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Commentary: Flawed Science Delays Smelter Cleanup and Worsens Health

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For 6,000 years, humans have known about smelter hazards. Yet these metals threats continue. Why? This commentary provides one preliminary answer. It (1) summarizes the history of smelter pollution and (2) suggests that at least 3 problems—especially flawed smelter-polluter science—allow continuing health threats. It (3) illustrates this flawed science by using one of the most dangerous of U.S. former smelters, in DePue, Illinois. There polluters are avoiding violating the law yet trying to minimize smelter–caused health threats, thus clean-up costs, by using two questionable scientific claims. The causality-denial claim denies that smelter metals cause neurodegenerative diseases. The biomagnification-denial claim denies that food-chain biomagnification of smelter metals can put citizens at risk. The commentary shows both claims err, and (4) suggests ways to address flawed smelter science and resulting health harms.

Keywords: biomagnification, DePue, Doe-Run, LaOroya, metal, neurodegeneration, Renco, smelter

Governments usually have taken heavy-metals poisoning of wine very seriously. In 1427, both French and Spanish authorities issued edicts prohibiting wine adulteration with the typical sweetener, boiled-down grape juice made in leaden pots. Lead-acetate, leaching from the pots, makes the sweetener toxic. By 1478, German authorities made adulterating wine with this lead sweetener a crime punishable by death. Although the ancient Greeks and Romans also boiled down grape juice in leaden containers, they too recognized the resulting metals' threats. Thus, by the 400s B.C., Hippocrates was able to accurately describe lead poisoning and how to prevent it (Lessler, 1988).

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BACKGROUND ON SMELTERS

Today, however, 600 years after western governments prohibited metals-poisoned wine, society allows another form of heavy-metals poisoning, from smelters. World Health Organization (WHO) data show the three industries—that prematurely kill and disable the most people globally—all involve metals smelting. These are primary smelting, refining, and purifying metal ores with furnaces and chemicals; mining or ore processing; and secondary smelting, that is, recycling and refining metals such as lead from batteries (Mills-Knapp et al., 2012, p. 5; WHO 2009).

Metals smelters are risky because, through airborne particulates, water, soil, and food grown in smelter-contaminated soil (Brougham et al., 2013, Robins and Hagan, 2012), smelters expose people to metal-metalloid neurotoxins and carcinogens such as arsenic, cadmium, lead, and mercury that have no safe dose and no biological use (Csavina et al., 2012; Landrigan, 2011; Pichery et al., 2011; Zahran et al., 2009; Bellinger., 2008; Lanphear et al., 2005; WHO, 1999). Smelters also expose people to other metals, such as copper or zinc, that are neurotoxic and carcinogenic even at doses only slightly above the 1 mg obtained daily through food and needed for metabolism (WHO, 1996; Pope et al., 2007).

Of course, smelters are also beneficial. They contributed to the rise of the Roman Empire which, before the birth of Christ, produced a total of 88,000 tons lead and 16,500 tons copper, an output unmatched until the 18th century. Yet 4,400 tons/year of Roman-smelter metals proved "catastrophic for . . . millions of individuals" in areas like the Balkans, Britain, Greece, Jordan, and Spain; there, smelters reduced intelligence and learning ability "on a substantial scale" (Keys, 2003, pp. 47-48). Excavations reveal that Britons living near Roman-Empire smelters had 10 times or more lead in their bodies than did city dwellers, many of whom nevertheless ate from leaden vessels. Ice cores show Roman-metal particulates also travelled globally, dumping 880 tons copper and 440 tons lead on Greenland alone (Keys, 2003, pp. 47-48; see WHO, 2010; Kabir et al., 2012; U.S. Congress, 2012).

Such smelter-pollutant harm is "substantial and long-lasting" because smelter metals, given that they are elements, can never disappear, be broken down, or be biodegraded. Thus metals in ancient, near-smelter soils still cause metals-contaminated pastures, plants, animals, humans, milk, and meat. They still cause increased mental retardation, lowered I.Q., violent behavior, and neurological and other problems among nearby residents. The globe's highest-average-soil-metals concentrations, especially lead, zinc, copper, and cadmium, are all around current and former smelters (U.S. Congress, 2012; Kabir et al., 2012).

The result? An "epidemic" of chronic metals toxicity (Grandjean and Landrigan, 2014), "a major public-health problem" (Todd et al., 1996). Indeed,

most near-smelter, soil-metals levels "exceed the common regulation-guideline levels enforced by many countries" (Kabir et al., 2012). Unless "three feet of topsoil" is removed from near-smelter residential yards, living or growing vegetables there poses health risks (U.S. Congress, 2012; Horak et al., 2006).

Why has it been easier for governments to regulate heavy-metals poisoning from wine than from smelters? Several reasons help explain why current and former smelters continue to pose global health threats. These include poor government regulation and enforcement, inadequate health standards, polluter abdication of responsibility, and flawed polluter science.

POOR SMELTER REGULATION AND ENFORCEMENT

Because of poor government record-keeping and inadequate resources for former-smelter-site cleanup, even the U.S. government is not aware of many of its former-smelter sites. In 2001, scientists used historical evidence to find 639 U.S. secondary-lead smelters that operated only 50 years ago, 1931–1964. Yet, only 170 (27%) were listed in the U.S. Environmental Protection Agency (US-EPA) database, although 90% appeared to have soil-metals levels above allowable norms (Eckel et al., 2001).

In 2012, 11 years after this highly-publicized discovery, investigative-newspaper reporters did follow-up studies on former U.S. smelters. On one hand, they found government has left near-smelter families "in harm's way, doing little to assess the danger around many sites" (Young et al., 2012). Near most current and former sites, children have metals-related diseases and dysfunctions; hundreds of thousands of people live within four miles; and exposures continue from high soil-metals and windborne-particulate dispersion (Beals, 2004).

On the other hand, US-EPA says Congress has given it inadequate resources to handle smelter threats. It has only about 80 people and $20 million/year to assess hundreds of former U.S. smelter sites. Yet assessment and cleanup for a single site can be $250 million or more (Young et al., 2012).

Another reason for former-smelter-site threats is flawed government enforcement of regulations. For instance, in 1997 the Peruvian government allowed St. Louis–based Renco to buy La Oroya–Doe Run copper-lead-zinc smelter, one of the world's largest, with 3,500 employees. Peru allowed the sale, for only $247 million, because Renco contracted to do a multimillion-dollar clean-up and smelter-pollution-reduction. Yet by 2014, after 17 years, Renco has done only part of the mandated 1997 cleanup. In fact, it has worsened the heavy-metals poisoning of most LaOroya children because of releasing 3,312 tons particulates/year, 847 tons lead/year, 423 tons arsenic/year, 226 tons nitrogen oxides/year, and 43 tons cadmium/year. Though Renco's

global sales are $8 billion/year, it claims its contracted pollution-cleanup is too costly (U.S. Congress, 2012; Tegel, 2012).

Of course, La Oroya's smelter operated for 90 years, so Renco has not caused all its pollution. Nevertheless, U.S. investigations show that since 1997, Renco has been in "constant noncompliance with environmental standards." Although average blood-lead levels in the town of 35,000 are triple the WHO limits, Peruvian-government enforcement also has been lax, partly because the smelter is the town's main employer. Perhaps because Renco is a U.S. corporation, Congressional investigators claim the United States has not enforced the health and environmental provisions of the United States–Peru Trade-Promotion Agreement. Enforcement would require Renco to accept Peru's environmental regulations. Unsurprisingly, in 2007 the Blacksmith Institute named La Oroya the world's sixth-worst-polluted site (U.S. Congress, 2012; see Mills-Knapp et al., 2012; National Geographic News, 2007).

Partly in response to La Oroya pollution and publicity, Peruvian authorities recently shut the smelter until Renco honors its 1997 contract and follows environmental regulations. However, because Renco wants to resume operations without doing contracted cleanup, in 2010 Renco initiated international arbitration. Demanding $800 million Peruvian compensation for temporarily shutting the smelter, and arguing against $21 million in Peruvian environmental fines, Renco has alleged violation of its rights under the U.S.–Peru Free Trade Agreement. The case is still pending (U.S. Congress, 2012; FIDH, 2013).

Similar metals-pollution problems beset smelters in developed nations. In 2009, US-EPA said that within a mile of Renco's Missouri smelter, one-third of residences had soil-lead levels exceeding US-EPA limits, although Renco had already performed court-ordered lead remediation on them (Schmidt, 2010, p. A79). In 2010, Renco had to pay $65 million for U.S. environmental violations, and another 7 million in civil penalties. Rather than install pollution equipment, Renco decided to close the Missouri smelter at the end of 2013 (Thorsen, 2010).

FLAWED SMELTER STANDARDS AND FAILED POLLUTER RESPONSIBILITY

Closing smelters, however, does not solve pollution problems any more than it did in Roman times. Given weak government standards, enforcement, and cleanup, health threats continue, as from the former smelter in DePue, Illinois. A town of 2,000, DePue is majority Latino. One-third of its population is under age 18, and 64% of households are below average-Illinois-household income (U.S. Census, 2010). US-EPA (2013b) says it is one of the 10-worst-health hazards among U.S. Superfund sites. Its operations ceased after US-EPA (2013a) warned in 1980 of severe onsite and offsite contamination of its soils and lakes with antimony, arsenic, barium, cadmium, chromium, copper, cyanide, iron,

lead, mangangese, mercury, selenium, silver, and zinc, including 750,000 tons of zinc slag. Although site owners New Jersey Zinc-Exxon Mobil (ZEM) signed a 1995 legally-binding contract with US-EPA that forced them to do hazard assessments and cleanup, after 34 years they remediated only an onsite ditch, similar to clean-up failures at LaOrroya. Metals-contaminated water still flows into Lake DePue, all offsite- and residential-soil contamination remains (IEPA, 2011; US-EPA 2012, 2013a).

Residents face DePue health threats, despite the apparent absence of currently illegal polluter acts, partly because of weak pollution regulations, defended by flawed polluter science. On one hand, the US-EPA defends its smelter-heavy-metals regulations. It says they have "an adequate margin of safety," employ the "most stringent criteria," and protect "the most sensitive" persons, typically children, near former smelters (Falco, 2012b, pp. 1-2, 4-5).

On the other hand, US-EPA metals standards are less protective than scientists and other government agencies recommend. US-EPA offsite-residential-soil-lead regulations, for instance, require offsite-residential-soil-cleanup when lead exceed 400 parts per million (PPM), so that blood-lead levels do not exceed 10 micrograms/deciliter (ug/dL). Yet in 2012 US-CDC adopted twice-as-protective lead standards of 5 ug/dL, affirming that the tiniest dose of lead is unsafe, especially for children (Csavina et al., 2012; Godwin, 2009; Schmidt, 2010; Barbosa et al., 2005).

Similarly, in 2007 California EPA (CAL-EPA) adopted blood-lead standards of 1 μg/dL, 10 times stricter than US-EPA's 10 μg/dL, and offsite-residential-soil-lead limits of 80 μg/g (mg/kg or ppm), to avoid blood-lead increases exceeding 1 μg/dL and I.Q. losses exceeding one point (OEHHA, 2009; IWP, 2013). Other states have followed suit. Minnesota, for instance, uses 100 ppm soil-lead limits when children are present, not US-EPA's 400 ppm, claiming "it has been known for many years that any amount of lead in children can be harmful" (MDH-ATSDR, 2012, p. 2).

Moreover, economists say that because lead harms I.Q. and thus decreases future earnings, reducing US-EPA's soil-lead limits, from 400 to 100 ppm, would give future-earning benefits of $4,710–$12,624 to each affected child (Zahran et al., 2011). Because lead does more damage at lower doses, if government allowed blood-lead exposures of 1.5 and 2.4 μg/dL, respectively, close to California's 1μg/dL standards, not its 10 μg/dL limit, 1.5 standards would be 50 times more cost-effective than 10 μg/dL standards, and 2.4 standards would be 25 times more cost-effective (Pichery et al., 2011). Each dollar used for lead-hazard prevention thus will return $17–$221 in future earnings, depending on avoided-dose values (Gould, 2009).

If scientific consensus is correct that lead has no safe dose, then CAL-EPA's lead standards are correct to be 10-times-stricter than US-EPA's, and US-EPA's weaker standards are flawed. Yet even the stronger, CAL-EPA standards allow residential-soil-lead levels that can cause a one-point-I.Q. drop that, in

turn, causes about a $20,000 per-child decrease in lifetime wages (Rice and Hammitt, 2005). These I.Q. and wage data mean that the weaker, US-EPA lead standards allow even worse I.Q. and income harms. But if so, how can US-EPA claim that its metals and smelter standards prevent all "adverse" effects and protect "the most sensitive persons" (Falco, 2012b, pp. 1-2, 4-5)? At a minimum, it appears EPA's standards ought to be 10 times stricter, and as protective as those of the state of California.

SMELTER POLLUTERS' SCIENTIFIC EXCUSES

Another reason for continuing smelter threats is polluters' frequent failure to accept responsibility for citizens' dangerous blood-metals levels. Partly because quantifying the relative blood-metals-levels contributions from different-metals sources, such as smelters, is difficult (MDH-ATSDR, 2012, p. 11), smelter owners and their trade groups, such as Battery Council International, often deny responsibility for harms. They say inhaling resuspended soils contaminated by leaded gas or house paint contributes more to elevated blood-lead levels than smelters do; they claim government unfairly blames smelters when most lead threats occur through old contamination from leaded gas and paint (Schmidt, 2010, pp. A77–79). Thus, US-EPA officials, for instance, say that because of long-ago leaded-gas and leaded-paint contamination, government may not be able to impose stricter smelter standards (IWP, 2013).

In response to metals-industry claims, however, US-EPA officials say most contributions of paint, gas, and smelters to lead contamination can be distinguished because paint-caused, soil-lead contamination typically occurs only at house foundations, whereas leaded-gas-caused contamination typically occurs only near larger, older, busy intersections (IWP, 2013). Because government phased out dangerous leaded gas and paint, physicians say it should do the same for smelter pollutants. They also say that although automobiles, not smelters, overall have released more lead, the crucial issue is lead "hot spots" near smelters. Because smelters, not autos, always have caused most near-smelter, high-blood-lead levels (e.g., Landrigan et al., 1975; Van Geen et al., 2012), and because soil-metals levels decrease with distance from smelters, physicians say smelter-polluters' excuses fail (e.g., Diaz-Barriga et al., 1997; Soto-Jiménez and Flegal, 2011).

FLAWED SMELTER SCIENCE

Another reason for current and former-smelter-site threats is polluters' often using flawed science to minimize smelter harms and reduce their cleanup costs. In the United States, for instance, under Superfund or the Comprehensive Environmental Response, Compensation, and Liability Act

(CERCLA), US-EPA may identify parties responsible for hazards like smelters and compel remediation. However, given polluters' legal rights under CERCLA, often US-EPA scientists must (i) wait for polluters to do scientific assessments and clean-up proposals, and then (ii) spend time evaluating these assessments and proposals. Next, they must (iii) decide whether and how polluters must correct the scientific assessments and proposals, and then (iv) wait while polluters make corrections and submit revised assessments and proposal. Finally, US-EPA scientists must (v) check whether polluters' corrections are adequate, and then (vi) repeat steps (iii)–(v), again and again, before cleanup begins. Obviously, fairness requires steps (i)–(vi). Nevertheless, polluter delay, in performing these steps, means Superfund victims, including hundreds of DePue residents, must wait for justice. They have been living with contaminated homes and yards, and they have waited for 34 years with no residential cleanup.

To illustrate how flawed science delays smelter cleanup, consider two questionable claims being currently and successfully made by DePue Superfund polluters. Their causality-denial claim is that although some studies show increased, near-smelter MS, the DePue smelter did not cause it because "there is not yet consensus regarding the causative agents" of neurodegenerative diseases (Falco, 2013, pp. 3-4). Their biomagnification-denial claim is that despite many heavy-metal-DePue-smelter pollutants, residents are unlikely to be harmed through food-chain biomagnification because "mercury is the only metal of significant concern for biomagnification" (Falco, 2013, pp. 2). Later paragraphs show that both claims are flawed, that there appears to be scientific consensus that metals can cause neurodegenerative harms, and that food-chain biomagnification of heavy metals might harm residents near former smelter sites.

THE CAUSALITY-DENIAL CLAIM

Although DePue polluters claim lack of scientific consensus that heavy metals can cause neurodegenerative diseases, this section shows their causality-denial claim errs both scientifically and ethically. It errs scientifically because it ignores the fact that hundreds of epidemiological, mechanistic, and experimental studies show that metals can cause neurodegenerative and autoimmune diseases, and that scientists have found no threshold for damaging neurological effects of metals. It errs ethically because it misunderstands the epistemic role of scientific consensus and ignores EPA's professed duty to protect the most vulnerable. Consider first the background of the causality-denial claim, and then its ethical problems.

This background is that, on one hand, during 1971–1990, "a significant excess of MS cases" occurred in DePue," 10 times more cases than expected, and scientists documented residents' "significant exposure . . . to mitogenic

trace metals, including zinc" (Schiffer et al., 2001). On the other hand, DePue polluters reject these studies and deny the smelter caused increased MS, because they deny that heavy metals can cause neurodegenerative diseases (Falco, 2013, pp. 3-4).

In denying metals cause neurodegeneration, however, DePue polluters err. They err logically because alleged dissensus—about metals' causing neurodegenerative and autoimmune diseases—is not a sufficient condition for rejecting causality. Instead, dissensus can be the result of ignorance or flawed science orchestrated by parties with conflicts of interest, as with climate science. Analogously, consensus is not a sufficient condition for accepting causality claims because it can be the result of prejudice or bias. After all, for centuries, the consensus position apparently was that because of their different biological "natures," women, minorities, and other groups did not have enough ability to enable them to vote. Thus neither consensus nor dissensus are sufficient conditions, respectively, for accepting or rejecting any scientific claims. Instead, scientific claims are worthy of acceptance or rejection, respectively, because facts and logic support or falsify them, not because most people "think" they are correct because of consensus, or incorrect because of dissensus.

Later paragraphs show there is no dissensus about the metals-disease connection. However, if there were dissensus on this metals-disease connection, DePue polluters would be bound ethically to take precautions to avoid diseases. Why? In cases of uncertainty, EPA guarantees the "most stringent" protection of the "most sensitive" people (Falco, 2012a, pp. 2, 4). Such protection requires assuming that, given supposed dissensus about otherwise deadly contaminants, one should assume possible metals-disease causality. Otherwise, near-smelter residents could be seriously harmed, given no safe dose of many metals.

The causality-denial claim likewise errs scientifically in ignoring epidemiological, mechanistic, and experimental evidence that metals can cause neurodegenerative and autoimmune diseases. Scientists agree there is "no evidence of a threshold" below which heavy-metals like lead cause no "adverse consequences" on neurocognitive functioning (Csavina et al., 2012). Indeed, at least 13 of 14 different DePue-site-elevated metals/metalloids (arsenic, barium, cadmium, cobalt, copper, iron, lead, manganese, mercury, nickel, silver, sodium, vanadium, and zinc) are already epidemiologically implicated in neurodegenerative and autoimmune diseases. Epidemiologists have tied increased

- MS to increased exposure to 6 DePue metals/metalloids: arsenic (Tsai and Lee, 2013), barium (Valera et al., 2014), copper (Ghazavi, 2012), mercury (Attar et al., 2012; Suzuki et al., 2011), silver (Suzuki et al., 2011), and sodium (Maarouf et al., 2014; Paling et al., 2013).

- Amyotrophic Lateral Sclerosis (ALS) to increased exposure to 8 DePue metals: cadmium, cobalt, copper, iron, lead, manganese, vanadium, and zinc exposures (Roos et al., 2013; Hoizumi et al., 2011).
- Alzheimer's Disease (AD) to increased exposure to 3 DePue metals: copper (Hozumi et al., 2011; Squitti et al., 2013), lead (Gu et al., 2011), and sodium (Vivitsky et al., 2012).
- Parkinson's Disease (PD) to increased exposure to 3 DePue metals: copper (Hoizumi et al., 2011), manganese (Hoizumi et al., 2011; Wang et al., 1989) and zinc (Hoizumi et al., 2011).

DePue polluters also ignore evidence of pollution mechanisms for metals-induced neurological harm. Scientists confirm that at least two related sets of mechanisms can be responsible for this harm. One mechanism is metals-induced, chronic central nervous system (CNS) inflammation, characteristic of multiple sclerosis (MS), PD, and ALS, which causes free-radical-mediated oxidative damage, impaired mitochondrial function, and neuronal death (Choi et al., 2013; Sharma and Sharma, 2013; Urrutia et al., 2013; Verina et al., 2013; Roos et al., 2013; Hoizumi et al., 2011; Suzuki et al., 2011). A second mechanism is metals-induced brain increases in beta-amyloid-plaque accumulation, characteristic of AD (e.g., Choi et al., 2013; Sharma and Sharma, 2013; Squitti et al., 2013; Urrutia et al., 2013; Vivitsky et al., 2012; Suzuki et al., 2011; Hoizumi et al., 2011).

DePue polluters likewise ignore experimental evidence confirming metals-caused neurological harm. Many controlled animal studies demonstrate the same metals-induced inflammation, plaque buildup, and brain disease that occurs in humans. For example, young-adult, nonhuman primates, exposed to manganese, exhibit "diffuse Aβ plaques" at age 6–8, while unexposed monkeys express no plaques until (old) age 20. Scientists think heavy metals can accelerate AD pathology (Guilarte, 2010).

Thus, at best, DePue polluters' denial that metals can induce neurodegenerative disease ignores epidemiological, mechanistic, and experimental evidence for pollution harm. At worst, the denial is false.

THE BIOMAGNIFICATION-DENIAL CLAIM

Another prominent DePue-polluter scientific assertion, the biomagnification-denial claim, likewise errs. Later paragraphs show it ignores abundant scientific data showing that cadmium, copper, zinc, chromium, and barium each biomagnify in many food chains, and that studies denying metals-biomagnification are scientifically flawed in at least one of two ways. They generalize to hundreds of thousands of food chains on the basis of one or two

nonrepresentative food chains, and they average their data in ways that dilute or deny obvious biomagnification in many food chains.

Contrary to the DePue polluters biomagnification-denial claim, scientists have shown cadmium transfers from soil to primary producer (plant or photosynthetic microbe) to herbivore and up microbial food chains, and also increases in concentration as it ascends trophic levels (10,107 ug cadmium/g of biomass to 85,034 ug/g dry weight in microbe food chains). Consequently, cadmium can enter food chains at their most basal, microbial source and be transferred up to organisms eaten by humans. In fact, Lake-DePue-fish species such as bass pose serious cadmium-biomagnification problems when used as food, especially because Illinois Department of Natural Resources touts DePue as a "significant sanctuary with dependable food resources," many fish species, "rich histories of commercial hunting and fishing," and home to "the two oldest private duck [-hunting] clubs in the state (IDNR, 2014). Yet cadmium concentrations in bass, sunfish, bluegill, silverside, and goby—many of which are found in Lake DePue—can increase as much as 15 times within 2 trophic levels (Croteau et al., 2005).

Of course, scientists know that not all species exhibit cadmium biomagnification, including DePue's white-footed mice (Cardwell et al., 2013, Mann et al., 2011). However, not all species need exhibit biomagnification, for it to harm residents. Besides, residents do not eat mice, but they do eat Lake DePue fish, in many of which cadmium biomagnifies. Because long-lived homeotherms (e.g., mammals and birds) have high metabolisms that require large, constant diets over extended lifetimes, they cannot avoid taking up large amounts of metals (Mann et al., 2011). Therefore, one mice study cannot dismiss cadmium-biomagnification in fish (Mann et al., 2011), especially because poverty-level and minority citizens are more likely to fish for free food in local waters (US-FWS, 2004), and as noted, DePue's population is majority Latino with high poverty levels (U.S. Census, 2010).

Copper likewise presents a significant DePue-biomagnification concern because it can transfer from plant (plant shoot: 1.4 mg/kg fw) to insect (larvae 6.9 mg/kg fw) in increasing amounts (Zhuang et al., 2009). It also appears to biomagnify from aquatic plants to the common carp, with biomagnification factors 2.12–14.35, depending on the plant (David et al., 2012). Because Lake DePue has carp and is a well-known fishing area (IDNR, 2014), copper biomagnification also threatens local food.

Of course, some scientists object to copper-biomagnification claims at DePue. They might say that, although copper bioaccumulates, it will not affect humans at DePue because it does not biomagnify up the food chain to carnivores (Laskowski, 1991).

However, those who deny copper magnification, up to carnivores, err because they ignore the fact that several metals, including copper and lead, biomagnify in shrews and invertebrate predators (e.g., Mann et al., 2011).

In addition, studies that deny copper biomagnification (Laskowski, 1991) are methodologically flawed in at least three ways. First, Laskowski diluted possible biomagnification effects by using whole-body-metals measurements (ug metals/g dead-carnivore-specimen dry weight) to deny biomagnification, although carnivores' different body parts (liver, kidney, muscle, etc.) accumulate metals at different rates (Jakimska et al., 2011; Naccari et al., 2013). Second, Laskowski did no tissue- or organ-specific measurements of different body parts. Third, he did no meta-analysis of different sampling techniques, to determine whether whole-body measurements diluted biomagnification. Thus, all methodologically-reliable data confirm potential DePue-smelter-metals biomagnification that threatens humans (Liang et al., 2009; Rubio-Franchini and Rico-Martinez, 2011; Veltman et al., 2007).

Likewise, contrary to DePue polluters's biomagnification-denial claim, metals such as zinc biomagnify in plant to aphid to ladybird (Green et al., 2003). Metals such as chromium biomagnify in herbivorous to carnivorous fish (Jabeen et al., 2012). Metals such as barium biomagnify in soil to plant root to shoot to leaf (Kowalska et al., 2012). Because they biomagnify up food chains, they also could threaten DePue citizens.

Even if water, not diet, is sometimes a greater source of heavy-metals poisoning, local residents swim, boat, fish in Lake DePue, and carnivorous fish's metal-toxicity levels can strongly correlate with, or increase, metal-toxicity levels in herbivorous prey. For instance, from herbivore to carnivore, barium can rise from 0.19 ug/g to .28 ug/g. From herbivore to carnivore, chromium can rise from 3.1 ug/g to 5.07 ug/g. From herbivore to carnivore, zinc can rise from 51.64 ug/g to 80.08 ug/g. Thus diet is a major heavy-metals contaminant for some fish, and these metals can move up aquatic food chains (Jabeen et al., 2012). As a result, fish-eating DePue residents could be harmed by smelter-metals biomagnification, especially given intensive area fishing.

The preceding evidence for cadmium, copper, zinc, chromium, and barium biomagnification suggests that the DePue polluters's biomagnification-denial claim is false, at least for some trophic levels and for some metals. It also suggests that because polluters did inadequate DePue-site studies, they failed to find this evidence. At a minimum, the evidence shows that scientists don't know enough about DePue's trophic construction, component organisms, or *in-situ* metals concentrations to disconfirm site biomagnification, thus possible food threats to DePue residents.

Although some studies show copper, nickel, lead, and zinc do not biomagnify in a primary producer-macroinvertebrate-fish food web similar to DePue systems (Cardwell et al., 2013), using such studies to deny DePue biomagnification is scientifically erroneous for at least three reasons. First, because biomagnification is context-dependent, generalizations about one ecosystem mean little, given contaminant-uptake complexities in different ecosystems (Clements et al., 2012). Because biomagnification depends on

diet choice, habitat, salinity, temperature, physiological biodynamics, food-web structure, and trophic positions (Croteau et al., 2005; Vieira et al., 2011), scientists need ecosystem- and species-specific studies, before dismissing metals-biomagnification. Second, Cardwell et al. (2013, pp. 116, 118) would not make sweeping generalizations, denying all DePue biomagnification because they warn biomagnification studies "have not accounted for variability within species, over time, or between habitats." In fact, they explicitly caution against generalizing their results, and they note great variability in biomagnification "between the organisms found in different habitats, and years sampled."

A third reason to question this biomagnification-denial study is its flaws. Cardwell et al. (2013) admit that Cd, Cu, and Zn can biomagnify in specific food chains. They use Trophic Transfer Factors (TTF), where TTF levels >1 indicate biomagnification, to assess their data. Their own data show the TTF exceeds 1 for Cd in 3 species of fish, for Cu in 3 species of fish, and for Zn in 14 species of fish. Yet they dismiss this biomagnification data by diluting it and averaging it with data from other food chains in which these metals do not biomagnify. However, biomagnification remains a problem for those who eat DePue fish. The issue is not whether metals biomagnify in average or in all global food chains, but whether they biomagnify in any DePue food chains. Why? Poor, minority, local residents are likely at the top of several DePue food chains, especially because of fish from Lake DePue.

Thus, at worst, the metals-biomagnification denial is false. At best, it is doubtful because it ignores epidemiological, mechanistic, and experimental evidence for biomagnification in existing DePue fish and game species.

RESULTING HARMS, ESPECIALLY TO CHILDREN

One ethical and scientific consequence of De Pue polluters' ignoring epidemiological, mechanistic, and experimental evidence for metal' causing neurodegenerative disease and biomagnification is that DePue children are put at serious risks. As already noted, there is no evidence of a threshold for harm from metals/metalloids like lead, and no safe dose of particulate matter or PM (e.g., Pope et al., 2003, 2007; Krewski et al., 2009). Yet PM is a key form of smelter-metals exposure; and DePue is one-third children, majority Latino, with high poverty levels. Moreover, DePue poverty-level children are twice as likely to live within a mile of the former-smelter site as other local children, and nonwhites are seven times more likely to live within a mile (Scorecard, 2014). Even worse, DePue's poorer children and mothers face special smelters-metals-risk because those with poorer nutrition absorb more metals. For all these reasons, even the lowest soil-metals levels may cause metals-biomagnification and decreases in children's cognitive functions, "especially attention and memory" (Yorifugi et al., 2011).

Also, as already mentioned, some metals do more neurodegenerative damage at lower doses because they have supralinear dose-harm relationships. Thus, children with blood-lead levels below10 μg/dL have I.Q. losses double those of children with 10–20 μg/dL. They have I.Q. losses quadruple those of children with 20–30 μg/dL of blood lead (Pichery et al., 2011; see Landrigan, 2011).

Moreover, the youngest children are harmed most. Lead exposure during childhood-brain development, neonatal, and puberty periods can permanently damage brain structure (Cecil et al., 2008). Because exposure-timing helps determine gene expression (Schwartz et al., 2008), prenatal-lead exposure, for instance, affects expression of genes whose products cause brain plaque and protein deposition and later AD (Mazumdar et al., 2012). Thus DePue polluters denial that metals can biomagnify and induce neurodegenerative disease ignores epidemiological, mechanistic, and experimental evidence for pollution harm. It also puts children at greatest risk.

SUGGESTIONS AND CONCLUSIONS

Preceding remarks suggest that current and former smelters threaten health not only because of poor pollution standards and enforcement, but also because flawed, smelter-polluter science can reinforce these flawed standards and also delay cleanup. As illustrated at DePue, the article shows that smelter-polluters' science, perhaps designed to minimize site-cleanup costs, errs both in denying food-chain biomagnification of metals at the site, and in denying that metals can cause neurodegenerative disease. The biomagnification-denial claim relies on ignoring the facts that abundant scientific data showing that cadmium, copper, zinc, chromium, and barium each biomagnify in many food chains, and that studies denying biomagnification either invalidly generalize from one or two cases or dilute biomagnification data through invalid aggregation and averaging. The causality-denial claim errs because it ignores abundant experimental and observational evidence for metals-induced neurodegeneration, for precise mechanisms of metals-induced neurodegeneration, and for the apparent absence of a threshold for metals-induced brain risks. All these scientific problems with polluters' claims suggest that government should follow its own guarantees. It should require the "most stringent" protection of the "most sensitive" people, partly by ensuring that polluters use the best science to assess and remediate their damage (Falco, 2012a, pp. 2, 4).

The La Oroya situation likewise suggests that developed-nation companies, operating in the developing world, ought to follow more-stringent, home-country, public-health protections. As Roman-empire-smelter examples revealed, one reason is that smelter-metal pollution travels and thus harms globally, not just among those nearby. A second reason U.S. and

developed-nation companies should follow stricter regulations, especially in the developing world, is the relative powerlessness of developing nations to enforce pollution standards, given their need for industry and investment. Besides, stricter smelter standards are consistent with 2012 recommendations of the American Academy of Pediatrics (AAP) for global pesticide protection (AAP, 2012). AAP recommends banning U.S. export of pesticides that are banned in the United States. Arguably, government also should ban companies from using smelter practices abroad, when those practices are banned at home. Banning such smelter-practices, and policing polluter science, would not solve all smelter problems, but they are important first steps.

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