition to, these other strategies. The answer to this question will depend upon the underlying goals of the policy makers—for example, how they weigh distributional fairness concerns against aggregate suffering.[222]

Another pervasive issue in environmental law is that it relies on chronically incomplete information.[223] The solution to this is to regulate based on the best interpretation of the best possible infor-

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221 See, for example, notes 136–54 and accompanying text (discussing distributional concerns). See generally Shi-Ling Hsu, *Fairness Versus Efficiency in Environmental Law*, 31 Ecol L Q 303 (2004).

222 For an attempt to operationalize this tradeoff, see Adler, *Well-Being and Fair Distribution* at 307–405 (cited in note 69) (advocating for a prioritarian social welfare function).

223 See Holly Doremus, *Constitutive Law and Environmental Policy*, 22 Stan Envir L J 295, 319 (2003) (noting that "[t]he most universally recognized feature of environmental problems is the pervasive uncertainty that surrounds them"). See also Daniel A. Farber, *Uncertainty*, 99 Georgetown L J 901, 907–13 (2010) (distinguishing between analytically distinct types of uncertainty).

mation.[224] But this solution does not mean that the uncertainty goes away.

To the extent that exposure allocation depends upon evolving information about dose-response relationships, it will always be based on some amount of incomplete information.[225] But this is not a reason to refuse to consider the different impacts of different allocations, because allocations—and harm levels—accrue whether we try to understand their effects or not. Any information we have—even if it turns out to be incomplete or wrong—gets us closer to reducing harm. Regulatory mechanisms that allow for periodic revisitation of policy decisions—such as the recent Executive Order that requires agencies to engage in retrospective analysis[226]—might help keep inevitable mistakes from becoming entrenched. In uncertain situations, we might also prefer to set regulatory defaults that have relatively cheap opt-outs, again with the thought that future information may push towards alternative solutions, or to develop adaptive regulatory mechanisms.[227] We might also set defaults based on distinct ethical principles—such as a commitment to fairness—that tend to be resistant to the vagaries of uncertain outcomes.[228]

A more extreme response to uncertainty in calculations of human health harms would be to turn away from technocratic and scientific measurements of harm like those on which toxicological studies depend. Instead, we could imagine a regulatory system that used people's perceptions of the risk of pollutants as the "harm" to be reduced.[229] This approach would limit the kinds of epidemiological

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224 This is not meant to be flippant; clearly it is an enormous task to determine what the "best" interpretation of the "best" information will be. For a thoughtful analysis of the difficulties agencies face in trying to implement requirements, see Robert L. Glicksman, *Bridging Data Gaps through Modeling and Evaluation of Surrogates: Use of the Best Available Science to Protect Biological Diversity under the National Forest Management Act*, 83 Ind L J 465, 465–69, 479–82 (2008).

225 Significant ongoing sources of uncertainty in dose-response relationships include extrapolation from animal studies to human health effects; the effects of chemical "cocktails," or mixtures, on dose-response relationships; variability in the responses of individuals and subpopulations; and responses at low doses, which are often hard to detect and difficult to isolate. See generally Rowell, *Risk Assessment* (cited in note 38).

226 President Obama's recent Executive Order 13563 requires agencies to perform retrospective analyses of old regulations. 76 Fed Reg at 3822 (cited in note 64).

227 For a presentation of one such adaptive mechanism, see Bradley C. Karkkainen, *Adaptive Ecosystem Management and Regulatory Penalty Defaults: Toward a Bounded Pragmatism*, 87 Minn L Rev 943, 997–98 (2003).

228 For an outline of various approaches to fairness, see Adler, *Well-Being and Fair Distribution* at 314–39 (cited in note 69).

229 This approach would tie in to the literature on social versus technocratic perceptions of risk. See note 33.

and other scientific uncertainty imbedded in the analyses, although it would do so at a price, since people's intuitive understandings of toxicology do not track scientific measurements of human health harms caused by exposure.[230] In some ways, this approach would mimic applications of the precautionary principle—a regulatory principle often seen as a competitor to CBA.[231]

Adopting this approach to risk regulation—an approach based on social rather than technocratic constructions of the harm of pollution—would be a radical change from current policies.[232] But exposure allocation would remain a useful tool, so long as harm and exposure remained occasionally divergent.[233]

A broader objection to the approach this Article takes to exposure allocation is that it focuses on a human-centered, health-based approach to harm reduction.[234] This may be an importantly incomplete approach to measuring harm.[235]

Consider health. Human health is explicitly the goal of many environmental statutes.[236] But it is surely not the only value we can imagine, even if we stick—for the moment—with humans as the only value source. Many people might be willing to give up their health to achieve their heart's desire, for example, or for the sake of their loved ones. Often people trade money for health, and these tradeoffs are presumably meaningful prioritizations that represent people's

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230 See note 27 and accompanying text (discussing intuitive toxicology).

231 See generally Cass R. Sunstein, *Laws of Fear: Beyond the Precautionary Principle* (Cambridge 2005) (arguing that the precautionary principle is typically invoked against affective risks). Note that, insofar as fear of pollution is a real harm, this approach could have real benefits. See generally Matthew D. Adler, *Fear Assessment: Cost-Benefit Analysis and the Pricing of Fear and Anxiety*, 79 Chi Kent L Rev 977 (2004) (arguing that fear is a meaningful setback in welfare).

232 Consider notes 39–65 and accompanying text (discussing the current approach to risk assessment).

233 One interesting implication of this approach is that people's marginal sensitivity to the thought of pollution exposure decreases with additional levels of exposure—that is, it tends to exhibit a sublinear dose-response relationship. As this was the only type of substance for which bunching was optimal, a regulatory regime built on explicit social constructions of harm should tend to prefer bunching strategies to spreading ones, all else equal.

234 See, for example, Elizabeth Anderson, *Value in Ethics and Economics* 204–10 (Harvard 1993) (arguing for broader conceptions of valuation of endangered species and other environmental goods).

235 See id. See also Martha C. Nussbaum, *Women and Human Development: The Capabilities Approach* 34–110 (Cambridge 2000) (identifying diverse, objective goods that represent different dimension of human welfare).

236 See, for example, Clean Air Act, 42 USC § 7401(b) ("The purposes of this subchapter are—(1) to protect and enhance the quality of the Nation's air resources so as to promote the public health and welfare and the productive capacity of its population.").

preferences for nonhealth goods.[237] To the extent that we focus solely on health, therefore, we will be missing many of the things that make a life worth living. This is not to say, however, that life and health are not important goods to be protected; it is merely to say that policy makers' work is not done when the right health goals have been achieved. So long as human health is one important policy goal, a risk analysis focusing on human health is helpful even if not complete.

How can the approach presented in this Article be used in nonhealth or nonhuman contexts? All we need is something quantifiable (like a substance or an organism) and the possibility that exposure to small amounts of that item are beneficial, whereas large amounts are damaging.

Along this line, EPA has started to do what it calls "ecological risk assessments," where it attempts to quantify the impact of environmental stressors on whole ecologies.[238] For example, in *An Ecological Assessment of Western Streams and Rivers*, EPA evaluated a number of environmental stressors affecting western water quality, including four indicators of chemical stress: excessive concentration of salt, phosphorus, nitrogen, and mercury in fish tissue.[239] Salt serves a useful purpose in many ecologies but in excessive concentrations can pose risks to many fish.[240] Insofar as substances like salt exhibit dose-response relationships with the relevant endpoints, an exposure allocation analysis can be helpful in informing policy makers as to whether spreading or bunching is likely to cause less damage to the relevant ecology.[241]

Finally, consider that the methodology underlying exposure allocation could be broadened further still to apply to legal and politi-

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237 For a defense of the use of monetized preferences in policy, see Rowell, 85 Notre Dame L Rev at 1510–17 (cited in note 65).

238 See EPA, Guidelines for Ecological Risk Assessment, 63 Fed Reg 26846, 26846 (1998). Assessors typically identify an assessment endpoint, which might be defined by reference to particular protected individuals, populations, communities, ecosystems, or even landscapes and then model the relationship between that endpoint and the pollutant or environment stressor they are analyzing. See 63 Fed Reg at 26895 (cited in note 238) (identifying "salmon reproduction and population recruitment" in a particular river as an example of a "good assessment endpoint"). See also EPA, *Generic Ecological Assessment Endpoints (GEAEs) for Ecological Risk Assessment* 1–22 (Oct 2003), online at http://www.epa.gov/raf/publications/pdfs/GENERIC_ENDPOINTS_2004.PDF (visited Sept 23, 2012).

239 EPA, *An Ecological Assessment of Western Streams and Rivers* 8–9 (Sept 2005), online at http://www.epa.gov/emap/west/html/docs/Assessmentfinal.pdf (visited Sept 23, 2012).

240 See id at vi.

241 For an analysis of the implication of hormesis for ecological risk assessment—risk assessment that focuses on impacts on ecologies rather than on human health—see Peter M. Chapman, *Ecological Risk Assessment (ERA) and Hormesis*, 288 Sci Total Envir 131, 135–39 (2002).

cal contexts outside environmental law—that is, to apply to broader conceptions of pollution.[242] Ignoring exposure allocation is a problem wherever legal policy has the goal of reducing harm and uses exposure to some "pollutant" as a proxy for that harm. Examples might include: regulating violence on TV or in video games as a proxy for reducing psychological harm to children or actual violence;[243] restricting "hate speech" as a proxy for violent action incited by that speech;[244] regulating pornography as a proxy for sexual violence;[245] and restricting discriminatory housing ads as a proxy for the target groups' inability to find housing.[246]

Where harm from these proxies comes from concentration—as may be the case, for example, where discriminatory housing practices freeze out some minorities from finding appropriate housing—policy makers have the option to reduce harm even where eliminating discriminatory practices is impossible, by seeking out spreading strategies that lead to a broader dispersal of discriminatory advertisements.

Harm—and its relationship to the relevant proxy—may be even harder to quantify in these contexts than in the context of pollution exposure allocation. But to the extent there is any relationship between a proxy for harm and harm itself—and the goal is to reduce *harm*—the same strategies of bunching and spreading can be used to arbitrage across people and contexts in order to reduce total harm without necessarily reducing the incidence of the underlying behavior.

CONCLUSION

How much harm pollution causes is determined in large part by its exposure allocation: whether the pollution is spread or bunched across the target population. Law provides multiple mechanisms for affecting exposure allocation, which means that it has multiple

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242 Such as advocated by Professor John Nagle. See Nagle, 43 UC Davis L Rev at 60–72 (cited in note 13).

243 See, for example, Eric T. Gerson, Note, *More Gore: Video Game Violence and the Technology of the Future*, 76 Brooklyn L Rev 1121, 1125–37 (2011) (discussing the historical regulation of video game violence as a proxy for real-world violence).

244 See, for example, Jeremy Waldron, *The Harm in Hate Speech* 1–143 (Harvard 2012) (describing doctrinal definitions of hate speech as involving the likelihood of inciting hatred or hostility and arguing that the harm of hate speech is better understood as a threat to social inclusion).

245 See, for example, Catharine A. MacKinnon, *Not a Moral Issue*, 2 Yale L & Pol Rev 321, 323–24 (1984) (identifying the link between sexual violence and pornography as a reason to regulate pornography).

246 For a long list of additional potential cultural, social, and environmental "pollutants," see Nagle, 43 UC Davis L Rev at 60–72 (cited in note 13). Exposure allocation provides an alternative method for dealing with all of these pollutants. Special thanks to Richard Ross for suggesting this application.

means of affecting how much harm is caused by pollution, even when the amount of pollution is held constant. These tools can be used for ill, to distribute harms in inequitable ways. But they can also be used to minimize the harm caused by pollution and, in some cases, to eliminate it entirely.