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LINDANE - A CONTAMINANT OF GLOBAL CONCERN

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ABSTRACT

LLindane belongs to the organochlorine class of pesticides that have been banned in most of the developed countries in the 1970s. There are basically five stable isomers (actually eight isomers) of lindane – á, â, ã, ä and å. Only ã isomer has insecticidal properties and due to its persistence in the environment (presence of recalcitrant chlorine groups), it is considered an ecologically toxic substance. Its presence in the environment is due to its extensive use as insecticide for control of a broad spectrum of phytophagous and soil-inhabiting insects, public-health pests, and animal ectoparasites and also on a wide range of crops and in seed treatments. There is a possibility of availability of lindane (due to its semi-volatile nature) into the three major environmental compartments- air, water and soil. Various mechanisms responsible for the transport are hydrolysis, diffusion, volatilisation, sorption, biodegradation (slow), bioaccumulation and photo-oxidation. Bioavailability of lindane is dependent on many factors like pH of the medium, K_{ow}, vapour pressure, temperature, solubility in water, residence time or half life. Lindane acts as a convulsant agent causing both acute and chronic neurotoxic, hepatotoxic and uterotoxic effects. It might also act as an endocrine disrupter and has also been declared as a potential teratogen, mutagen and carcinogen. The current ecotoxicological and human toxi-

cological intervention values for lindane are 2.0 and 21.1 mg/kg dry matter soil, respectively. This paper reviews the ecotoxicology of lindane comprising sources in the environment, processes that determine its fate and impacts on biological systems.

INTRODUCTION

The use of pesticides started to increase dramatically in the early fifties due to the demand for efficient food production which was dictated by the strong worldwide increase in population (Eichers *et al*. 1970). Due to their effectiveness, pesticides were highly commended but, soon after, the negative side-effects of pesticides attracted more and more attention. Eventually, it also became clear that the "first-generation" organochlorine pesticides (OCPs) – were bioaccumulating in the food chain and causing severe damage to e.g. several bird populations (Moriarty and Walker, 1987; Belfroid *et al*. 1995). In the sixties and seventies the focus was shifted to consumer protection because of the awareness that residues in food could cause chronic toxic effects. Subsequently, attention shifted to the negative effects on other organisms such as mammals, birds, fish and insects. In the eighties and nineties, attention was focussed on the 'second-generation' P-, N- and S-based pesticides. They are, admittedly, less persistent than the older OCPs but they are produced and used on a very large scale. This uncontrolled use has led to various environmental and health problems.

Thus, lindane is a persistent organochlorine compound (ist produced by Faraday in 1825) which is widely distributed in the environment. Long distance transport of lindane is evidenced by its presence in the Arctic, where it has never been used. Most of the lindane present in the environment is in water, although a significant amount is also found in the soil/sediment and some in air. Lindane has also been shown to bioaccumulate in the fatty tissue of organisms.

This review probes the interrelations between lindane in the environment and biota, as well as the processes responsible for its fate in the environment. The review actually examines the lindane effects at molecular, cellular and organism level, at different trophic levels from communities to the populations in the ecosystem.

Sources

Lindane is an organochlorine insecticide and fumigant which has been used on a wide range of soil-dwelling and plant-eating insects. Hexachlorocyclohexane (HCH), also known as benzene hexachloride (BHC), is an organochlorine insecticide that is available in two formulations - technical grade HCH and lindane. Technical grade HCH is a mixture of different isomers: a-HCH (60-70%), b-HCH (5-12%), g-HCH (10-15%), d-HCH (6-10%), and e-HCH (3-4%). Lindane is the g-isomer (> 99% pure) of HCH (ATSDR, 1997). The relatively high volatility (vapour pressure = 9.4×10^{-6} mm Hg at 20°C) of lindane has led to global transport, even into formerly pristine locations such as the

Arctic referred to as grasshopper effect as presented in Figure 1. As it has an atmospheric residence time of > 2 days, it is present as a vapour over different environmental compartments, for instance due to the spray on corn seedling in Texas, it was detected over St. Lawrence river valley (Environment Canada, 1998). It has been detected in air, surface water, groundwater, sediment, soil, fish and other aquatic organisms, wildlife, food, and humans. The most probable route of lindane exposure in humans is oral ingestion of food containing the insecticide. It may be released to the air during its formulation or use as an insecticide, from wind erosion of contaminated soil, or from release from hazardous waste sites. It has been detected in groundwater and surface water samples collected near hazardous waste sites; however, the chemical has only very rarely been detected in drinking water supplies. Lindane has been listed as an EPA priority pollutant due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment (Sinkkonen and Paasivirta, 2000) as also presented in Table 1.

Lindane is widely used as an insecticide and as a therapeutic scabicide, pediculicide, and ectoparasiticide for humans and animals (Budavari et al., 1989). As an insecticide, it is used on fruit and vegetable crops including greenhouse vegetables and tobacco, for seed treatment, in forestry and for animal treatment. Registered uses also include domestic outdoor and indoor uses by homeowners such as dog dips, house sprays, and shelf paper; commercial food or feed storage areas and containers, farm animal premises, wood or wooden structures, and military use on human skin and clothing (ASTDR, 1998; U.S. EPA,1998).

Lindane has been recently listed as a persistent organic pollutant (POP) under the United Nations' Economic Commission for Europe Convention on Long-range Transboundary Air Pollution (LRTAP POPs Protocol) and the Great Lakes Binational Toxics Strategy between United States and Canada. It is also the subject of a joint reevaluation in the US and Canada under NAFTA's (North American Free Trade Agreement) Technical Working Group on Pesticides. Canada intended to phase out the remaining use of lindane seed treatments by 2004 and it has been phased out completely as of date (www.agri-canada.gc.ca). In the US, the final re-registration decision was scheduled for July 2002 (www.cec.org, updated information on March, 2002). Environment Canada data show use of 455 tones of lindane in 1997 and 510 tons in 1998. More than 99% of this use was in three Prairie Provinces (Manitoba, Saskatchewan and Alberta) (WWF Canada Study, 1999; Li *et al*. 2003; Waite *et al*. 2005). In North America, under North American Free Trade Agreement, the US, Canada and Mexico are finalizing a North America Regional Action Plan for lindane. Mexico has already committed to phase out all uses of lindane by the end of 2005 and Canada has phased out all agricultural uses in 2004. But there has been no such progress in the U.S., where regulators are facing strong pressure from U.S. seed treatment companies to maintain current lindane uses. However, lindane is still being used in Canada in pharmaceutical products.

While the use of the lindane is debated in the west and international forums, the continued production of lindane in a developing country like India, with lax environmental laws and almost non-existent enforcement of environmental

protection and occupational safety regulations, still continues. The technical HCH continues to be used in Asia, mainly India for cotton protection and malaria control (CACAR, 1997) and this could serve as a potential potent source of lindane in the environment. Recent studies in India have shown alarming concentrations of lindane in Bay of Bengal and groundwater of the state of Andhra Pradesh (Rajendran *et al*. 2005; Shukla *et al*. 2006) and even diet samples in the agriculturally prosperous state of Punjab (Battu et al., 2005). In the public health sector, there was a proposed move by the Ministry of Health to use lindane to control malaria vectors a few years back, which got shelved due to commercial barriers relating to procurement and costs. Presently there is no policy for phasing out lindane in India. In fact, license for manufacturing lindane is easily available with the concerned government agency. India manufactures lindane for in-house as well as exports.

The regulatory status of lindane in different countries is presented in Table 2. The regulatory status throws light on the grim situation of lindane use in various countries (controlled and uncontrolled) as also reported in a recent study in Spain where high levels of HCH isomers were found in soils, leachates and river water, higher in most cases than the limit values established by the legislation (local, national and European, Concha-Grana *et al*. 2006). This inadvertent use will create a pollution problem in other countries as lindane and its by-products will be transported across the globe by different environmental processes (discussed later).

The international community could take years to review and debate whether HCH/lindane qualifies for a global phase out under the Stockholm Convention. Meanwhile, gross violations and criminal negligence by dirty production facilities in developing countries continues. This is reason enough for the international community to move quickly to phase out this dangerous organochlorine. A global ban of lindane is long overdue.

Toxic Effects

It has both acute and chronic toxic effects. Acute exposure mainly effects the central nervous system . Human volunteers ingesting a dose of 17 mg/kg have experienced severe toxic symptoms and a lethal dose to an adult is estimated to be 0.7 to 1,4 g((Brooks1990). The International Agency for Research on Cancer(IARC) has concluded that lindane is a possible human carcinogen. According to various estimates the for a 60 kg adult the daily maximum dose should not exceed 0.06 mg/kg

Key health issues related to lindane are Aplastic anaemia, congenital abnormalities, breast cancer etc.

Environmental transport processes
General

Lindane is found in various compartments of the environment, with most in water, and the rest in soil, sediment and air as also presented in the schematic in Figure 2. The most contaminated areas are locations where lindane is formulated, used or disposed of. When present in soil, it can leach to groundwater, sorb to soil particulates, or volatilize to the atmosphere. In general, the leaching

of organic chemicals through soil is governed by the water solubility of the chemicals and their propensity to bind to soil as presented in Table 3. It also depends on patterns of use, soil texture, and total organic carbon content of the soil, pesticide persistence, and depth to the water table (Pitt *et al*. 1999).

Persistent organic pollutants (POPs) are characterized by their stability and resistance to degradation processes in the environment, and their tendency to partition in fats and to accumulate in food chains. However they possess a range of physico-chemical properties that will lead to a partitioning between the gas and particle phases in the atmosphere, and between the air compartment and the surface compartments of soil, vegetation and pore water. This provides them environmental mobility (Jones, 1998). Lindane exhibits all these properties and gets proportionally partitioned in the environment.

Like other POPs, lindane can be transported over long distances through the atmosphere. It vaporizes and condenses, touching down on oceans and freshwater bodies, where it begins the cycle again. This is known as the "grasshopper effect." POPs tend to accumulate in colder climates such as the Arctic, where they are trapped by low evaporation rates.

Soil and sediments

Based on the results of a number of laboratory soil column leaching studies that used soils of both high and low organic carbon content as well as municipal refuse, lindane is generally immobile in soils (US EPA, 1992; Landrum *et al*. 1985). Adsorption of lindane to soil particulates (octanol/water partition coefficient – log K_{oc} is 2.38 -3.52) is generally a more important partitioning process than leaching to groundwater. However, the presence of groundwater sediments, which have low organic carbon content, are not sufficient to adsorb lindane so that groundwater contamination is prevented (Fliednar, 1997). Lindane which is adsorbed to sediments may be recycled to the atmosphere as gas bubbles formed in the sediment by the methanogenesis and denitrification processes of bacteria. It is estimated that 85% of the lindane associated with the sediment gas bubbles will be released to the atmosphere with the remaining 15% being dissolved in the water column as the bubble rises toward the surface (Breivik *et al*. 1999). The partitioning behaviour of lindane into various environmental compartments is principally governed by various parameters - pH, K_{ow}, vapour pressure, solubility in water, temperature, residence time or half life.

In soil, lindane is adsorbed to the soil particles, volatilized to the atmosphere, taken up by crop plants or leached into groundwater. In soils and sediments, lindane is degraded primarily by biotransformation, however the major removal mechanism from soil is volatilization. Decomposition and dispersion rates in the soil depend upon many factors, including pH, temperature, light, humidity, air movement, compound volatility, soil type, persistence/half-life and microbiological activity (Pitt *et al*. 1999).

High temperatures and flooding are considered the key elements in increasing the volatilization rate of lindane from soil surfaces (Bintein and Devillers, 1996; ADSTR, 1998). It has a half life of 107 days under uncropped conditions in soil. Temperature, humidity and solar radiation have been found to be responsible for the rapid dissipation of HCH isomers from Indian sub-tropical

soils (Samuel and Pillai, 1990). Generally, lindane is concentrated more in the upper layer of soils as compared to lower layers (Bintein and Devillers, 1996).

Air

In the environment, lindane is potentially transformed into a variety of chemicals, most of which are volatile. These include ã-pentachlorocyclohex-1-ene, ã -3,4,5,6-tetrachlorocyclohex-1-ene, á-HCH, â-HCH, and ä-HCH (Bintein and Devillers, 1996; Cornacoff *et al*. 1988). The ratio of á -HCH to ã -HCH concentration in air has been used as an indicator to estimate the possible origin of the air mass in the long-range transportation of contaminants (Iwata *et al.* 1993). Earlier, bioisomerization of ã -HCH to á -HCH was thought to be the principal route for long range contamination, however, current field studies have found that only a small percentage of ã -HCH is converted to á -HCH as a result of biological activities (Waliszewski, 1993; Singh *et al.* 1991).

Other explanations for the higher global presence of á -HCH and higher á / ã ratios in some places could be attributed a) to their variable physical-chemical properties, for example the Henry's law constant for á –HCH and ã -HCH are 0.524 Pa m^3/mol and 0.257 Pa m^3/mol respectively at 20°C indicating water solubility of lindane and its tendency to partition faster from a gas phase into the water phase. Therefore, during global atmospheric transportation of HCH isomers, lindane will be more easily removed from the air by rain, leaving proportionally higher levels of á -HCH in the air (Walker *et al*. 1999; Cosley *et al*. 1998).

The atmospheric life-time of lindane based on hydroxyl radical reactions using a rate constant model is 96 days (Brubaker and Hites, 1998). Levels of lindane in the atmosphere are seasonal and temperature dependent. For example, yearly concentrations were found to vary in US and Canadian cities, with highest air concentrations in the summer and lowest in the winter, as would be expected from agricultural use (Whitmore *et al*. 1994).

Water

Water is one of the sinks of lindane. Three major transport pathways for atmospheric inputs to surface waters are wet deposition, dry deposition and gas exchange across the air-water interface. Despite its high vapour pressure, evaporative loss of lindane from surface water is not considered significant. It depends on water temperature and occurs only during the warmest months of the year. Biodegradation in aquatic systems is considered the most dominant process in the removal mechanism from water. The estimated degradation half-lives in rivers, lakes and groundwater are 3-30 days, 30-300 days and >300 days, respectively (Padma and Dikchut, 2001). Adsorption and desorption mechanisms predominate in the sediment systems; this could eventually lead to recycling of lindane back to the water bodies by microbiological activity (Fendinger *et al*. 1992). The half-life in sediments has been estimated at 90 days (Bintein and Devillers, 1996).

Bioaccumulation

Vegetation

Plants are exposed to lindane during direct application and from the air and water (EXTOXNET). Possible routes of entry into plants are: a) partitioning from contaminated soil to the roots and from there to other parts, b) through the atmosphere by gas-phase and particle phase deposition onto the leaf surface and c) by direct uptake through the stomata. Lindane is concentrated (log K_{ow} = 3.20-3.89) more in plants with high lipid content, for instance, carrots (Singh *et al*. 1991).

Thus, lindane does happen to enter the plants which form an important diet of the animal kingdom leading to catastrophic effects on health and life.

Animals

Lindane residues have also been found in liver, fat, blood, brain and muscle tissue of exposed rats (DeJongh and Blaauboer, 1997). The mean concentration was over 800 ppb wet weight in the liver, while in the brain, kidney and testis, levels were less than 400, 700 and 400 ppb, respectively. Lindane is stored in various tissues and then excreted over time. Initial concentrations in the liver and adipose tissue of rats on the day after exposure were 8.64 and 437 ppb, respectively, and down to 0.56 and 11 ppb by the seventh day. Levels in whole blood and plasma ranged from 1.5 and 2.21 on the first day to 0.22 to 0.09 ppm, respectively, on the seventh day (Junqueira *et al.* 1997). Similarly, lindane residues decreased over time following administration in rabbits. For instance, rabbits given 4.21 ppm bw/day lindane orally were found to have fat tissue residues of lindane of 38.51-61.85 ppm in 28 days, with lower levels of lindane (12.31-21.52 ppm fat) in rabbits sacrificed seven days following the last dosing (Ceron *et al*. 1995). Lindane residues have also been found in the eggs of water birds on the Danube River delta, with increasing concentrations found in birds higher on the food chain (Walker and Livingstone, 1992).

Despite the accumulation in the lower animals and plants, no reported evidence of lindane bioamplification was reported.

Humans

Almost all human exposure to lindane is from dietary intake (>99 per cent), with the rest coming from drinking water, from dermal contact with contaminated soil, and some inhalation of contaminated water (Ragas and Huijbregts, 1998; Bintein and Devillers, 1996). The Acceptable Daily Intake (ADI) stipulated by WHO is 1ppb bw/day (FAO, 1998) and oral reference dose (RfD) of 0.3 ppb bw/day is set by US EPA (US EPA, 1988). A study of adult dietary intake of table-ready foods in the US in 1990 estimated the mean intake of lindane at 0.2 mg/day, with a maximum of up to 3.2 mg/day (MacIntosh *et al*. 1996). The mean uptake is much lower than the RfD of US EPA. Lindane and HCH residues have also been found in maternal serum, the placenta, the umbilical cord and cord serum (Nair *et al*. 1996). This suggests the potential for exposure of the unborn foetus to HCH.

Invertebrates

The study of the uptake and elimination of chemicals by edaphic species and the concomitant increase of the interior body concentration of the organisms have been recommended as tools to explain the distribution of pollutants in soils (Moriarty and Walker, 1987; Sousa *et al*. 2000). Terrestrial isopods also play an important role in the decomposition processes and nutrient availability cycling in soil, affecting matter and energy flow through ecosystems (Drobne, 1997). The most suitable changes are observed in reproduction strategies, food consumption, moulting and bioaccumulation processes. The woodlouse species *Porcellionides pruinosus* is often chosen as a study target in several ecotoxicological tests such as studies of the bioaccumulation of pesticides (Sousa et al., 2000) and is one of the species that are in direct contact with soil particles. Storage in body fat is directly proportional to concentration in feed (Bigsby *et al*. 1997). In a study carried out on the isopod *Porcellionides pruinosus* exposed to a constant concentration of lindane via food, it was found that animal body burdens showed higher values, and a lower assimilation rate constant, although the elimination rate constant was twice the value previously observed (0.150 µg/g of original loading of 0.3 µg/g leaf) (Loureiro *et al*. 2002). In one of the studies, the lethal body concentration (LBC) for isopods were found to be 2.36 µg/g animal for bulk soil concentrations and 2.79 µg/g animal for extracted water concentrations. LBC is an alternative to LC_{50} and could be best for determining acute toxicity (Santos *et al*. 2003). Lindane also acts as an ecdysone agonist in an in vitro ecdysone receptor assay using the insect *Drosophila melanogaster* (Dinan *et al*. 2001). As moulting in Crustacea is also regulated by ecdysones, perturbation of development might be predicted as a result of lindane exposure (Brown *et al*. 2003). Table 3 illustrates the toxic effect of lindane on other aquatic organisms.

Toxic substances acting on zooplankton reduce the grazing pressure, thereby diminishing a possible controlling effect on phytoplankton development (Gliwicz and Sieniaswska, 1986; Jak, 1997). Assessing the effects of toxicants on zooplankton may thus be relevant for predicting their impacts on the whole ecosystem and especially on its trophic level. Numerous studies have shown that toxic substances can impair daphnid filtration and ingestion rates at sublethal concentrations (Clément and Zaid, 2003). The toxic effects of lindane on the zooplankton communities like *Daphnia longispina and Daphnia magna* were directly dependent on food availability (Antunes *et al*. 2004). Also, freshwater rotifers can play an important role in assessing the toxicity of a compound to aquatic life. Ferrando *et al*. (1993) reported that the effects of chronic exposure of *Brachionus calyciflorus* to lindane altered the demographic parameters - intrinsic rate of natural increase, generation time, net reproductive rate, reproductive value and life expectancy.

Some studies focussed on the role of bioirrigation in the toxicity determination of lindane in sediment systems. Bioturbation (same as bioirrigation) can easily affect the partitioning of lindane between sediment and water and enhance remobilization of sediment associated lindane. A laboratory study comprising gradients in *Chironomus* (burrowing macroinvertebrate) density resulted in remobilization to the interstitial and overlying water. This led to differences in label (^{14}C – Lindane) recovery. Label recovery on sediment particles ranged from 49-61% of initial label added without *Chironomus*, 41-56% at low larval densities and 15-50% at high larval densities (Goedkoop and Peterson, 2003). The discrepancies in test concentrations and true exposure conditions could be due to volatilization processes taking place. Hence, each toxicity test and bioassay encompasses many limitations and constraints. A multi-faceted approach has to be adopted to achieve concrete decision making.

Impacts

Lindane is metabolized fairly rapidly in standard test species (e.g., rainbow trout, rats) under laboratory conditions. In humans, the half-life of lindane is approximately one day. However, data from some arctic mammals, birds and fish indicate that under conditions of long-term exposure, the bioaccumulation of lindane can be greater than its metabolism. Although there is evidence that lindane has the tendency to bioaccumulate in arctic animals, in contrast with other POPs, there is no clear evidence of biomagnification in the food chain as it is rapidly eliminated once continuous exposure ceases. Indigenous people (autochtones) who rely heavily on animal fats and protein in their traditional diets are particularly at risk from the effects of lindane and other POPs (Sinkkonen and Paasivirta, 2000). A wide variety of toxicological effects are recorded for lindane, such as reproductive and endocrine impairments. Effects from acute exposure to lindane may range from mild skin irritation to dizziness, headaches, diarrhea, nausea, vomiting, and even convulsions and death. Toxicological data indicate that chronic/long-term/lifetime exposure to lindane at high concentrations can adversely affect the liver and nervous system of animals, and may cause cancer and possibly immuno-suppression (Olgun *et al*. 2003). Lindane is also a suspected carcinogen with possible links to breast cancer incidence, and has been found in breast milk and blood samples throughout the world (Ginsburg *et al*. 1977, WHO 1991, Moses 1993, ATSDR 1994, US EPA 1998). A mean concentration of 36.7 pg I-TEQ/g (Toxicity equivalent) fat was found in a group of 54 men and women (mean age of 50 years) in a study conducted in Belgium (Koppen *et al*. 2002).

Acute exposure of fish to a sublethal concentration of lindane (0.05 ppm) resulted in biochemical changes in liver and brain tissue, as well as hyperglycemia (Soengas *et al*. 1997). It affects early development and reproduction of zebrafish (Gorge and Nagel, 1990), rabbit (Seiler *et al*. 1994), bovine (Alm et al., 1998), sea urchins (Pesando *et al*. 2003); shrimp (Huang *et al*. 2004) and mouse (Alm *et al*. 1996; Traina *et al*. 2003) embryos. Lindane also alters the acrosome reaction of human sperm as determined from representative studies on mice (Ronco *et al*. 2001). Effects such as decreased feeding activity were seen in the amphipod, *Gammarus pulex,* exposed to lindane at levels as low as 8.4 ppb, although no effects were seen at 4.1 ppb (Pascoe *et al*. 1994; Blockwell *et al*. 1998). Additionally, the exposure of two aquatic species, *G. pulex* and *Artemia aquaticus* to lindane concentrations of up to 6 ppb at the same time resulted in competitive exclusion. In a recent study, it is shown that lindane from 50 to 200 µM induced deaths of rainbow trout phagocytic cells after *in vitro* exposures. These deaths were related to increases in Reactive Oxygen Species (ROS) production and $[Ca^{2+}]_i$ in the cells (Betoulle *et al*., 2000). A con-

centration of 100 µM lindane also decreased Macrophage-Activating Factor (MAF) production, associated with cellular death (Duchiron *et al.* 2002). If this is left to happen inadvertently for a long time, it would lead to biodiversity loss, a serious secondary effect.

Lindane has also been shown to have effects on estrogen activity *in vitro* as well as *in vivo*. Beard and Rawlings (1998) indicated that lindane is anti-estrogenic as it blocks the response of estrogen-dependent tissues to estradiol. A hormonal imbalance, due to inhibited ovarian steroidogenesis, was also reported in fish exposed to lindane at levels of more than 4 ppm, which included altered sex steroid metabolism and steroid regulation (Singh and Singh, 1992; Silvestroni and Palleschi, 1999). Another *in vitro* test showed lindane to have some estrogenic activity on bovine cells (Tiemann *et al*. 1996). It was also shown to have estrogenic activity in yeast that expresses the human progesterone receptor B-form (Jin *et al*. 1997). There were no adverse reproductive effects found in rats exposed to up to 100 ppm lindane in the diet (approximately 8 ppm bw/day) in a three-generation study (Sample *et al*. 1996). However, Mink?? appears to be more sensitive to the reproductive effects of lindane than rats, with effects on reproductive efficiency at levels as low as 1 ppm bw/day (Beard and Rawlings, 1998).

Table 1
The superlist classification code of Lindane

ATSDR Profile Priority List, rank : 32
Reasonably anticipated to be a carcinogen
Reportable Quantity (RQ) = 1 lb
TWA (0.5 ppm); skin;
TWA 0.5 mg/m^3; skin; Animal carcinogen
Threshold Planning Quantity (TPQ)= 1000/10000 lb
Humans: LD100 = 150 mg/kg and 10-20 mg/kg (acute toxicity)

Table 2
Regulatory status of Lindane

Banned Totally Limited Use Allowed
Bangladesh, Belize, Bolivia, Brazil, Bulgaria, Ecuador, Chad, Denmark, Dominican Republic, Ecuador, Egypt, Finland, Guatemala, Honduras, Hong Kong, Hungary, Indonesia, Japan, Korea, Kuwait, Mauritania, Moldova, Mozambique, New Zealand, Netherlands, Nicaragua, Paraguay, Republic of Korea, St. Lucia, Singapore, Sweden, Former Soviet Union, Taiwan, Tonga, Yemen. Argentina, Australia, Austria, Belgium, Bulgaria, Chile, China, Colombia, Cyprus, Dominica, European Union, Fiji, Germany, India, Israel, Italy, Jamaica, Kenya, Madagascar, Morocco, New Zealand, Philippines, Poland, Sri Lanka, Switzerland, Venezuela, Yugoslavia.

Table 3
Physical-chemical properties of Lindane

Properties	Values
Solubility in water	10 mg/L
Density	1.85 gm/mL
Partition coefficient (octanol-water)	Log K_{ow} 3.20-3.89
Soil-organic carbon-water coefficient	Log K_{oc} 2.38-3.52
Vapour pressure	0.03 mmHg (20°C)
Henry's law constant	2.43 x 10^{-7} atm.m^3/mol (23°C)

References: IARC, 1979; Windholz *et al*. 1983; Montgomery *et al*. 1993

Table 4
Aquatic Toxicity of Lindane

Aquatic Organism	Toxicity concentrations	References
Leuciscusidus melanotus	LC_0 0.05/0.02 mg/L (48h) LC_{50} 0.28/0.003 mg/L (48h) LC_{100} 0.5/0.07 mg/L (48h)	Juhnke & Ludemann, 1978
Brachydanio rerio	LC_0 0.07 mg/L (48h) LC_{50} 0.06/0.09 mg/l (48h)	Braunbeck *et al.* 1990;
Golden orfe	LC_{50} 0.03-0.25 mg/L	Schimmel *et al.* 1977
Carp	LC_{50} 0.28 mg/L	Cossarini-Dunier *et al.* 1987; Svobodova *et al.*,2003
Brown trout (*Salmo trutta*)	LC_{50} 0.0017 mg/L (96h, 13°C)	Czeczuga *et al.* 2002; Triebskorn *et al.* 2002
Lebistes	LC_0 1.3 mg/L (96h)	Chen *et al.* 1971
Water flea (*Daphnia magna*)	EC_0 0.02 mg/L (24h) EC_{50} 0.7 mg/L (24h) EC_{100} 7.0 mg/L (24h)	Zou and Fingerman, 1997; Schulz, 2004
Green algae	EC_{50} 1.7-3.8 mg/l (96h)	Das and Singh, 1978;
Green algae (*Chlorella* spec.)	EC_{50} 0.2-0.3 mg/l (96h)	Lal and Saxena, 1982; Mostafa and Helling, 2002

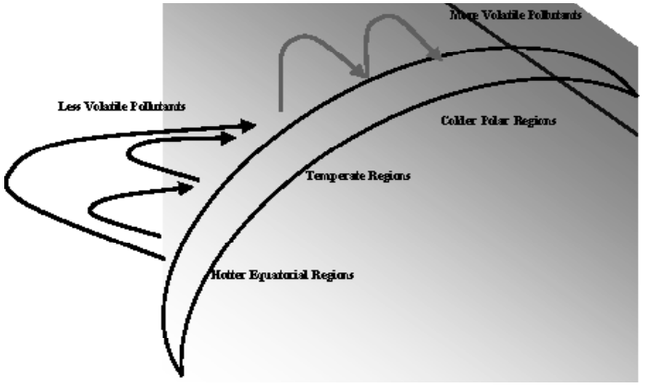

Fig. 1 Grasshopper or global distillation effect of Lindane

Effects on the nervous system, including behavioural effects, have been seen in mammals following exposure to lindane (Rivera *et al*. 1998, Anand *et al*. 1998). GABA (gamma-aminobutyric acid) is the major inhibitory neurotransmitter in the central nervous system of vertebrates. It is responsible for maintaining the electrochemical gradient in the neurons. Lindane has been reported to inhibit GABA-stimulated chloride ion influx in primary cultures of rat cerebellar granule cells, rat dorsal root ganglia or mouse neocortical neurons, by most likely interacting with the non-competitive blocker site of

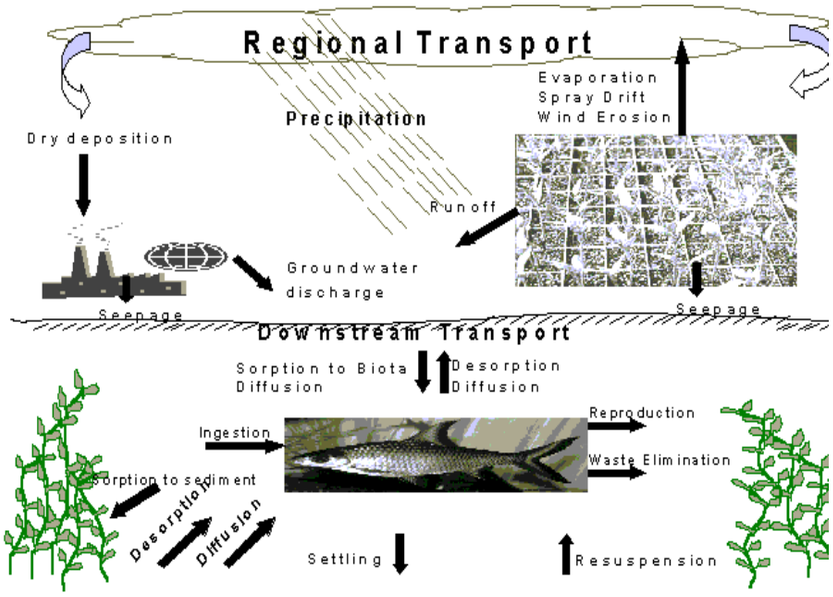

Fig. 2 Mechanism of Lindane transport in the environment.

the $GABA_A$ receptor (Huang and Casida, 1996). Low dose chronic exposure of lindane causes neurobehavioural, neurochemical and electrophysiological effects involving GABA mechanism(s) (Anand *et al*. 1998).

Lindane exposure has been shown to have adverse effects on the immune system of fish, including immunosuppression, at sublethal concentrations of lindane (10 or 15 ppm) (Dunier *et al*. 1995). Liver toxicity, including effects on liver enzyme levels and morphological effects, has also been seen in mammals exposed to lindane at levels of 20 or 60 ppm (Junqueira *et al*. 1997). People exposed to lindane occupationally via inhalation have been shown to have effects on the blood, liver, nervous system, cardiovascular system, immune system and levels of sex hormones. It was also found to be cytotoxic in human leukemia cells *in vitro* (Kang *et al*. 1998; Gulden *et al*. 2002).

Hence, lindane as listed as a priority pollutant and hazardous substance by US EPA and WHO gains further importance in terms of trade off between agricultural production and environmental protection. A sound decision with alternatives needs to be taken to eliminate the existing impacts of this global pollutant and similarly avoid further repercussions in the future.

CONCLUSIONS

Lindane is a persistent and organochlorine pesticidal compound which is widely distributed in the environment. Long distance transport of lindane is obvious by its presence in the Arctic, where it has been never used. It is concentrated in the environment mostly in water, although a significant amount is also found in the soil/sediment and some in air. Lindane has also been

shown to bioaccumulate in the adipose tissue of organisms. It is considered to be highly toxic to aquatic organisms and moderately toxic to birds and mammals. Effects can be manifested in the central nervous system, liver and kidneys. Immunotoxic effects have also been observed in mammals, fish and birds. Marine organisms in all trophic levels, including fish, seals and polar bears, contain significant body burden of this pesticide. Although the use of lindane and technical grade HCH has been banned and/or restricted in many countries, there are a number of developing countries that still use them. The use of technical HCH is banned in North America; however, lindane is still used in Canada. This draws a grave picture of the deteriorating global environment and demands conspicuous and concerted efforts towards elimination and smoother transition to environmentally benign alternatives (for e.g. biopesticides or less toxic chemical pesticides) .

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