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RE: Position of the Non-Governmental Organizations on Lindane, to advise the U.S. Government position at the NARAP meeting, September, 2004

The United States is working with Canada and Mexico to develop a North American Regional Action Plan (NARAP) on the chemical lindane and will be participating in a meeting of the Lindane Task Force to draft the NARAP document in Montreal in September, 2004. At that meeting, we urge the U.S. to call for the rapid elimination of lindane uses. Our position is detailed below, and supported by a short review of available scientific literature. Our position has been presented to the U.S. EPA numerous times, in writing and orally. We urge the U.S. EPA to take seriously the scientific data presented here and in our earlier comments, and take the necessary steps outlined in the letter to protect human health and the environment.

Position Statement: ***The undersigned organizations call for the rapid elimination of pharmaceutical, veterinary, and agricultural uses of the pesticide lindane throughout North America.***

A phaseout of lindane is fully justified given the well-documented health and environmental effects of this toxic, persistent and bioaccumulative pesticide, and the pervasiveness of its waste and breakdown products in the environment and in people. Safer, affordable and often more effective alternatives are available and in use for all applications of lindane. Further research and training promoting the adoption of these alternatives should be supported, giving top priority to preventative and least-toxic alternatives. We also call for immediate education programs about the risks of lindane, emphasizing the protection of exposed populations of children, Indigenous peoples, and workers. Further, we call on the governments of Canada, Mexico and United States to actively support the expeditious inclusion of lindane among new substances added to the Stockholm (POPs) Convention for elimination.

Lindane, also known as gamma-hexachlorocyclohexane (γ-HCH), is a persistent organic pollutant in the organochlorine pesticide class. Most organochlorine pesticides have been banned due to their toxicity, environmental persistence, and tendency to bioaccumulate. Several European countries have already banned all uses of lindane, including Denmark, Germany, The Netherlands and Sweden. Lindane has also been banned for all uses in Finland, Japan, Korea, New Zealand (since 1990), Norway (since 1991) and Turkey

(since 1978). It is also banned in more than two dozen developing countries, including Brazil, Chile, Colombia, Costa Rica, Ecuador, Egypt, Indonesia and Mozambique.[1]

Given the known persistence of lindane in the environment, its bioaccumulative properties, and its documented acute and chronic health effects, continued use of this pesticide represents a serious threat to public health.

Legal Obligations: ***U.S. EPA has not considered all uses of lindane in its risk assessment, as required by law***

In evaluating lindane under the Federal Food Drug and Cosmetic Act (FFDCA), EPA is obligated to include exposure through pharmacological uses in its risk assessments. EPA is required by law to consider "available information concerning the *aggregate* exposure levels of consumers (and major identifiable subgroups of consumers) to the pesticide chemical residue and to other related substances, including dietary exposure under the tolerance and all other tolerances in effect for the pesticide chemical residue, *and exposure from other non-occupational sources*." 21 U.S.C. § 346a(b)(2)(D)(vi) (emphasis added). The law does <u>not</u> say that EPA only need include exposure pathways that are within its regulatory jurisdiction, and EPA has not published any analysis or justification that could support its refusal to consider pharmacological uses. EPA is obligated to address lice and scabies treatments under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) as well. Under FIFRA sections 3 and 4, EPA may not register or reregister a pesticide unless the chemical will perform its intended function without causing any "unreasonable adverse effects on the environment," including human health. 7 U.S.C. §§ 136a(c)(5)(C), 136a-1(g)(2)(C). EPA therefore cannot legally reregister lindane for continued use, or approve any lindane tolerances on treated foods, based on faulty risk assessments that fail to consider the main exposure pathway for children to lindane.

Overview of Toxicity: ***Lindane is highly toxic, persistent, and bioaccumulative, and has neurotoxic and hormone disruption activity***

Lindane is highly persistent in a soil, with a half-life of more than a year, and has been found to contaminate the Great Lakes, and in groundwater in New Jersey, California, Mississippi, and South Carolina. Lindane residues have been reported in a variety of fatty foods such as hamburger, frankfurters, fish, bologna, peanuts, butter, cookies, and candy bars.[2] Residues in fatty foods are due to the fact that this chemical tends to accumulate and concentrate in fat.

Lindane is a known neurotoxicant in humans, and it has been reported to cause seizures and other adverse effects in children treated for head lice. Other effects reported in humans following exposure include tremors, memory impairment, irritability, and aggression.[3] To our knowledge, studies looking at potential long-term impacts on children exposed to lindane have not been well investigated. In animal studies, lindane is

known to suppress levels of dopamine and norepinephrine, critical neurotransmitters.[4] At lower doses, lindane causes abnormal behavior patterns, abnormal brain wave patterns on electroencephalogram (EEG), hyper- or hypoactivity, interference with learning, temperature dysregulation, and anxiety.[5]

Lindane is a known endocrine disruptor in animals, and is associated with a range of serious effects on reproduction and development. These effects include testicular damage, reduced sperm production, disrupted estrus (menstrual) cycles, delayed puberty in females, ovarian and uterine atrophy, and infertility.[6] Adult male rats treated with lindane develop atrophy of their sex accessory organs, including the epididymis, seminal vesicles, and vas deferens, consistent with treatment with an anti-androgen.[7] The same investigators also identified decreases in testicular weight and degeneration of the Leydig cells, resulting in diminished testosterone levels in adult male rats dosed with lindane.[8]

Lindane is a weak estrogen, a more potent anti-estrogen and anti-androgen, and may also interfere with thyroid and adrenal gland function. Ewes fed lindane have significantly decreased thyroid hormone (thyroxine) and pituitary hormone (LH) concentrations and significantly increased insulin and estrogen levels.[9] In adult female mice, administration of lindane results in atrophy of the adrenal glands and abnormalities of the gland structure. The mice also have increased cholesterol levels and decreases in ascorbic acid (Vitamin C) content of the glands.[10]

Low, environmentally relevant, doses of lindane inhibit the binding and production of androgens in the prostate, even at the tiniest dose tested. The inhibition does not appear to occur via direct binding to the androgen receptor. These investigators reported a synergistic interaction between malathion and lindane resulting in inhibition of testosterone metabolism in the rat prostate.[11]

One European study of women with reproductive problems identified elevated levels of lindane in the blood of women with infertility and menstrual dysfunction. Exposure to lindane was from off-gassing of treated wood used in home construction. Removal of the exposure resulted in normalization of menstruation. Although this study does not conclusively prove a link between lindane and reproductive abnormalities in humans, the results are consistent with animal studies in numerous species.[12]

Atmospheric Transport: ***Lindane is transported in significant quantities through the atmosphere, and is detected as a contaminant across North American***

Multiple lines of evidence conclude that lindane is transported in significant quantities through the atmosphere. Lindane was present in measurable amounts in the air above field planted with lindane-treated canola seeds; an estimated 12-30% of lindane applied to seeds is volatilized. Extrapolating from known amounts of canola seeds used in the Canadian prairies, this could result in approximately 60-180 tonnes of lindane contaminating the atmosphere.[13] (Waite et al, 2001).

Dr. Bidleman of the Meterological Service of Canada describes a recent (2000-01) large-scale North American survey of atmospheric concentrations of alpha- and gamma-HCHs deployed passive air samplers (PAS) for one year at 40 stations along transects from the Canadian Arctic, down the east coasts of Canada and the U.S., along the Canada - U.S. border and in southern Mexico and Central America.[14] "Concentrations of alpha-HCH in air were fairly uniform across the east-west transect along the border of the two countries, somewhat higher along east coast of Canada and lower in Mexico - Central America. The elevated levels in eastern Canada were explained by outgasing of alpha-HCH from cold arctic water that flows south, warms, and releases the alpha-HCH back to the atmosphere." [15]

In a letter to U.S. EPA, Dr. Bidleman of the Meterological Service of Canada points out his recent work demonstrating "concentrations of alpha-HCH in arctic air have declined by over an order of magnitude from 1979 to the late 1990s.[16] The levels in arctic air show close correlations with the global usage of technical HCH[17] and with estimated global emissions."[18] Dr. Bidleman describes the changes in lindane air concentrations in two discrete steps; the first drop occurring around 1983-84 and coinciding with the ban of technical HCH production in China, and a second (smaller) drop around 1990-91, co-incident with the elimination of agriculture uses of lindane in India and the former Soviet Union. "This is a dramatic demonstration of how large-scale emission controls on a fairly volatile chemical can reduce its atmospheric concentrations in remote regions".[19]

Breast Milk: ***Lindane is detected in breast milk, and therefore contaminates the primary food source of infants during critical periods of development***

Evidence of lindane in breastmilk is plentiful. While precise figures on the number of breastfeeding infants are not available, it is safe to say that breastmilk is the primary food source for hundreds of thousands of infants in Mexico, United States, and Canada at any given time. Lindane is found in breastmilk around the world,[20] and EPA's own analysis notes that lindane is transmitted "efficiently" through breastmilk, and that nursing offspring are exposed to this contamination "during critical periods of post-natal development" (*Lindane: Environmental Fate and Ecological Risk Assessment, p. 12*). Lindane breaks down more slowly in temperate climate zones than in the tropics.[21] Thus, levels of lindane in the environment, and in breast milk, are likely to be relatively higher in cool climates. The concentration of lindane in breast milk is also strongly related to diet. A German study found that women who followed a low-fat diet had lower beta-HCH levels in their breast milk than women whose diet included large quantities of meat.[22] A study of women whose diets consisted primarily of fish, found particularly high levels of lindane isomers in their breast milk.[23] Levels of lindane in breast milk in Germany remained over 5 ng/g lipid in the most recent data reported.[24]

Conclusion: ***The undersigned advocate the rapid elimination of lindane uses throughout North America, to protect human health and the environment***

The undersigned organizations advocate a rapid elimination of pharmaceutical, veterinary, and agricultural uses of the pesticide lindane throughout North America. As outlined in this letter, a phaseout is fully justified given the well-documented health and environmental effects of this toxic, persistent and bioaccumulative pesticide, and the availability of safer, affordable and often more effective alternatives.

Respectfully,

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[1] Combined sources include: UN Department for Policy Coordination and Sustainable Development 1994, IRPTC/NEP database 1995, Pesticide Information Update No. 22, PAN UK, August 16, 2000 and the UN web site for the Rotterdam Convention/Prior Informed Consent procedure: http://www.pic.int, viewed October 2001.

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