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Conceptual analysis and special-interest science: toxicology and the case of Edward Calabrese

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Abstract One way to do socially relevant investigations of science is through conceptual analysis of scientific terms used in special-interest science (SIS). SIS is science having welfare-related consequences and funded by special interests, e.g., tobacco companies, in order to establish predetermined conclusions. For instance, because the chemical industry seeks deregulation of toxic emissions and avoiding costly cleanups, it funds SIS that supports the concept of "hormesis" (according to which low doses of toxins/carcinogens have beneficial effects). Analyzing the hormesis concept of its main defender, chemical-industry-funded Edward Calabrese, the paper shows Calabrese and others fail to distinguish three different hormesis concepts, H, HG, and HD. H requires toxin-induced, short-term beneficial effects for only one biological endpoint, while HG requires toxin-induced, net-beneficial effects for all endpoints/responses/subjects/ages/conditions. HD requires using the risk-assessment/regulatory default rule that all low-dose toxic exposures are net-beneficial, thus allowable. Clarifying these concepts, the paper argues for five main claims. (1) Claims positing H are trivially true but irrelevant to regulations. (2) Claims positing HG are relevant to regulation but scientifically false. (3) Claims positing HD are relevant to regulation but ethically/scientifically questionable. (4) Although no hormesis concept (H, HG, or HD) has both scientific validity and regulatory relevance, Calabrese and others obscure this fact through repeated equivocation, begging the question, and data-trimming. Consequently (5) their errors provide some undeserved rhetorical plausibility for deregulating low-dose toxins.

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For more than 20 years, Walter Allen was a maintenance worker at Baton Rouge General Hospital. His duties included replacing cylinders containing ethylene oxide (ETO), a compound used to sterilize medical/surgical devices. After Allen died of brain cancer, in 1996 his widow and son sued the sterilizer manufacturer for wrongful death and claimed Allen's exposure to ETO contributed to his brain cancer. Their lawsuit should have been an "easy win." After all, the International Agency for Research on Cancer had showed ETO is a potent carcinogen and genotoxin. Acting directly on the genes, it causes chromosomal and genetic damage in both humans and other mammals. Because of its small size, ETO directly penetrates cell DNA and crosses the blood–brain barrier (IARC 1994, p. 73; APEC 1996).

The court granted a judgment against the Allens and denied them a jury trial. Why? Making false evidentiary inferences, the pretrial judge claimed workplace ETO did not contribute to Walter Allen's brain cancer. The judge admitted ETO can cause human stomach cancer and leukemia but said "no [human] epidemiological study had established" a link between ETO and brain cancer (Cranor 2006, p. 20; see pp. 18–20, 324–325).

1 Faulty evidentiary inferences and harm to pollution victims

The Allen family lost its lawsuit partly because the pretrial judge erroneously relied on several faulty scientific inferences, including SI. SI is the claim that, although animal tests (and human and animal cell data) show ETO's carcinogenicity and mutagenicity, human brain-cancer-specific epidemiological data also are necessary to show ETO exposure might cause brain cancer. Using the flawed SI, and lacking brain-cancer-specific human epidemiological data, the court decided against the Allens.

SI, however, relies on several faulty assumptions. One assumption is that human studies are usually superior to animal studies. Yet at least four reasons suggest that human epidemiological studies often are inferior to animal studies. (1) One reason is that because many human experiments are ethically prohibited, and researchers ought not subject humans to known experimental harms, it is more difficult to gather human-exposure than animal-exposure data. As a consequence, human epidemiological studies often are purely observational, not experimental. That is, they have no random assignment of subjects to different exposure conditions, no random assignment to control or study groups, and so on. As a consequence, when these purely observational human studies employ any classical statistical-significance tests, their conclusions have no strict interpretability and reliability because of the lack of randomness (Greenland 1990; Rothman 1990; Wing 2003; Shrader-Frechette 2008e). However, because non-human animals may be subjected to known experimental harms, and because these studies may meet conditions of randomization, control of confounders, and so on, the experimental animal studies are more statistically robust than, and typically rely on better data and conclusions than, the observational human studies.

Animal epidemiological studies also are frequently superior to human studies because (2) there often are more errors in human- (as opposed to animal-) exposure data because human studies typically rely on observational, rather than experimental studies. For instance, many human data are from workplace exposures which often

are estimated/extrapolated after the fact, not directly measured. Still another problem is that (3) human-study selection biases, such as the healthy-worker survivor effect, typically do not plague animal studies. Moreover, (4) although animal studies provide less precise extrapolations to humans, they often reveal much stronger, more reliable disease associations, partly for the reasons already noted. This means that, provided researchers do not confuse the precision of exposure-disease relations with their strength, animal studies often are preferable.

A second SI flaw is its ignoring the dominant scientific practice of using animal evidence as sufficient to justify causal claims about human carcinogens. This is accepted practice partly because human epidemiological studies are very long, difficult, and expensive, even if they can legally and ethically be done (Shrader-Frechette 2008b, pp. 1–6).

A third SI problem is its erroneous presupposition that, to show something causes brain cancer, brain-cancer-specific epidemiological studies are required. Yet in the ETO case, two facts precluded need for brain-cancer-specific ETO epidemiological studies. (1) Because scientists agreed that ETO is a "multisite mutagen," potent carcinogen, and potent genotoxin (IARC 1994; APEC 1996; Cranor 2006, pp. 325–326), they recognize it likely causes brain and other cancers. (2) Because of its small size, ETO can "cross the blood–brain barrier" and reach the brain/most other human-body targets (IARC 1994; APEC 1996; Cranor 2006, pp. 325–326). If (1) and (2) eliminate evidentiary need for brain-cancer-specific, human-epidemiological studies to show ETO is a probable brain carcinogen, why did the Allen-case pretrial judge miss these scientific facts? One reason is that a prominent industry-consultant, toxicologist Edward Calabrese, wrote a report for the court that ignored these facts. Instead, Calabrese erroneously claimed that ETO's causing stomach and hematopoietic-system cancers was "irrelevant" because the Allen issue was whether ETO caused brain tumors (Calabrese 1993; Cranor 2008). Yet these stomach/blood cancers are relevant to brain cancers because ETO is a multisite mutagen and able to cross the blood–brain barrier. The flawed Calabrese claims thus helped mislead the court and cause the Allen family to lose its case (Cranor 2008).

Calabrese's misleading claims also raise important questions of research ethics. A Ph.D. toxicologist (like Calabrese) arguably knows (or should know) that ETO is a multisite mutagen and able to cross the blood–brain barrier. Likewise, given ETO's confirmed high carcinogenic/mutatoxic potency, its having caused blood/stomach cancers, and publications as early as 1969 strongly suggesting ETO's human-mutagenic potential (Ehrenberg et al. 1974; Sulovska et al. 1969), Calabrese knows (or should know) that one needs no brain-cancer-specific, human-epidemiological data to show ETO is a probable brain carcinogen. In US National Science Foundation-funded research at the University of California, outside reviewers confirmed that Calabrese's Allen-case, research-ethics behavior was questionable. They said Calabrese

> relies on inadequate epidemiological data as well as misstatements about the value of data on brain–tumor induction… [His] opinion is contrary to that of all national and international agencies… Such speculation and subjective opinions are inconsistent with evaluative procedures used by expert review panels of IARC, NTP, USEPA, CALEPA, etc.… The environmental and biomedical com-

> munities have long rejected this approach [requiring human-epidemiological studies of brain-cancer risk, despite the two biological facts just given] for public-health decisions. In fact, the IARC evaluation of ETO, that was published in 1994, used the same available animal, human, and mechanistic evidence…to reach the conclusion that ETO is carcinogenic to humans… The misleading statements and unsupported assumptions used by the [sterilizer manufacturer's] defendant's expert [Calabrese]…are clearly outside the range of respectable scientific disagreement by experts in cancer risk assessment (Cranor 2008).

2 Philosophers of science and special-interest science

Using their traditional methodological tools, should scientists and philosophers of science help prevent flawed science (and apparent research-ethics misconduct), like that in the Allen case? The scientific research society, Sigma Xi, believes so.

> Because the pathways that we pursue as research scientists are infinite and unfrequented, we cannot police them as we protect our streets and personal property. We depend on those other travelers—other research scientists whose work happens to take them along such lonely byways of knowledge (Jackson 1986, p. 33).

If philosophers of science might help prevent scientific errors and misconduct, how might they do so? One way is by policing special-interest science (SIS). SIS is science funded by special interests, so as to establish predetermined conclusions, those consistent with the funders' profit motives; for instance, cigarette manufacturers delayed tobacco regulation for nearly half a century by funding research claiming tobacco was harmless (Shrader-Frechette 2007, chap. 2). SIS proponents are like the Queen in Lewis Carroll's *Through the Looking Glass.* Just as the Queen claimed she could believe 6 impossible things before breakfast, SIS proponents often use scientific concepts/methods in ways that are "impossible"—that is, inconsistent, question-begging, arbitrary, or contra-indicated for the case at hand. Like Alice, philosophers of science could help investigate these questionable manipulations of science. Moreover, such investigations would help science progress and help avoid SIS. Otherwise, SIS scientists (like the Queen) can continue promoting "impossible things," causing scientists, policymakers, and judges (as in the Allen case) to rely on erroneous or misleading science. Such reliance harms science as well as justice.

3 Conceptual analysis as a tool for promoting better, and socially relevant, science

One way philosophers of science might use their disciplinary tools to police SIS is by analyzing controversial scientific concepts. Besides helping to reveal SIS manipulations and misrepresentations, this conceptual analysis is important to help science progress. As Ernst Mayr (1988) emphasized in his *Toward a New Philosophy of Biology,* recent progress in evolutionary biology has come mainly from conceptual clarification, not from improved measurements or better scientific laws. This article

presupposes that conceptual clarification is an important key to progress in toxicology, to policing SIS, and to avoiding scientific errors, like those of Calabrese in the Allen case. After all, had Calabrese properly clarified concepts like "multi-site carcinogenicity" and "passing the blood–brain barrier," he arguably would not have claimed ETO-induced stomach and blood cancers were "irrelevant" to ETO-induced brain cancers.

To illustrate how clarification of controversial scientific concepts might promote scientific progress, socially relevant science, and recognition of flawed SIS, consider the hormesis concept. Hormesis refers to the notion that low doses of toxins/carcinogens have some beneficial effects—just as low doses of some vitamins have some beneficial effects, despite some vitamins' high-dose harms. Not surprisingly, industrial/agricultural polluters are spending millions of dollars to fund scientific research designed to show that low-dose toxins/carcinogens have beneficial effects (e.g. Calabrese 2007). Their SIS goal is to use hormesis claims to avoid expensive pollution clean-ups and to promote weaker regulations for toxin/carcinogen exposures. This paper argues that their SIS goal, however, is being accomplished by question-begging equivocation, conceptual obfuscation, and failure to distinguish three quite different hormesis concepts, none of which is both scientifically plausible and relevant to regulation.

As espoused by Calabrese (who fails to distinguish among different hormesis concepts), one hormesis concept (that I call H) is that, for at least one biological endpoint/response/subject/age/condition, some low-dose toxin/carcinogen exhibits a "beneficial" effect (Calabrese and Baldwin 2003a, p. 191), or an "adaptive response characterized by biphasic dose responses" that result from "compensatory biological processes following an initial disruption in homeostasis" (Calabrese and Baldwin 2002, p. 91). For instance, low-dose-cadmium exposure is one of the six main examples that (according to Calabrese) allegedly satisfies H (Calabrese and Baldwin 2003b, p. 691), because it reduces some tumors in some species (a beneficial effect at one biological endpoint for some individuals). However, there is scientific consensus that, despite this single-endpoint beneficial effect, low-dose cadmium causes excess prediabetes and diabetes, pancreas damage, glucose dysregulation, and kidney damage—harmful effects at other biological endpoints (Axelrod et al. 2004, pp. 336–338). As this example illustrates, claims of H may appear both true and uncontroversial, mainly because they require so little: at least one non-monotonic effect, on one endpoint, from one pollutant, for one short period of time—regardless of their devastating effects on other endpoints during longer periods of time. H claims thus would be satisfied if a pollutant caused cancer (one biological endpoint), but increased hair growth (another biological endpoint). The upshot? Given Calabrese's minimalist definition of hormesis (H), if low-dose responses to toxins/carcinogens are beneficial for some endpoints but harmful for others, the response nevertheless satisfies his definition of H.

Moreover, Calabrese and others call responses "hormetic" (Calabrese and Baldwin 2001, pp. 286, 290, 285)—H—even when they fail to satisfy criteria for statistically significant changes from control. Thus, Calabrese calls a not-statistically significant "beneficial" change in incidence from 2 to 3, in a sample of only 20, a 33-percent change, evidence of hormesis—H (Thayer et al. 2005, pp. 1272–1275). Likewise, Calabrese and Baldwin use a study of a pollutant's no-observed-adverse-effect level (NOAEL) to "confirm" H. Yet because sample size, statistical power, data variability,

endpoint measured, exposure duration, exposure route, exposure rate, and so on, all affect a pollutant's NOAEL, alleged H responses appear to be merely artifacts of the preceding factors (Thayer et al. 2005, pp. 1272–1275; SAB 2000). Given Calabrese's flawed scientific criteria for H, alleged instances of H are thus easy to find. However, H reveals nothing about an organism's all-endpoint, lifetime, synergistic, cumulative, or net responses to multiple low-dose toxins. Yet such responses are crucial to reliably assessing the medical/scientific/policy relevance of low-dose responses to toxins—like TCDD (dioxin), one of the six main examples that (according to Calabrese) allegedly satisfies H (Calabrese and Baldwin 2003b, p. 691).

Consider four methodological flaws in a two-year, low-dose TCDD study of hormesis. Its flawed allegations of decreased tumor-incidence in rats are typical of studies that allege hormesis, H (Kociba et al. 1978, pp. 279–303). First, the study trimmed the data on adverse effects by including only two-thirds of the rats' lifespan, not infancy and old age, when subjects exhibit more tumor-sensitivity to toxins. After all, if 80% of human cancers are diagnosed in the last one-third of life (Mead 2006, p. 114), and if the rat analogy holds for human lifespans/cancers, the study may have captured only 20% of TCDD-induced cancers. A second flaw is that although the study documented increased liver, lung, tongue, and nasal tumors—but decreased pituitary, uterine, mammary, pancreas, and adrenal tumors—it invalidly aggregated all tumors. Because no individual tumor response was non-monotonic, the alleged hormetic or H response was only an artifact of invalid aggregation. A third flaw is that the study ignored early mortality and confounders (like lower body weights), when it calculated tumor rates, relative to controls. Yet obviously scientists who ignore confounders (that could explain decreased tumor response) can draw no valid conclusions about alleged pollutant and hormetic effects. A fourth flaw is that the study's results have not been replicated; other TCDD studies (in primates) have shown many low-dose adverse effects (Reir et al. 1993, pp. 433–441). Despite these four methodological problems, however, hormesis proponents say the study supports H (Axelrod et al. 2004, pp. 336–338; Thayer et al. 2005, pp. 1272–1275). As already mentioned, Calabrese says that TCDD or dioxin is one of the six main examples that allegedly satisfies H (Calabrese and Baldwin 2003b, p. 691).

3.1 Conceptual obfuscation: concept H versus concept HG

As the previous section argued, because Calabrese ignores sample size, statistical power, statistical significance, data variability, endpoint measured, exposure duration/route/rate, methodological differences among studies, and so on, because he simply looks at earlier studies without doing any experimental research, and because he uses no rigorous scientific conditions for his alleged confirmation of hormesis (H), his conclusions are questionable. Subsequent paragraphs show that, partly as a consequence of Calabrese's questionable ways of "confirming" single-endpoint hormesis (H), there are obvious scientific problems with his using H to generalize—across all biological endpoints/responses/species/subjects/conditions of exposure—to (what I call) HG. HG is the claim that H is "generalizable across biological model, endpoint measured, and chemical class" (Calabrese 2005, p. 643). Calabrese and Baldwin claim

HG "is not an exception to the rule [of linear, no-threshold (LNT) dose-responses, according to which harmful effects increase linearly with dose]—it [HG] is the rule" (Calabrese and Baldwin 2003b, p. 691). Thus, as later arguments show, after invalidly inferring HG from H, Calabrese and his coauthors claim that HG shows that epidemiologists ought not assume that the dose of a toxin is proportional to its harmfulness.

Perhaps the greatest indicator of HG's conceptual problems is that the research, from which Calabrese and Baldwin most often infer HG (Calabrese and Baldwin 2001, pp. 286, 290, 285), includes no epidemiological or field studies (Thayer et al. 2005, pp. 1272–1275)—the studies whose conditions most mimic real-world exposures and easily refute HG. Instead, Calabrese and Baldwin merely generalize from several alleged instances of H, as illegitimately claimed by other authors. For instance, using the preceding flawed studies on TCDD, Calabrese and Baldwin (2003b, 691) say TCDD or dioxin is one of the six main examples that allegedly satisfies H. Yet each alleged instance of H has conceptual problems like those already noted in TCDD studies.

Another HG problem is that Calabrese and others often assume HG merely because they have limited data showing that low-dose-toxic/carcinogenic exposures are harmful. Consequently they have little evidence of errors in claims positing HG. They fallaciously appeal to ignorance partly because low-dose studies require large sample sizes (to detect effects), and most toxicological/carcinogen studies are at higher doses. Without low-dose data, Calabrese and others fallaciously take absence of evidence, against HG, as evidence for it (Calabrese 2005, p. 643; Calabrese and Baldwin 2003b, p. 691).

In fact, fallacies of appeal to ignorance frequently typify SIS. For instance, US National Academy of Sciences' studies have warned that, despite children's known higher sensitivities to pesticides/herbicides, data are lacking to precisely define their higher sensitivities, e.g., to neuro-developmental effects (US NRC 1993). Without neuro-developmental-effects data, chemical-industry scientists often appeal to ignorance, assume low-dose toxins cause no harm, and posit HG (SAB 2000)—as Calabrese does. Many US government regulatory agencies also make similar appeals to ignorance, particularly when the regulated industries push them to assume that no harm will result from some product or pollutant (Shrader-Frechette 1993b, pp. 105–114). For instance, the US Department of Energy explicitly affirmed, in evaluating scientific data regarding the proposed Yucca Mountain nuclear repository, that if "current information does not indicate that the site is unsuitable," then "at least a lower-level [Yucca Mountain site] suitability finding could be supported" (Shrader-Frechette 1993b, p. 106).

As the preceding examples suggest, Calabrese's appeal to ignorance is so common that prominent epidemiologist Kenneth Rothman confirms that most scientists probably equate a lack of data (that shows harm) with evidence for lack of harm (Rothman 2002, p. 126). Perhaps one reason for the error is that, given the burden of proof in US law, courts often require toxic-tort plaintiffs to provide conclusive evidence demonstrating harm, or they assume that the agent did not harm them (Cranor 2006, p. 227).

Besides appealing to ignorance, HG proponents also exhibit an inductive (or invalid-extrapolation) fallacy when they generalize from H claims to HG claims about all

endpoints/populations/time-periods/conditions, on the basis of only a few endpoints /populations/time-periods/conditions that allegedly satisfy H. They also generalize purely on the basis of simple, quantitative, context-dependent, subject-dependent, low-dose measurements, to dose effects that also are determined by when the dose is received, who receives it, how it is received (e.g., the dose rate, health/nutritional status of subjects), and with what it is received (e.g., other exposures to other toxins/carcinogens). The earlier (Sect. 3) case of low-dose cadmium, one of the six main examples that (according to Calabrese) allegedly satisfies H (Calabrese and Baldwin 2003b, p. 691), illustrates why such extrapolation to HG errs. It ignores how individual differences in intra-species genetics/lifestyle/medication/context/endpoint/age affect responses to toxins—as with children's greater sensitivity to toxins. Half-lives of some chemicals are 3–9 times longer in neonates than adults, and neonates may take more than 10 times longer (than adults) to eliminate many chemicals. Likewise, drinking 1.2–2.2 alcoholic beverages per day may have some beneficial maternal effect on some endpoint, while only 0.5 drinks per day can cause adverse fetal behavioral/developmental effects. Even among adults, pesticide-exposure responses may vary significantly because of factors like sevenfold differences in levels of detoxifying enzymes (Axelrod et al. 2004, pp. 336–338).

HG proponents' extrapolation fallacies are especially objectionable because they are inconsistent. On one hand, they explicitly, harshly criticize those who extrapolate from high-dose to low-dose toxic/carcinogenic effects (Cook and Calabrese 2006a, A688). On the other hand, they themselves invalidly extrapolate from H to HG, from some to all biological endpoints; from adult, pure-bred, homogenous animal populations to non-adult, non-pure-bred, heterogeneous human populations; from some, to net, adaptive responses; and from a few, to all, chemicals. For instance, they extrapolate from some to all chemicals when they say that [single-endpoint] hormesis [H] "is a ubiquitous natural phenomenon…across the biological spectrum," therefore generalizable [as HG], so that HG "is the rule" (Calabrese and Baldwin 2003b, p. 691), although they claim H has been demonstrated only for some "inorganic preservatives, antineoplastic drugs, pesticides, and various industrial chemicals" (Cook and Calabrese 2006b, p. 1631). Obviously the move from H to HG, from some to all chemicals, is invalid. HG proponents should practice what they preach and avoid questionable extrapolation, especially because biological grounds suggest that an individual, low-dose, beneficial/adaptive response H (even if it exists) is unlikely to be generalizable as beneficial/adaptive. Why? The cases of cadmium and TCDD (dioxin), two of the six main examples that (according to Calabrese) allegedly satisfy H (Calabrese and Baldwin 2003b, p. 691), already illustrated one reason, that beneficial effects on one endpoint cannot be generalized to other endpoints. Low-dose cadmium, for instance, reduces some tumors in some species but causes excess pre-diabetes, diabetes, pancreas damage, glucose dysregulation, and kidney damage (Axelrod et al. 2004, pp. 336–338). Low-dose TCDD (dioxin) likewise reduces some tumors but increases liver, lung, tongue, and nasal tumors (e.g., Kociba et al. 1978, pp. 279–303). As already mentioned, a second reason that single-endpoint hormesis H cannot be generalized, to all people and groups, is children's roughly ten-times-higher sensitivity to the same dose of a toxin. A third reason—admitted by Calabrese and Baldwin (2001, 285–6, 290)—is that hormesis effects are likely "overcompensations in response to disruptions

in homeostasis." But when organisms overcompensate in response to threats/disruptions, they pay a price. There is no free lunch. As Calabrese admits, so-called hormetic responses are cases of "only reparative responses to the injury that has been done" by the toxin or carcinogen, cases of "reparative overcompensation" (Calabrese 2005, p. 650). While overcompensations, like temporarily adaptive adrenalin rushes, might help fight some assault/toxic threat, they can be maladaptive because of their metabolic costs, e.g., energy expenditures or stress responses that can cause long-term harm. In admitting that hormetic "overcompensation" is a response to injury, and not generally adaptive, HG proponents inconsistently undercut their own case. If so, H cannot be generalized to a supposedly adaptive response HG.

3.2 Conceptual obfuscation among concepts H, HG, and HD

More generally, because HG proponents like Calabrese commit inductive fallacies and appeals to ignorance when they generalize from H to HG, to all chemicals/endpoints/responses/subjects/ages/exposure conditions, they ignore detailed empirical evidence and instead beg the question (Calabrese 2005, p. 643; Calabrese and Baldwin 2003b, p. 691). This question-begging was illustrated earlier when Calabrese and Baldwin claimed that hormesis [HG] is "the rule" (Calabrese and Baldwin 2003b, p. 691), "ubiquitous," demonstrating beneficial effects "across the biological spectrum" (Cook and Calabrese 2006b, p. 1631). Yet, as already mentioned, effects on children, reparative overcompensation, and cases like cadmium and TCDD or dioxin—two of the six main examples that, according to Calabrese, allegedly satisfy H (Calabrese and Baldwin 2003b, p. 691)—show that beneficial effects H are not generalizable to HG, to all endpoints/people/contexts/ages/chemicals/biological processes. But this unjustified, question-begging assumption (that H is generalizable to HG, the rule) is precisely what Calabrese and others are supposed to argue, not merely assume.

Why do Calbrese and others want to claim HG is "the rule" and that harm from toxins is not proportional to dose (Calabrese and Baldwin 2003b, p. 691)? They want to use HG to justify weakening regulations, to allow low-dose toxic exposures. From HG, Calabrese and others want to infer a third hormesis concept, HD. (What I call) HD is the claim that "a strong case can be made for the use of hormesis as a default assumption in the risk-assessment [therefore risk-regulation] process," the default assumption that low-dose toxins are not harmful (Calabrese and Baldwin 2001, pp. 286, 290, 285). To support their move from HG to HD, Calabrese and others make three questionable claims, none of which is either argued or supported with empirical data. All three claims beg the question because they are simply asserted by fiat. They are that accepting HD would improve public health, save money, and lead to better science.

(1) Because low-dose effects of some chemicals (like vitamins) have some beneficial effects, and because accepting HD would "not only prevent excess disease or death over background but also promote better health," therefore "public health might be better served by setting exposure standards [HD] at levels using data collected, based on the hormetic model [HG]" (Cook and Calabrese 2006a, p. A688; Cook and Calabrese 2006b, pp. 1632–1634).

(2) Developing HD regulatory policies that are based on accepting the hormetic model HG would have "economic implications" that are "substantial" because HD "could lead to less costly…clean-up standards" for pollutants (Calabrese and Baldwin 2003b, p. 692).
(3) Developing HD regulatory policies, based on accepting the "hormetic model" [HG], would promote science because HD would "encourage the collection of data across a broader range of dose" (Cook and Calabrese 2006a, p. A688).

Because Calabrese and his coauthors have defended none of the previous three claims, and because the dominant scientific position is that there are substantial reasons to believe that (weakening pollution standards by) accepting HD would greatly harm public health (e.g., Thayer et al. 2005), especially for vulnerable groups like children, Calabrese's three question-begging claims need no further consideration.

However, at least five considerations (a,b,c,d,e in later paragraphs) suggest that inferring HD from HG is questionable. The general reason is that, even if HG were true (i.e., low-dose toxic/carcinogenic exposures caused beneficial responses for all endpoints/responses/subjects/ages/exposure conditions), this would constitute only necessary, not sufficient, conditions for inferring HD. Because default rules (like HD) are used in situations of uncertainty, their acceptance requires not only scientific judgments (like those regarding H and HG). Rather, their acceptance also requires ethical/policy judgments, e.g., how much HD risk is acceptable, whether HD risk-beneficiaries should be the same people as HD risk-bearers, whether alleged HD benefits are compensable and worth the risks, and so on (Shrader-Frechette 1993a). Because HD involves ethical/policy/regulatory/welfare conclusions, it cannot be validly inferred from H and HG—which include no ethical/policy/regulatory/welfare premises. That is, from purely scientific or "is" claims (e.g., H, HG), one cannot validly deduce ethical or "ought" claims (e.g., HD) because of the is-ought fallacy in ethics. The fallacy is an error because no evidence about an alleged scientific fact—what "is" the case—is ever enough to deduce an ethical conclusion—what "ought" to be the case (Hume 1975; see also Shrader-Frechette 1993a, pp. 24, 156). In using only H and HG to invalidly infer HD, Calabrese and others ignore ethical conditions required for validly inferring HD. These ethical conditions include showing that accepting HD would be (a) just, equitable, and compensable; (b) worthy of free informed consent by risk bearers; (c) consistent with basic rules of biomedical ethics, as set by Nuremburg, Belmont, Helsinki, the Common Rule and other classic sources (Jennings et al. 2003); (d) an adequately health-protective stance, in the face of uncertainty about precise risks (Shrader-Frechette 1993a, pp. 100–145; Shrader-Frechette 1994, pp. 9–12, 23–118; Jennings et al. 2003); and (e) operationalizable.

Calabrese and others have completely ignored ethical conditions like (a)–(e), and it is unlikely HD could meet them. HD arguably could not satisfy (a) because HD beneficiaries would be industries that avoided controlling low-dose pollution, at the risk/expense of others, potential pollution victims. Such a situation could violate fairness, equal treatment under law, and due process, including rights to compensation from HD-related harm. Ignoring fairness issues, Calabrese and others note only the expedient, supposedly desirable economic consequences (to industry) of accepting HD (e.g, Calabrese and Baldwin 2003b, p. 691). Moreover, because industrial pollut-

ers would be the primary beneficiaries of HD, whereas their pollution victims would be the primary HD risk-bearers, it also is unlikely that victims would consent to HD risk-imposition (Shrader-Frechette 1993a), thus unlikely that HD could meet consent condition (b). After all, people generally agree to bear risks (e.g., those associated with HD) only when they get something in return. Breast-cancer patients may take tamoxifen—despite its uncertain but excess risks of thrombosis, stroke, uterine hyperplasia, uterine cancer, and uterine sarcoma (Axelrod et al. 2004, pp. 336–338), because they get something in return, reduced breast-cancer-recurrence risk. Likewise, virtually all pharmaceuticals impose one risk, in exchange for reducing another risk. Because HD victims would get little/nothing in return, from bearing increased HD risks, their consent to HD is unlikely (Jennings et al. 2003), and HD would not meet condition (b). Nor would HD likely meet condition (c) because classic biomedical-ethics codes require that potential risk victims exercise "rights to know" the risks imposed on them (Faden and Beauchamp 1986; Beauchamp and Childress 1989). Yet because many pollution victims are unaware of their increased risks (e.g., polluting industries do not distribute right-to-know disclosure forms to neighborhoods where they release toxins/carcinogens), universal rights to know are unlikely to be fully recognized. Nor would pollution victims likely enjoy an acceptable benefit-risk ratio, another requirement of classic biomedical-ethics codes. Because most benefits of HD pollution would go to industry, while the public would bear most HD risks, HD arguably would not meet condition (c) (Jennings et al. 2003; Beauchamp and Childress 1989). Nor would HD likely meet another necessary biomedical-ethics condition, that exposures not target a special group who would bear significantly higher risks (Shrader-Frechette 2008c; Jennings et al. 2003; Hume 1975). Yet by accepting HD, not LNT, Calabrese targets a vulnerable group, children, as they are roughly 10 times more sensitive to toxins/carcinogens (Shrader-Frechette 2007). Because meeting ethical condition (d), providing an adequately health-protective stance, arguably requires protecting vulnerable populations like children, HD also appears unlikely to meet (d).

Finally, meeting ethical condition (e) also is arguably unlikely. At least four reasons suggest that applying HD to the real world (and allowing no more than low-dose-toxic exposures) is impossible/not operationalizable. One reason is that (i) each person's toxic/carcinogenic exposures cannot be individually titrated, to achieve total exposures that are only low-dose, because every person's doses (from tens of thousands of pollutants) cannot be measured, every instant, in order to provide immediate feedback to polluters, about whether total exposures exceed low dose (Axelrod et al. 2004, pp. 336–338). Moreover, (ii) HD regulations (allowing no more than low-dose-toxic releases) cannot guarantee only low-dose exposures, because tens of thousands of pollutants together drive total exposures beyond low doses. For instance, by the time a child is born, background exposures already have given her more than a low dose of ionizing radiation, and effects of all radiation doses are cumulative, with no threshold for risky effects at any dose (Shrader-Frechette 2001, pp. 319–342). As the radiation case illustrates, HD could never apply to most exposures. Besides, Calabrese and Baldwin say maximal low-dose hormetic/beneficial responses occur at doses that are about one-fifth of the no-observed-adverse-effect level or NOAEL (Calabrese and Baldwin 1999, pp. 725–732). This means that simultaneous exposure to five equally potent toxins, each acting on the same biological mechanisms, and each at one-fifth

NOAEL, would move victims from the low-dose, to adverse-effects, range. Moreover, repeated US Environmental Protection Agency and Centers for Disease Control studies show all US citizens receive doses of not only 5, but thousands of, chemicals whose residues are measurable in their blood or tissue (Axelrod et al. 2004, pp. 336–338; Thayer et al. 2005, pp. 1272–1275). Immunological evidence also shows that combining many low-dose toxins often causes synergistic, not merely additive, effects—as when people are exposed to TCDD and dioxin-like compounds, to radon and smoking, to asbestos and smoking, to alcohol and smoking; additional exposures add to the total, harmful, immunologic and estrogenic burden (Lang 1995). Yet TCDD (dioxin), ionizing radiation, and alcohol are three of the seven main examples that (according to Calabrese) allegedly satisfy H (Cook and Calabrese 2006b; Calabrese 2005; Calabrese and Baldwin 2003b, p. 691). Given these combinations of exposures, as well as multiple exposures, HD thus is irrelevant in a world in which virtually everyone already has had more than low-dose-toxic exposures. Even if people were not already overloaded with toxins/carcinogens, (iii) HD would not be operationalizable because it would harm sensitive populations. Roughly 25% of the population (e.g., children, pregnant women) are more sensitive to toxins, thus more likely to exhibit harmful responses even to low-dose exposures. Also, (iv) HD regulations would not be operationalizable because of intra-species differences in absorption; the same toxic releases can cause radically different doses among people. For instance, adults absorb 10–15% of lead entering their gastrointestinal tracts, while pregnant women and children absorb about 50% (US NRC 1993, p. 187). Reasons (i)–(iv) thus mean HD cannot meet (e) the operationalizability problem. If not, HD is inapplicable to real-world policymaking and impossible to implement. Yet by the "ought implies can" rule, people ought not be required to do what is impossible (Aristotle 1985), thus ought not be required to adopt HD. Calabrese, Cook, and Baldwin forget this fundamental ethical rule (NRC 2004, pp. 3, 110–113; Calabrese and Baldwin 2003b, p. 691; Calabrese and Cook 2005, p. 26). Instead they claim that it is not US "policy to protect the most sensitive in the general population" (Cook and Calabrese 2006b, p. 1633)—a claim that both implicitly admits the operationalizability problems with HD and begs the question that sensitive populations can be ignored.

3.3 Bait-and-switch hormesis arguments

If preceding paragraphs are correct, no hormesis concept—H, HG, or HD—has both scientific validity and regulatory relevance. On one hand, H is scientifically valid (because one biological endpoint, among thousands, often shows a beneficial effect of some toxin/carcinogen), but trivial and irrelevant to the Calabrese/chemical-industry deregulatory goals, because sound regulations require HG—net-beneficial effects for all lifetimes/ages/endpoints/contexts/responses/individuals. On the other hand, although positing HG is relevant to chemical-industry deregulatory goals, it is scientifically flawed because of invalid extrapolation, appeals to ignorance, and ignoring biological "reparative overcompensation" costs. Similarly, although positing HD is relevant to chemical-industry deregulatory goals, it is scientifically and ethically indefensible, given its failure to meet health-protection, fairness, democratic-consent, rights-to-know, biomedical-ethics, and operationalizability norms.

If none of the hormesis concepts (H, HG, HD) has both regulatory relevance and scientific plausibility, why have Calabrese and others been able to publish their pro-hormesis essays in prestigious journals like *Nature* and *Environmental Health Perspectives* (e.g., see Cook and Calabrese 2006b, pp. 1631–1635; Calabrese and Baldwin 2003b, p. 691; Calabrese and Baldwin 1999, pp. 725–732)? Three possible explanations come to mind. Explanation (1) is that many Calabrese essays are "opinion" pieces, not basic empirical research, thus not subject to standard scientific peer review. Explanation (2) is that, when Calabrese and others claim some study illustrates H, only scientists who know this study, in detail, can evaluate Calabrese's hormesis claims (see Thayer et al. 2005, pp. 1272–1275). If journal reviewers do not know these other studies, they may illegitimately assume Calabrese is right about them. Explanation (3) for the prominence of Calabrese's hormesis articles is that journal referees may have been misled by Calabrese's failure to distinguish different hormesis concepts (H, HG, and HD). By calling all three concepts by the same name, "hormesis" (e.g., Cook and Calabrese 2006b), Calabrese may have confused reviewers. They may have recognized the scientific validity and triviality of one hormesis concept (H), the regulatory relevance of another hormesis concept (HD), then erroneously concluded the same hormesis concept had both scientific validity and regulatory relevance.

Explanation (3) appears plausible, because both Calabrese's explanations and his responses to objectors encourage confusion among H, HG, and HD. For instance, Cook and Calabrese commit the fallacy of equivocation when, under the heading "FDA Regulation of Hormesis," they claim to be "proponents of hormesis" and urge "regulation of hormesis" (Cook and Calabrese 2006b). They arguably should have said "proponents of HD," and "regulation via HD," because claims positing H are trivially true and irrelevant to regulation, while only invalid claims, positing HG and HD, are relevant to regulation.

Calabrese also equivocates in answering critics. For instance, without using my labels, Thayer et al. attack Calabrese's claims positing HG and HD (Thayer et al. 2005, pp. 1272–1275). Yet Cook and Calabrese respond equivocally, by defending only H (not HG and HD), and saying "hormetic dose-response curves [H] have been observed for a large number of individual agents" (Cook and Calabrese 2006b). Thus Cook and Calabrese first "bait" the reader, by supporting a biologically/ethically doubtful hormesis concept (HD). After scientists like Thayer et al. 2005 respond to this bait, by criticizing HD, Calabrese and coauthors "switch" the critics by defending an hormesis concept (H), one not at issue. Cook and Calabrese thus may appear correct, but only because they fallaciously equivocate, defend (H) a concept not at issue, and use the same label ("hormesis") for both (trivially true) claims positing H and (scientifically invalid) claims positing HG and HD,

3.4 Conflicts of interest and conceptual obfuscation

Investigating Calabrese's scientific and ethical errors reveals many insights about SIS. One is that much SIS may be caused by industry-related financial conflicts of interest. Calabrese and Baldwin note the financial stakes in hormesis debates, admitting that "the external influence of the enormous cost of environmental cleanups and the proper

allocation of limited societal resources have strongly encouraged a…reexamination of…hormesis" (Calabrese and Baldwin 2001, pp. 286, 290, 285). In less flattering terms, a US National Academy of Sciences' report warned about questionable chemical-industry motives behind promoting weakened regulations for low-dose chemical exposures (Oleskey et al. 2004, pp. 114–119; Lockwood 2004, pp. 1908–1915). The academy said "pesticide manufacturers" and other "economically interested third parties" are funding studies, trying "to justify reducing" chemical-safety standards, "thereby increasing the acceptable or safe human exposure level…that might otherwise have been precluded under [current]…safety standards" (NRC 2004, pp. 110, 112–113, 3).

A second insight is that SIS practitioners, like Calabrese, often violate standard disclosure guidelines regarding conflicts-of-interest. The guidelines of Calabrese's public/state employer, University of Massachusetts (1997, p. 38) dictate that the "University does not require disclosure and review of every Conflict of Interest, but only those involving a Financial Interest and… Compensation in an aggregate amount greater than $10,000 within the prior twelve-month period that is received by or contractually promised to a Covered Individual." (As later paragraphs show, Calabrese appears to have between $810,000 and more than $3,000,000 in funding (far more than $10,000) for which he has not disclosed his sources of funding.)

Likewise, the *Journal of the American Medical Association*, International Committee of Medical Journal Editors (ICMJE), Council of Science Editors (CSE), World Association of Medical Editors (WAME), and others have policies requiring authors to specifically indicate whether they have conflicts of interest regarding subject matters about which they write (Flanagin et al. 2006, p. 220). Calabrese, however, fails to reveal many funding sources. Years ago, he disclosed some chemical-industry support, e.g., from the Texas Institute for Advancement of Chemical Technology (Calabrese and Baldwin 1998; Calabrese et al. 1999, pp. 261–281), funded by Dow, BASF Chemical, Bayer Chemical, Shell Chemical, and Syngenta pesticide company (Axelrod et al. 2004, pp. 336–338).

However, in 2007 Calabrese's official University of Massachusetts online resume failed to disclose funding sources for 3 (of his 9) research projects, responsible for $810,000 (Calabrese 2006a). In another 2007 official University of Massachusetts online resume, Calabrese listed receiving more than $3 million from unnamed sources. Named sources include Atlantic Richfield Oil (ARCO), Chemical Manufacturers Association (CMA), Dow Chemical, Exxon Oil, Reynolds Metals, and Rohm and Hass Chemicals (Calabrese 2002a).

After the author blew the whistle, in a January 2008 toxicology publication about Calabrese's conflicts of interest and failure to disclose funding sources (Shrader-Frechette 2008c,d), Calabrese began disclosing even less, and his online university resumes changed dramatically. Individuals using the public-university website and trying in 2008 to access his first resume (Calabrese 2002a)—whose 2007 web version had $3 million in undisclosed funding sources—instead received the message: "FORBIDDEN: You don't have permission to access" this resume (Calabrese 2002b). Instead of providing the second resume (Calabrese 2006a)—whose 2007 web version had $810,000 in nondisclosed-funding sources—the shortened 2008 Calabrese resume had removed all references to chemical-industry funding, yet listed $570,000 from

undisclosed sources (Calabrese 2006b). The website also said one must "contact the professor" to obtain the complete resume (Calabrese 2006b). Calabrese's responses to whistleblowing (about his conflicts of interest and funding-disclosure failures) thus were increasing his funding-source cover-up and decreasing his disclosures.

A third SIS insight, gleaned from the Calabrese case, is that often authors publish with industry representatives/employees (having conflicts of interest) but fail to disclose their coauthors' affiliations. For instance, although Calabrese-coauthor Ralph Cook (e.g. Cook and Calabrese 2006a, p. A688; Cook and Calabrese 2006b, pp. 1631–1635), was "Director of Epidemiology, Dow Chemical, USA…Midland Michigan" and remains a Dow consultant (AJE 1984, p. 24), Calabrese ignores Cook's Dow ties and lists his affiliation merely as "RRC Consulting, LLC… Midland Michigan 48640–2636" (Cook and Calabrese 2006a, p. A688; Cook and Calabrese 2006b, pp. 1631–1635).

A fourth SIS insight, also from the Calabrese case, is that when other scientists point out SIS conflicts of interest, industry representatives (like Calabrese), attempt to censor whistleblower disclosures. For instance, when the author published an article (in a journal edited by Calabrese) criticizing flawed science in Calabrese's work (Shrader-Frechette 2008c, p. 47) someone (without the author's or issue-editor's consent) deleted from the page proofs the author's endnote 41, documenting Calabrese's conflicts of interest and missing industry-funding disclosures. The issue editor (Dr. Kevin Elliott) had to force Calabrese to re-instate the deleted material (Elliott 2007). Later, when this article was reprinted in *Human and Experimental Toxicology* (HET) (Shrader-Frechette 2008d, pp. 647–657) someone again deleted endnote 41 (without author consent) and moved some of its content to small print at the bottom of an irrelevant text page. These changes occurred when the Calabrese-edited journal sent the article to HET. Again Dr. Elliott had to pressure HET to re-instate endnote 41, although two sentences were deleted from it (Elliott 2008a,b). Why the deletion? The HET editor, Lee John Rourke, said he could not include the author's entire endnote 41 without Calabrese's permission (Rourke 2008a,b). Yet because HET is located overseas, and its editors are scattered throughout the world, phone exchanges about these problems were impossible on a practical level. Despite repeated email exchanges, Rourke would reveal neither why he needed Calabrese's permission to reverse the deletions from the author's paper, nor why he allowed Calabrese to cut the sentences from the author's paper (Rourke 2008a,b). Instead HET appeared to "stonewall" all requests for information and explanation. Facing obvious conflicts of interest in the case, Calabrese forced HET to cut these sentences from the author's article:

> (Author's note: When the author received these page proofs on 10-1-08, someone had deleted the relevant endnote number from the text; moved this endnote material to the bottom of an earlier page; and changed the location in the text where this material was cited; these unauthorized changes were made neither by the production editors at Sage nor by the issue editor, Dr. Kevin Elliott. Earlier, when this article appeared in *Biological Effects of Low-Dose Exposures*, a journal edited by Dr. Calabrese, someone also tried to delete completely (from the page proofs) the material in this endnote.)" (Shrader-Frechette 2008a).

4 What philosophers of science can do to help prevent scientific misconduct and flawed science policy

One of the most obvious questions, arising from this account of apparent scientific misconduct, is how it might be prevented/ameliorated. What can philosophers of science do, in the face of problems such as Calabrese's misleading the judge in the Allen case, Calabrese's failing to reveal funding sources that suggest he has financial conflicts of interest, and Calabrese's editing (and HET's and BELLE's permitting him to edit) other authors' scientific papers in ways that serve Calabrese's financial interests? Although there is no space here to discuss such questions in detail, several general responses are appropriate.

A first step is for philosophers of science to recognize that their primary professional duties, like those of scientists and engineers, are to protect the public. Virtually all professional codes of ethics recognize that scientific professionals have "an overriding duty to protect the public" (Shrader-Frechette 1994, p. 71), in large part because special professional abilities generate special professional responsibilities. These responsibilities include speaking out about science-related threats and, if necessary, working with professional societies, universities, journals, or government regulators, to blow the whistle on misconduct (AAAS 1980; David et al. 1992; Shrader-Frechette 1994).

Apart from science-related *professional* duties to protect the public, philosophers of science also have related duties as citizens. As argued elsewhere, upper-middle-class citizens often profit from flawed risk regulations and pollution management, because disproportionate pollution burdens fall on poor/minority, not wealthier, neighborhoods. Because upper-middle-class citizens tend to receive unfair medical and economic benefits, when "their share" of pollution (e.g., from waste management, incinerators, electricity production) is disproportionately imposed on poor people/minorities, they have justice-based duties to help correct the flawed science that is often used to justify such unfair risk distributions (Shrader-Frechette 2002, 2007, pp. 113–210).

On the research side, one way to fulfill these duties is to choose at least some practical, professional-research projects that can contribute to the public good. Given public-funding sources of much professorial-salary compensation, doctoral training, and research funding, philosophers of science do not have complete liberty to do whatever research they wish; instead they also have some contract-based obligations to do research that helps protect the public and the scientific enterprise (Shrader-Frechette 1994, pp. 25–44). One way of doing so is writing popular articles and newspaper op-eds, related to one's areas of expertise, perhaps after publishing similar material in technical/scholarly journals. Another way is publishing in broader professional journals like *Accountability in Research*, so that wider ranges of professionals learn of science-related policy/methodology problems.

On the teaching side, one way to fulfill the professional duties of philosophers of science, to protect the public, is by teaching "fully naturalized" philosophy of science, instead of focusing only on epistemic considerations. Revealing how social-political and financial influences often skew scientific methodology, one can help students analyze the methods/models/assumptions underlying alternative stances on contemporary, welfare-affecting, science-related disputes. For instance, every semester the

author and her students respond to a number of the roughly 3000 *Federal Register* "requests for comments" that accompany draft versions of federally mandated risk assessments, technology assessments, or environmental impact assessments that are required, prior to any regulatory change, new pollution standard, facility siting, or technological expansion. By carefully assessing, and responding to, the methodological/modeling flaws in these draft documents (and there are always flaws, because the assessments typically are done by those with commercial reasons for promoting some regulation, facility, or product), students gain four benefits. (1) They obtain real-world understanding of how/why scientific method goes wrong. (2) Students provide free scientific help, often to poor/minority communities who otherwise would not have it and who often are disproportionately affected when flawed science is used to justify often-questionable facility sitings, expansions, or regulations. (3) Students see that "scientific citizenship" pays off, because federal law forces regulators to respond to citizens' comments, thus clean up their science, improve science-based decisionmaking, and (often) abandon projects that are scientifically indefensible. (4) Students also sometimes develop publications from their projects. The preceding strategy, of analyzing draft assessments, easily can be used in the classroom (Shrader-Frechette 2009a), especially in conjunction with books that carefully outline how special interests can skew scientific methodology/models and thus harm the public (see Krimsky 2003; Cranor 2006; Shrader-Frechette 2007, pp. 3–112).

On the public-service side, by becoming involved in local science-related debates, philosophers of science often can assist labor unions or citizens groups (whose members may be harmed by occupational or environmental pollution or regulations). If philosophers of science are helpful to their communities, soon they will be asked to assist other experts, government groups, national non-governmental organizations (NGOs), and minority or poor plaintiffs, who otherwise could not afford scientific consultants to document health/safety/regulatory threats (e.g., Shrader-Frechette 2002, pp. 71–94). If philosophers of science both work with NGOs and publish books or scholarly articles on science-related, welfare-affecting issues, then eventually others will seek their scientific advice. Largely as a result of scientific, area-specific publications (e.g., on pesticide threats, nuclear-energy risks), the author has been invited to join important science-advisory/policy groups, e.g., boards/committees of the US National Academy of Sciences; regulatory committees of the US Department of Energy, the US National Commission on Radiological Protection and Measurement, etc.; international radiation-standard-setting committees (as the US delegate) of the International Commission on Radiological Protection; and various science-advisory committees, e.g., the US Environmental Protection Agency Science Advisory Board. A key benefit of philosophers of science doing such work is that (unlike many scientists) they typically have no financial conflicts-of-interest and frequently also have expertise in ethics.

What are the chances that philosophers of science might be effective/successful in helping to protect the public, prevent scientific misconduct, and guide science-related public policy? To illustrate the potential for such work, the editors specifically asked the author to give some examples of successes. Consider 5 typical examples, from work in Africa, Louisiana, Kentucky, Tennessee, and Nevada (see Shrader-Frechette 2009b).

- "Disposal" of US toxic and radioactive wastes, in poor African nations, has been prohibited by United Nations convention, in part as a result of the author's pro-bono scientific work with African nations. Organized internationally by the World Council of Churches, this pro-bono work outlined scientific and public-health risks from Africans' accepting these wastes (see Shrader-Frechette 1991).
- In response to a pro-bono request from the Sierra Club Legal Defense Fund (now Earthjustice), the author and her students stopped the siting of a substandard uranium-enrichment facility in Homer, Louisiana—a poverty-level, all-minority community opposed to the facility. Revealing crucial scientific flaws in site health-risk assessments and economic analyses, in 1998 the author and her students help achieve what is generally acknowledged as the first major US environmental-justice victory (see Shrader-Frechette 2002, pp. 71–94).
- According to a US Nuclear Regulatory Commission official, the US no longer allows shallow land burial of radioactive waste and transuranics, partly because the author documented extensive scientific flaws in government hydrogeological methods and models for the nuclear dump in Maxey Flats, Kentucky (Shrader-Frechette 1993b, pp. 49, 55–60).
- In response to a pro-bono request from the National Association for the Advancement of Colored People (NAACP), the author and her students analyzed the scientific methodology/models of government risk assessments that alleged no harm (to the local Black community of Scarboro, Tennessee) from repeated radionuclide, metals, and solvent releases from Oak Ridge National Laboratories. Re-analyzing the government's data, the author and her students showed great pollution harm and provided these results to the NAACP and pro-bono Tennessee attorneys—who used them to sue for compensation, health care, and cleanup in Scarboro (see Shrader-Frechette 2009b).
- Working with the Nevada state government and a Nevada citizens' group, the Nuclear Waste Project Office (NWPO), the author repeatedly and continuously criticized flawed scientific methodology and models used to justify siting the Yucca Mountain (Nevada) high-level-nuclear-waste facility. The state government provided the author's book, scientifically analyzing the site, to all Nevada federal, state, and local officials (Shrader-Frechette 1993b), and the author wrote some of the state's scientific "intervenor" documents, as it battled federal attempt to impose the facility. After 20 years of analyses/fighting, site-completion funding finally has been cut.

In using pro-bono philosophy of science work, to help correct scientific errors and protect the public from science-related harms, at least two considerations must be kept in mind. Perhaps the most important is that, (1) to be effective in science-policy disputes, philosophers of science need a wealth of area-specific scientific knowledge, perhaps best gained through post-docs, sabbaticals, coursework, or additional degrees. This means that one cannot be a jack-of-all-sciences. It is no accident that most of the preceding examples of successes focus on narrow, area-specific scientific knowledge dealing with risk-assessment methods, radiobiology, and hydrogeological transport of radionuclide-wastes. Another crucial point is that (2) the work is slow to yield successes. The author has been assessing hydrogeological and radioactive-waste risk

methods/models, and the Yucca Mountain project, since the late 1980s. Only in 2009, 20+ years later, did this work culminate in the apparent stoppage of Yucca Mountain work/funding.

5 Conclusion

Apart from these practical suggestions for how philosophers of science might help serve both science and society, perhaps the biggest lesson from the Calabrese case is that ethical shortcomings often accompany SIS scientific shortcomings. The Calabrese case illustrates at least four ethical problems, Calabrese's (1) having financial conflicts-of-interest (extensive industry funding) that could explain his scientific errors; (2) failing to follow guidelines for disclosing financial conflicts-of-interest; (3) failing to disclose coauthors' correct affiliations and conflicts-of-interest; and (4) censoring whistleblowers who reveal Calabrese's questionable behavior. One benefit of ethically analyzing (1)–(4) is their shedding light on whether Calabrese's scientific errors were deliberate. SIS ethical violations—like (1)–(4)—suggest that SIS scientific errors (benefitting one's industry funders) may not be unintentional. Such cases of SIS suggest that philosophers of science cannot afford to be ethically naïve about why science methods often are flawed.

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