DDT Risk Assessments

Two recent articles in *EHP* (1,2) and the latest Agency for Toxic Substances and Disease Registry toxicologic profile for DDT (3) make repeated references to DDT risks. These statements of risk, like so many others, are one-sided and give no consideration to colossal increases in diseases previously controlled with DDT. Behind disease statistics are grievous human tragedies, as with the case of a little girl who died of an infection that could have been prevented if her house had been sprayed with DDT. She lived in a village in the Andes and was 8 years old in 1998 when she died of bartonellosis. Bartonellosis was previously controlled through malaria house-spray programs, but without DDT, the disease returned.

One-sided and narrowly focused risk assessments form the bedrock of anti-DDT advocacy (4,5), but advocacy for global elimination of DDT through United Nations Environment Programme (UNEP) treaty negotiations failed (6). Countries can continue using DDT for disease control, and DDT is not listed for global elimination. This outcome was possible only through efforts of hundreds of scientists on behalf of hundreds of millions of people at risk of illness and death from malaria (7).

Environmental activists who still want DDT eliminated and who are surprised by the lack of cost-effective alternatives should understand that global vilification of DDT eliminated almost all research on public health insecticides. Lack of research support persists and contrasts sharply with the richness of funds for research on adverse health effects of DDT; 29 major projects are presently funded by the National Institutes of Health (NIAID) at the National Institute of Environmental Health Sciences, National Cancer Institute, National Institute of General Medical Sciences, and the National Institute of Child Health and Human Development (3).

The evidence of DDT efficacy in controlling diseases is irrefutable. In just 3 years, house spraying in Guaya reduced maternal and infant mortalities by 56% and 39%, respectively, and reduced malaria cases by 99% (8). Similar evidence from other geographic areas persuaded delegates to UNEP treaty negotiations that DDT is still needed. Yet, and in spite of all contrary evidence, the U.N. program to phase out DDT is unabated (9,10). The current “phase-out” program by the World Health Organization’s Roll Back Malaria initiative and the Global Environment Facility (Washington, DC) includes no publicized disease control performance standards and does not include appropriate on-site studies or tests to determine, under varying epidemiologic and environmental conditions, that DDT alternatives will provide adequate and sustained protection of rural populations. After years of successful efforts, the modus operandi of DDT elimination remains the same: apply political and economic pressures, convince country politicians that DDT is not needed, pass laws banning its use, and let impoverished rural populations quietly suffer spiraling increases in disease rates (11,12). Even short-term commitments of funds for purchasing the more expensive and less effective DDT alternatives are a continuation of past practices: in the end, disease rates will increase.

The Andean girl’s death is one of millions of preventable deaths that occurred as national and international regulations, trade barriers, international policies, and UN resolutions were applied to stop public health uses of DDT (13). With absolute certainty, the best measures of success in the anti-DDT campaign are increases in disease and death from malaria, leishmaniasis, bartonellosis, dengue fever, and dengue hemorrhagic fever. We can add to this list the renewed threat that urban yellow fever will once again ravage populations of the Americas. Even this emerging threat is linked to past failures to continue appropriate public health uses of DDT. The Andean girl’s unrecognized but precious stake in the DDT issue was her life, now lost. How many millions more must die because of hypothesized risks from minute quantities of DDT sprayed on internal house walls?

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associated with DDT exposure. South Africa’s experience underscores the importance of the flexibility provided by the POPs treaty.

Brazil and India offer important lessons about limits to DDT’s effectiveness. During the late 1980s and early 1990s, malaria rates in Brazil went up even as spraying of houses with DDT increased, but dropped after Brazil shifted strategies (10). With assistance from the World Bank, India is reducing its reliance on DDT. The main rural malaria vector (responsible for 65% of India’s malaria) is resistant to DDT (11). Indian researchers found elevated levels of DDT in buffalo milk, soil, water, and human blood where DDT had been sprayed to control malaria (12,13).

The ATSDR’s 2000 update of its toxicologic profile for DDT/DDE (14) reflects major concerns raised by the WWF and other environmental and public health groups during the POPs negotiations. In contrast to the previous profile published in the early 1990s, the update contains a large section, “Health Effects in Wildlife.” In the early 1990s, the update contains a large section, “Health Effects in Wildlife.” In the update, “Health Effects in Wildlife,” reminding readers that animals are sentinels for health effects in humans. A new section captioned “Children’s Susceptibility” reiterates a central message from the U.S. National Academy of Sciences’ landmark 1993 report on pesticides in the diets of infants and children (15): children are not little adults, but may be uniquely susceptible and exposed to pesticides.

The data in the toxicologic profile support the logic of the POPs treaty: DDT can be valuable for controlling malaria, but it is prudent to reduce human exposures. Recent studies on humans, too late to be included in the toxicologic profile, further support such caution. For example, Longnecker et al. (16) found that DDE concentrations in mothers are associated with increased risk of pre-term delivery and lowered birth weight.

Roberts takes EHP’s contributors to task for their “one-sided” references to DDT’s risks and their failures to account for DDT’s benefits. Roberts’ encomium to DDT is itself one-sided. Why expose humans to hazards from DDT when less risky strategies might be employed? The POPs treaty encourages development of alternatives and provides a new funding mechanism to support malaria control.

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Mercury and Autistic Gut Disease

We are challenged to consider the possible role of environmental toxins in autism and other childhood behavioral disorders (1), and creative research in this area surely is warranted (2). Perhaps particular scrutiny should be given to mercury and autism. M any signs and symptoms of mercury exposure correspond to autism (3), and pink disease (acrodynia) from inorganic mercurial teething powders and autism bear strong behavioral resemblance.

Gut disease with inflammation is becoming increasingly evident in autism. Enterocolitis and lymphonodular hyperplasia are found in nearly 90% of regressed autistic children (4). Widespread inflammatory changes with poor intestinal digestive enzyme activity (5), abnormal intestinal permeability (6), and malabsorption (7) have been reported in various autistic subgroups. It would be logical to consider toxins known to cause gut injury when we look for causes of autism.

Inorganic mercurial compounds are notorious for gut injury in humans. In animals, chronic low-nomolar exposure injures intestinal mucosa (8) and 30-min micromolar exposure injures the colon (9). Also, deposits of antibody in the intestine have resulted from chronic exposure to inorganic mercury (10).

Although systemic passage may be poor, inorganic mercury enjoyment can be by the small and large intestines (11). Organic and vapor forms are known to transit membranes quickly and distribute throughout the body, but their excretion is primarily renal and significantly inorganic, which may affect intestinal residence.

Biliary mercury excretion, predominant in adults, is not achieved in suckling animals and may not exist in infants (12). Ligation of the bile duct of adult animals results in retarded movement of systemic mercury to the feces, emphasizing an excretory role for the intestine (13). Poor biliary excretion in infants might be expected to increase intestinal exposure to mercury. In suckling animals, two-thirds of total ingested inorganic mercury is recoverable after 6 days from gut tissue, particularly the ileum (14).

Worrisome levels of inorganic mercury exist in domestic water supplies (15) and in industrial emissions and municipal sludge widely used as fertilizer on crops (16). Up to 40% of mercury emissions from hydrocarbon combustion and 60% from incinerators is in the inorganic form (17), and mercurial “fall-out” may exceed 1 ppm in soil (18). Individual inorganic mercury ingestion can vary widely and may be greater than expected (19).

Some specifics about autism should heighten interest in mercury. A long clinical tradition has evolved in the use of vitamin B12, and its activating enzyme (B12-kinase) is totally inhibited in the intestine at nanomolar concentrations (20). Organic forms of mercury such as methyl mercury from fish and ethyl mercury as a vaccine preservative (thimerosal) may also inflict gut injury. Methyl mercury in primates produces histologic abnormalities of one intestinal cell line: Paneth cells are enlarged and packed with secretory granules (21), also specifically reported in autistic children (5).

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In “Examination of the Melatonin Hypothesis in Women Exposed at Night to EMF or Bright Light” by Graham et al. (Environ Health Perspect 109:501-507 (2001)), the keys in Figures 4 and 5 are incorrect. The corrected figures are shown below. EHP regrets the error.

Figure 4. Mean (± SE) melatonin levels are plotted from 2100 hr to 0700 hr for eight women in the follicular menstrual phase (days 3-8) initially exposed for 4 hr to bright (5,200 lx) light or to dim (25 lx) light (study 3). Similar data are shown for eight women in the luteal phase (days 18-23) exposed to the same bright and dim light condi- tions. Bright light reduced the total amount of melatonin secreted (p < 0.0001) and delayed peak blood concentrations by 4 hr.

Figure 5. Mean (± SE) estradiol levels are plotted from 2100 hr to 0700 hr for the luteal group (n = 8) initially exposed for 4 hr to bright (5,200 lx) light or to dim (25 lx) light (study 3). Similar data are shown for eight women in the luteal phase (days 18-23) exposed to the same bright and dim light condi- tions. Unlike the profound changes observed in melatonin (Figure 4), no alterations in point-by- point matching measures of estradiol were found in either phase of the menstrual cycle.