The correspondence section is a public forum and, as such, is not peer-reviewed. EHP *is not responsible for the accuracy, currency, or reliability of personal opinion expressed herein; it is the sole responsibility of the authors.* EHP *neither endorses nor disputes their published commentary.*

Overlooked in Fallon?

The statistical examination of the Fallon childhood cancer cluster by Steinmaus et al. (2004) provides renewed justification for opening a larger window onto the expanse of possibilities regarding a possible cause for this extraordinary cluster. Although the cause(s) might very well be the culmination of simultaneous or sequential exposure to an array of chemical stressors (perhaps in conjunction with nonchemical stressors) at the needed concentrations for sufficient time and during critical windows of vulnerability (as dictated by health and nutritional status, age, sex, genetic susceptibility, etc.), it is worth considering possible new, plausible singular causes until each is ruled out.

Significant resources have been devoted to investigating the childhood cancer cluster discovered in 2000 in Fallon, Nevada (Churchill County). Although the magnitude of this cluster of acute lymphoblastic leukemia (ALL) could be attributed to happenstance, the recent analysis by Steinmaus et al. (2004) shows that such a cluster would be expected to occur in the United States by chance less frequently than every 20,000 years.

A surprising, contemporaneous incidence of ALL also developed in Sierra Vista, Arizona (Cochise County Health Department 2004). Other clusters have occurred in several additional western U.S. rural communities, as well as in various locales worldwide. Of possible significance has been the simultaneous emergence in both Churchill and Cochise counties of an extremely rare form of childhood cancer, rhabdomyosarcoma.

None of the hypothesized causes of the Fallon cluster has withstood scrutiny (with the possible exception of an unknown infectious agent—the "population mixing" hypothesis or "Kinlen theory," although not supported by the examination of Steinmaus et al. 2004), including exposure to arsenic, tungsten, radiation, and jet fuel. Any hypothesis must account for the important fact that these clusters seem to be limited to a span of several years, after which the incidence subsides. Another commonality seems to be arid agricultural locales that experience periods of drought.

Surprisingly, despite the extensive resources and time devoted to searching for an environmental etiology, no consideration has been devoted to one potential cause that would account for many, if not all, of the aspects of these clusters. Pyrrolizidine alkaloids (PAs) comprise a complex galaxy of highly bioactive natural products. Riddelliine, senkirkine, monocrotaline, retronecine, heliotridine, jacoline, jacozine, jacobine, seneciphylline, and senecionine are but a few of the numerous PAs produced by a wide spectrum of plants. PA-producing plants (e.g., tansy ragwort, coltsfoot, hound's tongue), especially *Senecio* species, have long been problematic in the western United States and are well known for livestock poisonings.

Fluctuating levels of PA contamination in the consumer food supply, especially via certain herbal teas (e.g., comfrey), honey (Beales et al. 2004), dairy products, beef, and grains, are a function of drought and harvest or foraging conditions, and therefore exhibit aperiodic cycles of high expression. Churchill County happens to be the center of honey production in Nevada (Churchill Co. 2005; Michigan State University Extension 2002); honey is also produced in Cochise County. Honey has been a particular focus for PA contamination; levels can vary from hive to hive by two or more orders of magnitude within the same foraging location and by time of year. PA-producing plants are particularly prevalent in both counties, where they can contaminate the domestic food supply as weeds; some, such as comfrey, continue to be sold by certain vendors of nutritional supplements and health foods.

Sporadic acute exposures or long-term exposure to low levels (e.g., as little as 10 μg/day) of PAs can lead to delayed toxicity (Australia New Zealand Food Authority 2001; Molyneux et al. 1988) (up to 1 or more years after exposure) and could therefore escape causal suspicion or elude measurement. Levels of metabolites insufficient for overt toxicity in adults could be passed from mothers to fetuses and nursing infants. Maternal transfer would also exempt the liver as the major target for the well-documented toxicity for these chemicals. Furthermore, ALL can originate *in utero* (Jensen et al. 2004). Although best known for their hepatotoxicity (where the bioactive metabolites, such as the dehydropyrrolic products, lead to veno-occlusive diseases and cirrhosis), activated PAs can elicit significant genotoxicity and can be carcinogenic as well as anticarcinogenic (which has led to their experimental use in chemotherapy). Some PA adducts persist in tissues from which metabolites can be released, even long after initial exposure, and migrate to other tissues or can be transported to fetuses or nursing infants (Molyneux and James 1990). It is noteworthy that honey, milk, and grains are also common foods for infants.

Although carcinogenicity data are lacking in humans, PAs have been shown in rats to cause both leukemia (Chan et al. 2003) and rhabdomyosarcoma [California Environmental Protection Agency (EPA) 1999]. The Food and Drug Administration (FDA 2001), the National Toxicology Program (NTP 2003), the World Health Organization (WHO 1988), the California EPA (1999), and others have identified PAs as a major human health threat, especially for fetuses and infants. Significantly, a recent study (Jensen et al. 2004) points for the first time to a link between maternal diet and ALL, where consumption of carotenoids and glutathione (via vegetables) is proposed as being protective. Although linkages of cancer with diet often ascribe the cause to deficiencies or insufficiencies of essential or protective nutrients, just as likely would be the presence of particular chemical stressors—anthropogenic and natural toxicants alike. This finding of Jensen et al. (2004) fits nicely with the fact that glutathione conjugation in particular is known to be a major detoxification route for PAs. A coordinated investigation by epidemiologists, toxicologists, and environmental chemists of a PA–leukemia linkage could prove to be a prudent investment.

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References

Australia New Zealand Food Authority. 2001. Pyrrolizidine Alkaloids in Food: A Toxicological Review and Risk Assessment. Technical Report Series No. 2. Canberra, Australia:Australia New Zealand Food Authority. Available: http://www.foodstandards.gov.au/_srcfiles/TR2.pdf [accessed 2 March 2005].

Beales KA, Betteridge K, Colegate SM, Edgar JA. 2004. Solid-phase extraction and LC-MS analysis of pyrrolizidine alkaloids in honeys. J Agric Food Chem 52(21):6664–6672.

California EPA. 1999. Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65). Availability of Draft Data Summaries and Draft Priorities for Chemicals With Respect to Their Potential to Cause Cancer: Request for Relevant Information. Sacramento, CA:California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Available: http://www.oehha.ca.gov/prop65/pdf/batch3_8.pdf [accessed 1 March 2005].

Chan PC, Haseman JK, Prejean JD, Nyska A. 2003. Toxicity and carcinogenicity of riddelliine in rats and mice. Toxicol Lett 144(3):295-311.

Churchill Co. 2005. Churchill County, Nevada Homepage. Available: http://www.usgennet.org/usa/nv/state/alhn-churchill/alhn-churchill.htm [accessed 2 March 2005].

Cochise County Health Department. 2004. Leukemia Cluster Information for Cochise County. County of Chochise, Arizona:Cochise County Health Department. Available: http://www.co.cochise.az.us/health/HealthDepartment/Leukemia.htm [accessed 2 March 2005].

FDA. 2001. FDA Advises Dietary Supplement Manufacturers

to Remove Comfrey Products from the Market [letter to industry]. College Park, MD:Center for Food Safety and Applied Nutrition, Food and Drug Administration. Available: http://www.cfsan.fda.gov/~dms/dspltr06.html [accessed 1 March 2005].

Jensen CD, Block G, Buffler P, Ma X, Selvin S, Month S. 2004. Maternal dietary risk factors in childhood acute lymphoblastic leukemia (United States). Cancer Causes Control 15(6):559–570.

Michigan State University Extension. 2002. Agricultural Tourism in Cochise County, Arizona Characteristics and Economic Impacts. Tourism Educational Materials - 33839801. Available: http://www.msue.msu.edu/msue/imp/modtd/33839801.html [accessed 2 March 2005].

Molyneux RJ, James LF. 1990. Pyrrolizidine alkaloids in milk: thresholds of intoxication. Vet Hum Toxicol 32(suppl):94–103.

Molyneux RJ, Johnson AE, Stuart LD. 1988. Delayed manifestation of Senecio-induced pyrrolizidine alkaloidosis in cattle: case reports. Vet Hum Toxicol 30(3):201–205.

NTP. 2003. Toxicology and Carcinogenesis Studies of Riddelliine (CAS No.23246-96-0) in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies). Technical Report 508. Research Triangle Park, NC:National Toxicology Program.

Steinmaus C, Lu M, Todd RL, Smith AH. 2004. Probability estimates for the unique childhood leukemia cluster in Fallon, Nevada, and risks near other U.S. military aviation facilities. Environ Health Perspect 112:766–771.

WHO. 1988. Pyrrolizidine Alkaloids. Environmental Health Criteria No. 80. Geneva:World Health Organization. Available: http://www.inchem.org/documents/ehc/ehc/ehc080.htm [accessed 1 March 2005].

Editor's note: In accordance with journal policy, Steinmaus et al. were asked whether they wanted to respond to this letter, but they chose not to do so.

"Arsenic in Food": Opinion Parading as Science

I write in response to apparent serious errors associated with Ellen K. Silbergeld's letter in the May 2004 issue of *EHP* (Silbergeld 2004). As a toxicologist of nearly 30 years, a private consultant, and associate editor of the *International Journal of Toxicology*, I am concerned that although Silbergeld's assertions on the risk of arsenic residues in poultry are presented under the cloak of good science, they appear to be her personal opinions and not a scientific conclusion based on sound methodology and evidence. In her letter I found at least three significant deviations from sound scientific methodology. These included the multiple mischaracterization of results presented in other publications and the introduction of a serious mathematical error. I will discuss in detail only one of these, the mathematical error, which should suffice to demonstrate the lack of science supporting Silbergeld's opinion letter.

In one of the articles Silbergeld relied upon, "Mean Total Arsenic Concentrations in Chicken 1989–2000 and Estimated Exposures for Consumers of Chicken" by Lasky et al. (2004), the authors estimated that, based on the consumption of 60 g/day of chicken meat, an average individual may ingest 1.38–5.24 μg/day inorganic arsenic. However, in employing these numbers in her letter, Silbergeld stated the units erroneously and reported the results of Laskey et al. as 1.38–5.24 μg/kg/day inorganic arsenic. This single error inflated the alleged "exposure" rate by 7,000%, a significant miscalculation. In fact, this error, by itself, completely negates Silbergeld's opinion that inorganic arsenic exposure through the consumption of chicken "would be a significant addition to drinking water exposure."

This misquoting of Lasky et al.'s (2004) results is but one of Silbergeld's significant mistakes in her letter. The result of each error of this type is either an inflation of the calculated exposure or a buttressing of Silbergeld's stated opinion.

As a long-time author, reviewer, and editor of scientific papers, I am aware of the difficulty in ensuring the detailed accuracy of manuscripts, particularly where letters are concerned. There is a historical, although incorrect, perception that letters deserve less review than full manuscripts. At the same time some individuals, knowing that letters are not peer-reviewed to the same degree as scientific articles, make use of letters to get into print content that would otherwise not be acceptable. Although this may not have been the objective of Silbergeld's letter, her scientifically unsupported opinion was repeated in the *Baltimore Sun* (O'Brien 2004) and other media (e.g., *Consumer Reports* 2005) as though it were scientifically proven fact. The result was unnecessary public alarm based on unsupported personal opinions.

Peer-review is meant to identify and weed out mistakes of this type. Ethical journals either require authors to correct errors before publication or decline to publish the article if the author refuses to make the warranted changes. Peer-review is not only the job of the publishing journal, but also the institution where the author resides (in this case, Johns Hopkins University). At many institutions, anything intended for publication must withstand internal review by an institutional committee before it can be sent to a potential publisher. For some reason, neither institutional nor editorial review detected these misquoted results and mathematical errors, a number of which appear to be obvious and would have been easily detected had the letter been checked. Both the journal and institution may wish to review their current procedures and make adjustments so they are not similarly embarrassed in the future.

As professionals, health scientists must be cognizant that respectability and trust are fragile commodities. We all know too well of a number of professions in our society that have lost significant amounts of respect and trust (e.g., politicians, lawyers, clergy) because of the misuse of the trust placed in them. Rational thought, balanced and unbiased evaluation, and honest reporting, as exemplified by the scientific method, are the primary underpinnings of the trust with which the nonscientific community honors us. Anything that causes loss of that trust, whether sloppy work or biased, self-serving presentations that distort the true state of scientific knowledge, demeans us all.

The author is a consultant for Alpharma, the manufacturer of roxarsone, an organic arsenic-containing drug approved by the Food and Drug Administration (FDA) for use in chicken and pigs. He has also, in the past, worked for the FDA, the regulator of this compound. The author's other clients include the National Academy of Science, the National Institutes of Health, the Department of Defense, and numerous private companies.

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References

Consumer Reports. 2005. You are what they eat. Consumer Reports 70(1):26–32.

Lasky T, Sun W, Kadry A, Hoffman MK. 2004. Mean total arsenic concentrations in chicken 1989–2000 and estimated exposures for consumers of chicken. Environ Health Perspect 112:18–21.

O'Brien D. 2004. Arsenic used in chicken feed may pose threat: Hopkins study explores risk to consumers, water. Baltimore Sun (Baltimore, MD) 4 May: 1B.

Silbergeld E. 2004. Arsenic in food. Environ Health Perspect 112:A338–A339.

Editor's note: Lasky et al. were given the opportunity to respond to Silbergeld's letter [Environ Health Perspect 112:A338–A339 (2004)], but they declined; we omitted that fact when we published Silbergeld's letter.

"Arsenic in Food": Silbergeld Responds

Bernard comments on the calculations in my letter (Silbergeld 2004) to criticize my conclusions, which were that the use of arsenic for growth promotion in poultry feeds results in contamination of chicken products (and other food animal products because of the use of poultry litter in feeds), and that the estimates of risk have not been adequately calculated, even by Lasky et al. (2004) because of unsubstantiated inferences of the arsenic concentrations in edible tissues and a puzzling use of outdated risk assessments for arsenic. I find it interesting that Bernard (who has consulted for the Food and Drug Administration, the agency that permits this nontherapeutic use of arsenicals in animal feeds) does not comment on these conclusions in his letter.

I acknowledge the error in quoting Lasky et al. (2004); I used the wrong metric in quoting her conclusions. Please do not ascribe responsibility to my colleagues, who

read this letter in manuscript form, or to *EHP*'s editorial office. However, I do not agree that this mistake invalidates the conclusions of my letter. If the concentrations of arsenic in edible chicken meat are not one-tenth of those in liver (as claimed by Alpharma, the manufacturer of roxarsone), then the exposure of Americans who consume chicken (such as my son, who appeared to exist largely on chicken wings during high school) is in fact 3–10 times higher than Lasky et al. estimated, resulting in an intake of 4–50 μg/day. This is still in excess of the current National Research Council (NRC) recommendation (NRC 2001).

This risk estimate does not include the potential for additional exposures to arsenic from confined animal feeding operation (CAFO) wastes via land disposal, which may reach human populations though soil contact, groundwater contamination, and plant uptake, as noted in my letter (Silbergeld 2004). These exposures may be important for regions such as the Eastern Shore, where between 600 and 800 million broiler chickens are raised each year. The U.S. Geological Survey has estimated that thousands of kilograms of arsenic may be land disposed with poultry wastes (Garbarino et al. 2003).

Given the article by Lasky et al. (2004) and new information on the environmental pathways of arsenic releases from CAFOs (Han et al. 2004; Jackson et al. 2003), as well as new studies on the health effects of arsenicals (Simeonova and Luster 2004), I suggest that it is time for a thoughtful consideration of the use of arsenicals as growth promoters in animal feeds.

The author declares she has no competing financial interests.

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References

Garbarino JR, Bednar AJ, Rutherford DW, Beyer RS, Weshaw RL. 2003. Environmental fate of roxarsone in poultry litter. I. Degradation of roxarsone during composting. Environ Sci Technol 15(8):1509–1514.

Han FX, Kingery WL, Selim HM, Gerard PD, Cox MS, Oldham JL. 2004. Arsenic solubility and distribution in poultry waste and long-term amended soil. Sci Total Environ 320:51–61.

Jackson BP, Bertsch PM, Cabrera ML, Camberato JJ, Seaman JC, Wood CW. 2003. Trace element speciation in poultry litter. J Environ Qual 32:535–540.

Lasky T, Sun W, Kadry A, Hoffman MK. 2004. Mean total arsenic concentrations in chicken 1989–2000 and estimated exposures for consumers of chicken. Environ Health Perspect 112:18–21.

Silbergeld EK. 2004. Arsenic in food. Environ Health Perspect 112:A338–A339.

Simeonova PP, Luster ML. 2004. Arsenic and atherosclerosis. Toxicol Appl Pharmacol 198:444–449.

"Sex and Ceruloplasmin Modulate the Response to Copper ..."

It is past time for *EHP* to stop accepting papers whose funding disclosure makes it obvious there is a conflict, yet "The authors declare they have no competing financial interests." Méndez et al. (2004) stated that "This investigation was funded by the International Copper Association [ICA] in the form of an unrestricted research grant." I fail to see that an unrestricted grant eliminates a conflict of interest. From the article's introduction, it is obvious that this research team has done a lot of copper toxicity work with this money from the ICA, but the conflict of interest would exist even if only the work reported in this article was funded by the ICA.

It is interesting, however, that the ICA is funding human experimentation. The pesticide industry's push to allow human experimentation in the toxicity tests to register their pesticides in the United States is driven by their resulting ability to drop the 10-fold interspecies safety factor in allowable exposure levels. Lockwood (2004) found all six human pesticide studies reviewed rife with financial conflict of interests. Think of the risks created to all toxicity testing when the most reputable general toxicology journal in the world, *EHP*, endorses human subjects for toxicity testing in very risky situations.

Many of us tolerate animal testing because we hope that eventually the current massive risk of toxic agents will be acknowledged.

It may seem that this work on Cu entailed little risk, as the authors claim in the opening of their discussion:

> … Liver aminotranferases were evaluated to satisfy ethical considerations. We detected no responses that may represent toxic effects of the Cu dose used.

First, the authors acknowledged that there are large data gaps on the toxicity of Cu at many doses. Critically, this demonstrable truth makes their statement about detecting no toxic responses false. Obviously they were not looking at many toxic end points—especially chronic effects. Also, they stopped looking for any effects after a very short period (82 subjects ingested 10 mg/kg/day Cu for 2 months).

The stated tolerable daily intake (TDI) in the introduction (Méndez et al. 2004) is unclear (and unattributed), but it appears to range from 0.9 to 10 mg/kg/day. The experimental dose chosen for this study was 10 mg/kg/day, and was justified by the authors as being a dose safe for 97.5% of humans. TDIs are typically derived from industry junk science (unpublishable in independent journals) and contain massive data gaps. However, even if we assume the claimed TDI is validated, the authors are admiting that their chosen experimental dose was above the "safe" level for about two of their subjects.

At the end of the discussion the authors admit that the Cu-induced enzyme changes they looked at are altered by hepatic diseases; Méndez et al. (2004) then state that their results can be used to monitor adverse liver effects.

The effect of the combination of risky dosing with acknowledged toxicity data gaps is stunning. In summary, I am disappointed that *EHP*'s manuscript reviewers and editors allowed such dangerous (unethical) statements and objective inconsistencies; I fear that a human toxicity experiment—in *EHP* of all places—has created a terrible precedent; and I am frustrated that authors and *EHP* continue to misstate obvious conflicts of interest. I look forward to discussion and solutions.

The author declares he has no competing financial interests.

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Lockwood A. Human testing of pesticides: ethical and scientific considerations. Am J Public Health 94:1908–1915.

Méndez MA, Araya M, Olivares M, Pizarro F, González M. 2004. Sex and ceruloplasmin modulate the response to copper exposure in healthy individuals. Environ Health Perspect 112:1654–1657.

Ceruloplasmin and Copper: Méndez and Araya Respond

Most unfortunately, Tweedale misunderstood and misinterpreted our article "'Sex and Ceruloplasmin Modulate the Response to Copper Exposure in Healthy Individuals" (Mendéz et al. 2004). The article is about copper homeostasis and homeostatic regulation, not about toxic effects associated with copper. The dose administered was 10 mg/day and not 10 mg/kg/day, which we agree may be toxic. The dosage and the time of study used allowed us to assess homeostatic mechanisms in normal human beings; this study will contribute to the development of specific recommendations for subgroups of the population that have genetic polymorphisms that render them more susceptible to minor copper deficiency and copper excess.

Aminotranferases are the current gold standard to assess liver damage; therefore, it is

correct that although no adverse effect was expected because the dose is safe, ethical considerations made it pertinent to measure their activities. These enzymatic activities are routinely measured for diagnostic purposes in individuals who manifest symptoms of illnesses. It is not known whether they reflect minor changes in hepatic function when enzymatic values are within the normal limits and there is no illness. Thus, it was most interesting to assess the potential of these enzymes to detect changes within the normal range in the studied individuals.

Certainly, our work is based on a series of concepts that include the upper safe limit (which represents the safe chronic average intake of the metal for human beings), tolerable daily intake (TDI), and several others [e.g., dietary allowances, adequate recommended oral intake (AROI), lower concentration of observed effects (LOEL)] that served to ensure that the protocol was within safe limits for human adults. Although approximately 2–2.5% of the normal population is not included in some of the concepts mentioned, this does not mean that this percentage will be damaged by the exposure but that they represent individuals with illnesses, and therefore are not included among the normal population, who require a different treatment. All these concepts, of course, are not "typically derived from industry junk science (unpublishable in independent journals)" and they do not "contain massive data gaps," as stated by Tweedale, but they do represent the state of the art on a specific topic produced by experts appointed by the National Institutes of Health, the World Health Organization, and other respected agencies.

Our study (Mendéz et al. 2004) indeed followed the ethical considerations required to work with human subjects. The protocol was approved by our institutional review board, which is registered (IRB00001493) with the Office for Human Research Protections.

Last and most important, how to deal with potential conflicts of interest in science is a hot topic, and there is currently no final solution (Blumenthal 2003; Morin et al. 2002; Nathan and Wilson 2003; Tufts University 2003). We made full disclosure of our financial support, and it is up to the readers to judge the situation for themselves.

The authors declare they have no competing financial interests.

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References

Blumenthal D. 2003. Academic-industrial relationships in the life sciences. N Eng J Med 349:2452–2459.

Méndez MA, Araya M, Olivares M, Pizarro F, Gonzalez M. 2004. Sex and ceruloplasmin modulate the response to copper exposure in healthy individuals. Environ Health Perspect 112:1654–1657.

Morin K, Rakatanska H, Riddick FA Jr, Morse LJ, O'Bannon JM III, Goldrich MS, et al. 2002. Managing conflicts of interest in the conduct of clinical trials. JAMA 287:78–84.

Nathan DG, Wilson JD. 2003. Clinical research and the NIH-A report card. N Engl J Med 349:1860–1865.

Tufts University. 2003. Conflict of Interest Policy. Available: http://techtransfer.tufts.edu/tufts/pol_guide/conflict.html [accessed 1 March 2005].

Errata

Lai et al. would like to clarify their financial support for their article "Understanding the Spatial Clustering of Severe Acute Respiratory Syndrome (SARS) in Hong Kong" [Environ Health Perspect 112:1550–1556 (2004)]. The acknowledgment should have stated that "The research was supported by the Research Fund for the Control of Infectious Diseases of the Health, Welfare and Food Bureau of the Hong Kong SAR Government."

The January Beyond the Bench article "Building Blocks of Learning" [Environ Health Perspect 113:A33 (2005)] incorrectly listed Kathleen Vandiver's affiliation as Cambridge Public Schools. Vandiver is actually with Lexington Public Schools.

The March Science Selections article "Fewer Frogs in Illinois: Organochlorines May Be to Blame" [Environ Health Perspect 113:A182 (2005)] listed incorrect percentages of intersex frogs collected during the periods studied. In fact, intersex frogs accounted for 1.2% of samples from 1852 to 1929, 7.5% of samples from 1930 to 1945, 11.1% of samples from 1946 to 1959, 6.3% of samples from 1960 to 1979, and 2.7% of samples from 1980 to 2001.

EHP regrets the errors.