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### OP Pesticides, Organic Diets, and Children's Health

The importance of "judicious use of language in regard to public communication of pesticide health risks" (Lu et al. 2006b) is clearly recognized and acknowledged in recent letters from Avery (2006) and Lu et al. (2006b). Their correspondence concerned perceptions of risk conveyed by the article "Organic Diets Significantly Lower Children's Dietary Exposure to Organophosphorous Pesticides," published by Lu et al. (2006a). My concern is more fundamental than the need for effective communication and the stated "public misunderstanding of this important issue" (Lu et al. 2006b). I believe the primary issue concerns science and how we accumulate knowledge.

There is no guarantee that judicious use of language can prevent misunderstanding of even the most rigorous and carefully performed studies. It is important, however, to put the results into the existing scientific and regulatory contexts. Lu et al. (2006a) noted that "the paucity of exposure data renders the debate over pesticide-related health risks in children controversial." Curl et al. (2003) stated that "reduction of children's risk from pesticides requires an understanding of the pathways by which exposure occurs." The primary objective of the longitudinal study by Lu et al. (2006a) was determination of "overall pesticide exposure in a group of elementary school-age children." The authors reported that children who consumed organic diets eliminated (via urine) nondetectable amounts of organophosphorous (OP) insecticide metabolites. The finding supports the consensus that the diet is the predominant source of OP compounds and OP metabolites excreted in urine (Barr et al. 2004; Duggan et al. 2003; Krieger et al. 2003).

Lu et al. (2006a) claimed "a convincing demonstration of the ability of organic diets to reduce children's OP pesticide exposure and the health risks that may be associated with these exposures." When the study was developed and throughout the period of data collection, analysis, and publication by the University of Washington investigators, there could be no doubt that dietary exposures were very low or miniscule relative to acute toxicity (Curl et al. 2003). Indeed, it is intuitive that the change in diet reduced OP metabolite elimination in urine. If this were not the case, one might expect parked cars to get speeding tickets.

Specific health risks have never been associated with such miniscule insecticide exposures. If risk is defined as the likelihood of an adverse effect in an exposed population, the risk of neurotoxicity caused by these dietary OP exposure(s) is zero; that is, disease has not been observed in the population who consumes food that sometimes contains OP pesticides or OP metabolite residues (Krieger et al. 2003). Back-calculated OP exposures are well below the experimental lowest observed adverse effect level (LOAEL), the estimated no observed adverse effect level (NOAEL), and the regulatory reference dose (RfD) for neurotoxicity of any OP insecticide used in crop protection (Barr et al. 2004; Duggan et al. 2003; Fenske et al. 2000). The research is misrepresented with respect to its relevance to risk reduction (that is the point of the fundamental "observed" in the LOAEL and the NOAEL upon which RfDs are based).

With zero cases of disease in the population exposed to dietary OP pesticide, the numerator of measurements of risk such as odds ratios or relative risk is also zero. As a result, measured risk of acute neurotoxicity is zero. The axiomatic truth that "dose determines a poison" and its corollary that "there is a safe level of everything" must both be considered in responsible risk communication. Careful choice of words may sometimes prevent misunderstanding of health research reports, but more importantly our common understanding and well-being require that we clearly distinguish chemical exposure and health risk. Lu et al. (2006a) wrote,

We were able to demonstrate that an organic diet provides a dramatic and immediate protective effect against exposure to organophosphorus pesticides that are commonly used in agricultural production.

Their findings are expected rather than dramatic, and the term "protective" in reference to a no observed effect exposure is misleading at best. Effective communication requires awareness that potential impacts of conjecture about matters of health and pesticides likely include heightened anxiety and fear, and may prompt misallocation of resources as some persons pursue something less than zero risk—a point where scientific evidence and mystical, supernatural beliefs must be distinguished.

*The authors declare they have no competing financial interests.*

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### OP Pesticides, Organic Diets, and Children's Health: Lu et al. Respond

Krieger et al. criticize the misrepresentation of our recent paper (Lu et al. 2006) with respect to the relevance to health risk reduction of dietary organophosphorus (OP) pesticide exposure in children. They argue that current OP exposures, measured in the form of urinary metabolites in children, are well below the "safe" level and therefore pose "zero" risk.

The basis for Krieger et al.'s extraordinary statement is the claim that "specific health risks" have never been associated with dietary pesticide exposures, and that "zero cases of disease" have occurred that can be attributed to such exposures. However, Krieger et al. must be aware of the tragic misapplication of the carbamate insecticide aldicarb to watermelons in California in 1986, resulting in 6 deaths, 17 hospitalizations, and > 1,000 probable or possible poisoning cases (Centers for Disease Control and Prevention 1986). The probability of such an event occurring again is certainly greater than zero. In fact, such an event was reported recently in Taiwan for an OP

pesticide found in vegetables (Wu et al. 2001). Krieger et al. also ignore the fact that some pesticides are categorized as carcinogens and that dietary exposures to these compounds carry some risk. For example, the fungicide chlorothalonil is classified by the State of California as a carcinogen [Office of Environmental Health Hazard Assessment (OEHHA) 2006], and the U.S. Environmental Protection Agency (EPA) estimated that the cancer risk from dietary exposure to chlorothalonil is  $1.2 \times 10^{-6}$  (U.S. EPA 1999). Although one might agree with the U.S. EPA that this is a *de minimus* risk, the risk cannot be characterized as “zero.”

Krieger et al. appear to dismiss the possibility that pesticides can produce non-acute adverse health effects, but recent studies have shown an association between adverse neurologic and growth outcomes in children exposed to OP pesticides *in utero* (Jacobson and Jacobson 2006; Whyatt et al. 2005; Young et al. 2005). To our knowledge, no epidemiologic studies of children’s dietary OP pesticide exposures and adverse health effects have ever been conducted. To quote our current Secretary of Defense, Donald Rumsfeld, “Absence of evidence is not necessarily the evidence of absence” (Rumsfeld 2003). A final judgment of the potential for OP pesticide exposure to cause adverse developmental or neurologic health effects in children will require rigorous epidemiologic studies that include sound exposure assessment.

Risk is a probabilistic concept and is generally considered to be dependent on exposure and toxicity. If exposure is reduced, then the corresponding risk is reduced. We believe that the jury is still out on the risk, particularly on the chronic neurologic health risk in young children. In our article (Lu et al. 2006) we raised the hypothesis that by reducing children’s dietary exposure to OP pesticides, the risk of the associated health effects may be reduced. We look forward to future scientific evidence sufficient to either accept or reject this hypothesis. If our article has heightened unnecessary anxiety and fear among the public, this was not our intent. However, the perception of risk in the world of public health depends on individual attitudes and beliefs. Krieger et al. have misinterpreted our conclusion (Lu et al. 2006) as much as they have misunderstood the enforcement of the speeding limit, which is obviously not to issue citations to parked cars, but rather to minimize the possibilities of automobile accidents. The relevance of health risk reduction of dietary OP exposure in children is analogous to many public health campaigns in this county, such as the use of seat belts, smoking cessation, and HIV (human immunodeficiency virus) prevention,

which are not adopted to penalize or inconvenience individuals, but are intended for public health protection.

*The authors declare they have no competing financial interests.*

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### Prolactin Changes as a Consequence of Chemical Exposure

We read with great interest the article by de Burbure et al. (2006) on health effects in children who live near nonferrous smelters in France, the Czech Republic, and Poland. We were especially interested in the inverse relationship found between levels of urinary mercury and serum prolactin. We found a similar result in an Italian multicenter

cross-sectional survey with adult subjects (Alessio et al. 2002) using a different statistical approach based on regression analysis with mixed linear models. We found that serum prolactin decreased as a function of both urinary mercury and occupational exposure to inorganic mercury (Lucchini et al. 2003). In another study (Carta et al. 2003), our group observed the opposite behavior of prolactin in adult individuals with a high dietary intake of mercury-contaminated tuna. In that study, serum prolactin was positively associated with urinary and blood mercury. Our interpretation of this dual behavior was that prolactin may be differently affected by inorganic and organic mercury based on the interference with different neurotransmitters implicated in the regulation of prolactin secretion (Carta et al. 2003).

The article by de Burbure et al. (2006) stimulates further consideration of the observed effects on serum prolactin after exposure to various metals and other chemical substances. In fact, prolactin can be increased by exposure to lead (Govoni et al. 1987; Lucchini et al. 2000), organic mercury (Carta et al. 2003), and manganese (Ellingsen et al. 2003; Smargiassi and Mutti 1999; Takser et al. 2004), but it can be decreased by exposure to inorganic mercury (de Burbure et al. 2006; Lucchini et al. 2003; Ramalingam et al. 2003), aluminum (Alessio et al. 1989), and cadmium (Calderoni et al. 2005; de Burbure et al. 2006). Subjects exposed to chemicals such as styrene (Bergamaschi et al. 1996; Luderer et al. 2004; Umamura et al. 2005), perchloroethylene (Beliles 2002; Ferroni 1992), and anesthetic gases (Lucchini et al. 1996; Marana et al. 2003) have shown an increase of serum prolactin, whereas polychlorinated biphenyls (De Krey et al. 1994) and the pesticide luthinate [U.S. Environmental Protection Agency (EPA) 2002] are known to decrease serum prolactin.

Possible mechanisms, other than direct effects at the cellular level, may be related to different neurotransmitters involved in the modulation of prolactin secretion. For example, the dopaminergic and serotonergic systems, respectively, are involved in the physiologic regulation of this hormone as a tonic inhibitor and as an excitatory modulator. Different chemicals may interfere with these two systems, resulting in different outcomes regarding serum prolactin. Recent studies have shown that the same chemical may even cause different effects on prolactin depending on the exposure doses (Lafuente et al. 2003).

We would like to know why this neuroendocrine hormone is affected differently by exposure to different chemicals. This is important because of the possible use of

prolactin, as described by de Burbure et al. (2006), as a sensitive indicator of early effects in toxicologic research and risk assessment (Mutti and Smargiassi 1998). Negative studies have also been published on the association of prolactin with the exposure to neurotoxicants (Myers et al. 2003; Roels et al. 1992). Therefore, it is vital to assess the causes of the variability that may limit the reproducibility of these tests. Further research should focus on multiple exposure to different chemicals, which may help to explain the lack of association.

*The authors declare they have no competing financial interests.*

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## Prolactin Changes as a Consequence of Chemical Exposure: de Burbure and Bernard Respond

We appreciate the letter from Alessio and Lucchini concerning the number and variety of toxicants able to affect serum prolactin levels. Reflecting on the wide variability of the currently available data, we would like to make two additional points.

The first point concerns the usefulness of serum prolactin as a potential indicator of neurotoxicity for populations at risk. This biomarker indeed appears to be influenced by a large number of both organic and inorganic chemicals, which have seemingly little in common in terms of mechanistic action (e.g., heavy metals, pesticides, styrene, polychlorinated biphenyls). Moreover, one chemical—cadmium, for example—can have a biphasic dose-dependent effect on serum prolactin (Lafuente et al. 2003), an effect we did not observe in our study (de Burbure et al. 2006) because of low exposure levels; this dose-dependent effect is reminiscent of the biphasic effects of lead on glutamate neurotransmission shown to be dependent on glycine receptor affinity (Marchioro et al. 1996).

As proposed by Alessio and Lucchini in their letter, these data reflect the complexity of the control of prolactin secretion, which is modulated not only by dopamine but also

by several other neurotransmitters. These neurotransmitters include serotonin,  $\gamma$ -aminobutyric acid (GABA) [as demonstrated by the hyperprolactinemia developed by GABA<sub>B1</sub> knock-out mice (Catalano et al. 2005)], glycine, and glutamate (Fitsanakis and Aschner, 2005; Nagy et al. 2005). In view of these neurotransmitters, serum prolactin—albeit sensitive—appears to be a rather non-specific biomarker for monitoring populations at risk; therefore, serum prolactin will likely remain a predominantly useful tool in the field of research until the multiple facets of controlling prolactin secretion are unveiled.

Another important issue to keep in mind concerns the biological significance of all of the modifications we observed in our study (de Burbure et al. 2006). Despite their statistical significance, are the observed small changes in serum prolactin at all clinically relevant? To what extent do the variations in serum prolactin induced by the various neurotoxicants correlate with changes in brain function? Because prolactin has a large number of potential determinants, probably with different mechanisms of action, it is a rather delicate intellectual exercise to give a correct interpretation of the observed changes in terms of the possible development of neurotoxicity.

Although the lack of specificity of prolactin reduces the immediate usefulness of these dopaminergic biomarkers, the question of the potential clinical impact of the small but significant changes in terms of neurotoxicity (de Burbure et al. 2006) certainly remains an important question that further research will have to address.

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