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Synthetic Chemical Pesticides and Their Effects on Birds

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ABSTRACT

Use of pesticides, in modern agriculture and vector-borne disease control, has increased tremendously. Pesticides affect the human, environment and wildlife including birds. Three main groups of chemical synthetic pesticides are organochlorine, organophosphate and carbamate. Because of persistent nature, organochlorines are no longer in use in several countries. But some of them like aldrin, dieldrin, lindane and endosulfan are still in use in developing countries. Most organochlorine inhibits Gamma-Amino Butyric Acid (GABA) receptor in brain and affects the central nervous system. They cause widespread population decline of raptorial birds like the peregrine falcon, the sparrow hawk and bald eagle. The well known effect of DDT (dithio dimethyl trichloroethane) in eggshell thinning of the peregrine falcon is caused by its highly persistent metabolite DDE [1,1 , bis-4- chlorphenyl)- 2,2 dichlorethylene]. Organophosphate and carbamate insecticides do not bioaccumulate in the food chains and are less persistent. They have replaced the more persistent organochlorines. Organophosphates like chlorpyrifos and carbamates like aldicarb and carbaryl severely affects birds. Worldwide, hundreds of incidents of OP and CM induced bird poisoning are reported. Both OP and CM inhibit the enzyme, acetylcholinesterase and in acute poisoning 50-70% inhibition occurs. Sub lethal effects of these pesticides are endocrine disruption, alterations in feeding behavior and compromised immune systems which affect avian reproduction. Critical bird habitat is affected by pesticide use. Pesticides cause the local extinction, behavioral changes, loss of safe habitat and population decline in several birds. Use of potential lethal pesticides should be restricted .A toxic regime must be established within the Protected Areas. Policy issues should be strict to save the natural resources. This communication elaborates the effect of synthetic chemical pesticides on birds along with a note on policy framework on use of pesticides.

Key words: Birds, organochlorines, organophosphates, carbamates, acute and sub lethal effects

INTRODUCTION

Avian species have a unique place in ecosystem. They constitute one of the diverse and evolutionary successful groups and occur in large number in the tropics. The threats leading to their population decline are many and varied, but agriculture alone affects 87% of the globally threatened bird species (BLI, 2008). Vast information on them is available which is largely lacking for other groups. Thanks to the efforts of global birdwatchers and ornithologists. Birds provide early warning of environmental problems. Healthy avian populations are indicators of ecological integrity. Decline in avian population show a collapsing ecosystem (US FWS, 2002). Avian populations have a central role in ecosystem functioning and ecosystem services, economic

benefits like seed dispersal, pollination, recolonization and restoration of disturbed ecosystems (Sekercioglu *et al*., 2004) and pest control. Decline of bird population in many parts of the world is a matter of serious concern.

Pesticide induced death in birds is difficult to estimate accurately. Birds may die away from the site of poisoning or their carcasses decompose quickly or may be eaten by the scavengers. As a result, a small portion of such deaths are documented. In the USA, one in three bird species is either endangered or threatened or in need of conservation (NABCI, 2009). The decline is highest in grassland and wetland birds (Rich *et al*., 2004). Forty one percent of the 1138 populations of water birds are known to decline and only 19% is increasing (Delany and Scott, 2002). Basing on such decline, 25 species of family Anatidae are now considered globally threatened, of which almost half have suffered declination by 30% or more over the past 10 years (BLI, 2004). On an average, population of all common and forest birds have declined by about 10% in Europe between 1980 and 2006 and in farmland bird populations, this is 48% (EBCC, 2008).The reduction of grassland habitats for agriculture affects birds detrimentally. For 187 globally threatened avian species, the primary pressure on survival is chemical pollution including pesticides. In the USA, some 50 pesticides are known to kill song birds, game birds, raptors, seabirds and shorebirds (BLI, 2004).

Many pesticides which do not remain at their source but travel long distances are called trans-boundary pollutants. During traveling a long distance, many birds get exposed to pollutants and pesticides. Persistent Organic Pollutants (POPs) like DDT can travel far from their source, tend to accumulate in the fatty tissue of organisms and increase in concentration as they move up the food chain. In the USA, predatory birds, like eagles and cormorants inhabiting the heavily polluted Great lakes region, suffered many problems due to POPs. Such problems were reproductive dysfunction, eggshell thinning, metabolic changes, deformities and birth defects, cancers, behavioral changes, abnormal thyroid activities, endocrine dysfunction, immune suppression, feminization of males and masculinization of females (Orris *et al*., 2000). Both acute and chronic exposure to pesticides increase mortality, while sub lethal exposure adversely affects avian population. One conservative estimate suggests that 672 million birds in the USA get direct exposure each year to pesticides on farmland and 10% of these birds die due to acute exposure (Williams, 1997). The commonly used chemical synthetic pesticides, organochlorines, organophosphates and carbamates are discussed here along with an emphasis on policy framework of their use.

PESTICIDES: THE RETROSPECTION

Pesticides are biocides designed to kill the particular groups of organisms. Some pesticide is specific and others are broad spectrums. The first pesticides used in US agriculture in the 1930s and their adverse effects were discussed in a Wildlife Conference. During that period, about 30 pesticides including pyrethrum, nicotine, calcium arsenate, mercurial fungicides dinitro-ortho-creasol were in use. Both types affect wildlife, soil, water and humans. The insecticidal properties of DDT (p,p'-dichlorodiphenyl-trichloroethane) was discovered in Switzerland in 1939 by Müller, who awarded Nobel prize in 1948 (Axmon and Rignell-Hydbom, 2006). DDT was very effective and has been used to control head and body lice and agricultural pests up to the 1970s. The discovery of DDT and its subsequent application in agriculture started the era of synthetic organochlorine. Benzene hexachloride (BHC) and chlordane were introduced during World War II. Two cyclodiene organochlorines, aldrin and dieldrin were introduced later followed

by endrin, endosulfan and isobenzene. All these insecticides block insect's nervous system, causing malfunction, tremors and death. The damage caused by DDT to wildlife and human was in the news by 1945-48 (Rattner, 2009). Studies for residues in bird tissues and field trials showed that DDT was more toxic to aquatic species than to terrestrial vertebrates (Mitchell *et al*., 1953). The Cyclodiene pesticides caused wildlife mortality, especially when they were used for budworm control and Dutch Elm disease in forests, grasshopper, mosquitoes and fire-ant control (Dustman and Stickel, 1969). DDT and some of the cyclodienes are also used for the preservation of dried and smoked fishes (Musa *et al*., 2010). Organophosphate insecticides originated from compounds developed as nerve gases during World War II. Those developed as insecticides such as tetraethyl pyrophosphate (TEPP) and parathion had high mammalian toxicities. In insects, they act by inhibiting the enzyme Cholinesterase (ChE) that breaks down the neurotransmitter acetylcholine (ACh) at the nerve synapse, blocking impulses and causing hyperactivity and tetanic paralysis of the insect, then death. Most OPs are not persistent and do not bioaccumulate in animals or have significant environmental impacts. The organophosphorus caused record number of wildlife poisonings (game birds, small animals). Parathion poisoning of geese was attributed to spray drift in 1950s (Livingston, 1952). Carbaryl, the first carbamate insecticide, acts on nervous transmissions in insects also through effects on cholinesterase by blocking acetylcholine receptors. Carbamates include aldicarb, methiocarb, methomyl, carbofuran, bendiocarb and oxamyl. Although they are broad-spectrum insecticides, with moderate toxicity and persistence, they rarely bioaccumulate ,or cause major environmental impacts. The cost in environmental and societal damages due to pesticide use in the USA alone was estimated at $12 billion per year: subdivided for public health $1.1 billion; pesticide resistance in pests $1.5 billion; crop losses caused by pesticides, $1.1 billion; bird, fish and other wildlife losses due to pesticides; $2.2 billion and ground water contamination $2.0 billion (Pimentel, 2009).

Pesticides have most striking effects on birds, particularly on carnivorous species which remain at higher trophic level of food chains, such as bald eagles, hawks and owls. These birds are often rare, endangered and susceptible to pesticide residues through food chains. Insect-eating birds such as partridges, grouse and pheasants have decreased due to loss or decrease in insect population in agricultural fields through insecticide use.

Laboratory investigations in the 1960s for toxic accumulation in tissues and reproductive effects of pesticides in game birds and mammals (foxes) were conducted. As early as 1962, some restrictions were imposed on the use of cyclodiene pesticides (aldrin, dieldrin) in Great Britain (Newton *et al*., 1992). In the book "Silent Spring" many pesticidal effects such as bioaccumulation, resistance, extensive contamination of freshwater and ecological imbalances were addressed (Carson, 1962). By the end of the year, more than 40 bills in different state (USA) legislatures had been introduced for regulation of pesticide use (Van Emden and Peakall, 1996). Classic study of Ratcliffe (1967) described the decrease in eggshell thickness in peregrine falcons and Eurasian sparrow-hawks following the application of DDT insecticide. Over the next decade, numerous papers appeared concerning the decline of numbers in fish-eating birds, mainly birds of prey including sparrow hawks, mallards, brown pelicans in relation to DDT and its DDE metabolite.

Gradually, the emphasis shifted toward organophosphorus and carbamate pesticides which were less toxic than chlorinated hydrocarbons. Studies showed that their ecotoxicological effects were less pronounced but adverse effects appeared in animal populations (Hill, 2003). In the next decades, studies projected in other clinical effects of pesticides other than acute and chronic toxicity. Embryo-toxicity, immune-toxicity, reproductive effects, histopathology, endocrine functions, serum

enzymes and cellular damage, oxidative stress, teratogenicity and biochemical indicators were examined (Walker, 2003). Besides direct toxicity, insecticides, herbicides and agricultural practices were found to exert ecological effects to wildlife by altering habitat, vegetation, insect prey base and other parameters.

In the 1990s, major research projects identified and tested sentinel species for potential effects of pesticides (Walker, 2006). Reproductive and endocrine functions in wildlife were launched, along with the publication of "Our Stolen Future" (Colborn *et al*., 1996) to screen pesticides for endocrine-disrupting properties. The scientific advances of the last decades in analytical techniques, biochemistry, environmental chemistry, ecology, population modelling and environmental risk assessment promoted the establishment of wildlife toxicology. Pesticide uses in the 1950s started the scientific interest at that time but many new and unexplained environmental problems continue to drive the field of wildlife toxicology and its ecotoxicological significance to ecosystems (Mineau, 2005).

Pesticide use constitutes a major anthropogenic source of pollution. Rapid expansion of chemical industries during and after world war has added further complexity to environmental chemistry (Mandal and Nandi, 2009). Pesticides at relatively high concentrations are lethal and thereby cause density-mediated indirect effects (Fleeger *et al*., 2003). At sub-lethal concentrations, they can changes the neurotransmitters, hormones, immune response, reproduction, physiology, morphology and behaviour including swimming ability and predator detection (Abrams, 1995). Few studies have examined heritable genetic variation for pesticide resistance in non-target aquatic organisms (Powles and Yu, 2010). Pesticides and environmental pollution have advanced into numerous ecotoxicological and risk assessment studies, as well as into Quantitative Structure-Activity Relationships (QSARs) for environmental toxicology. The scientific literature in the last 20 years contains a series of papers and reviews on environmental pollution and effects on ecosystems. Some toxicological studies have focused on adverse effects on birds and wild animals. A selection of these toxicological studies is included in the study of Van Wijngaarden *et al*. (2005).

Indian scenario: In India, 145 pesticides are registered for use at present. Pesticide production begun in 1952 from a BHC plant near Calcutta, West Bengal. India now stands as the second largest manufacturer of pesticides in Asia and twelfth globally. In India, 76% of the pesticides are used as insecticides, while globally the percent stands at 44 (Mathur, 1999; Saiyed *et al*., 1999).

During the first Five Year Plan (FYP), the pesticide utilization in India was 2350 tons which reached to 7500 tons by the end of the 1990s (Singhal, 1969). Use of pesticide was reduced in 1980s due to the introduction of new pesticide compound.

During the DDT era about 85% of the farmers of the India used organochlorine pesticide at the rate of 0.39 kg ha^{-1} covering 282 million hectares of agricultural land (NCAER, 1967). Now, the consumption of chemical pesticides is highest in Andhrapradesh (33%), followed by Punjab, Karnataka, Tamilnadu, Maharastra, Haryana, Gujrat, Uttar Pradesh and the remaining states account less 9.5 percent of the total (Singhal, 1969). Nearly 70% of the pesticides consumed in India are reported to be utilized for cotton (45%) and rice (22%) and such amount of pesticide use has remained almost unchanged during the last five decades (Vyas, 1998). In India, pesticide use has increased dramatically and now it is becoming a global problem (United States Environmental Protection Agency, 1978). Recent findings suggest that pesticide utilization was negatively related with the scientific orientation and the knowledge of the farmers (Mukherjee *et al*., 2006).

Pesticides/pesticides formulations banned in India are aldrin, benzene hexachloride, calcium cyanide, chlordane, copper acetarsenite, bromochloropropane, endrin, ethyl mercury chloride, ethyl parathion, heptachlor, menazone, nitrofen, paraquat dimethylsulphate, pentachloronitrobenzene, pentachlorophenol, phenylmercury acetate, sodium methane arsenate, tetradfon, toxafen, aldicarb, chlorobenzilate, dieldrine, maleic hydrazidfe, ethyl dibromide TCA. Some of the most troublesome pesticides are DDT, dieldrin, diazinon, parathion, aldicarb, atrazine, paraquat and glyphosate. About 5 million tons of pesticides are applied annually in the world, of which about 70% is used for agriculture and the remainder by public health and Government agencies for vector control and by some owners (Yadav, 2010).

ORGANOCHLORINES (OC) OR CHLORINATED HYDROCARBONS

The OCs are divided into three groups, *viz.* the DDT related compounds, the cyclodiene insecticides (aldrin, dieldrin, endrin, heptachlor and endosulfan) and isomers of hexachlorocyclohexane (HCH). The acute toxicity of p, p'-DDT is attributed mainly to action on axonal voltage dependent Na^+ channels (Eldefrawi and Eldefrawi, 1990). Normally when Na^+ current is generated during the passage of a nerve action potential, the signal is rapidly ended by the closure of the sodium channel. In DDT poisoned nerves, the closure of the channel is delayed causing disruption of action potential regulation which can lead to repetitive discharges (Walker, 2001). Apart from the action on Na^+ channels, DDT or its metabolites also acts as inhibitors of Ca^{++} ATPases in the membrane of avian shell gland and reduces the transport of $CaCO_3$ from blood into egg shell gland. This results in a dose dependent thickness reduction (Lundholm, 1997). DDE is responsible for the severe eggshell thinning of American kestrel, peregrine falcon, sparrow hawks and gannets (Wiemeyer and Porter, 1970). Extensive ecotoxicological investigation on the effect of DDT on eggshell thinning in the Himalayan Greyheaded Fishing Eagle was carried out (Naoroji, 1997). Regarding bioaccumulation of OC pesticide, Tanabe *et al.* (1998) studied the migratory birds of South India and ended that resident birds in India had the highest residues of HCHs and moderate to high residues of DDTs.

The cyclodiene compound, endosulfan alters the electrophysiological and associated enzymatic properties of nerve cell membranes and interferes in the kinetics of Na^+ and K^+ ion flow through the membrane (Hayes and Laws, 1991). Cyclodienes primarily act as inhibitor of GABA receptor and reduce the flow of chloride ions (Krieger, 2001) which leads to neurological disorders like tonic convulsion and clenched claws in predatory birds (Walker, 2003).

Acute toxicity of chlorinated hydrocarbon: DDE residues found in eggs of affected bird were nearly 10 ppm (Peakall, 1993). DDT has also caused local mass death of birds. LD_{50} of DDT in birds is<500 mg kg^{-1} (Edson *et al.*, 1966). Cyclodiene pesticides over stimulate the central nervous system and clinical signs of their acute poisoning include salivation, hyperactivity, respiratory distress, diarrhea, tremors, hunching and convulsions (WHO, 1988).

Cyclodienes have more potential effect than DDT to land vertebrates. The LD_{50} of dieldrin is 67mg kg^{-1} in pigeon (WHO, 1989). Residues of dieldrin, heptachlor epoxide and other OCs in the tissues of British sparrow hawk and kestrel from 1963 to the 1990s are recorded (Newton and Wyllie, 1992). The species show sharp declines in agricultural areas during the said period. Because of its lipophilicity and refractory character, the toxic effect of dieldrin may be carried out to the next generation (Moriarty, 1968). The cyclodiene endosulfan is highly toxic to birds (Kidd and

James, 1991). It is transported over long distances through the air and has been found in the Arctic far from any sources of use (Sang *et al*., 1999). Endosulfun remain deposited in the adipose tissue and in stressed conditions like migration, breeding, illness or inclement weather, their fat is metabolized releasing endosulfan and caused adverse effect even after single exposure (Douthwaite, 1995).

Endosulfan, a neurotoxic pesticide, is highly to moderately toxic to bird species. Administration of endosulfan by the dietary route resulted in lethargy, weakness and diarrhea in Japanese quail (Prakash *et al*., 2009). Acute oral studies conducted in 3-4 months old mallards treated with endosulfan resulted in birds exhibiting wings crossed high over their back, tremors, falling and other symptoms after ten minutes of oral gavage dose administration. The diarrhea and the nervous symptoms produced by endosulfan are due to stimulation of the central nervous system (Hudson *et al*., 1984).

The risk to non-target animals is enhanced, when the top level consumers consume contaminated food. Pesticides (lindane, hexachlorohexane and DDT and its metabolites, heptachlor, heptachlorepoxide, aldrin, endrin, dieldrin and endosulfan α and β) level in human adipose tissue and breast milk (Ebadi and Shokrzadeh, 2006) is noteworthy (Alle *et al*., 2009).

Sub lethal toxicity of chlorinated hydrocarbon

Effect on behavior: Chronic low level OC exposure affects the reproductive success of birds and changes their mating behavior. The affected birds ignore territorial barriers, exhibit less attentiveness to young and decrease the extent of their home range (Fry, 1995). When fed with DDE for longer duration, courtship behavior in ring doves (Haegele and Hudson, 1977) and nocturnal activity in white-throated sparrow (Mahoney, 1975) were disturbed. Sub lethal doses of dieldrin affect the aggressive behavior of mallard duck, social and breeding behavior of bobwhite quail and a variety of effects in the pheasant (Peakall, 1985).

Effect on development: The developing chicks showed malformed beaks and skeleton, fluid retention in their heart and problems in sex determination, after chronic sub lethal OC exposure (Gilbertson and Fox, 1977). Congenital abnormalities and defects of feather growth of young terns are reported after OC exposure along the East coast of the USA (Bourne *et al*., 1977).

Effect on the endocrine system: The United States Environmental Protection Agency (2001) has identified endosulfan as a potential endocrine disrupter. Birds may be exposed to pesticides through contaminated seed consumption. Small birds are particularly at risk due to their low body weight. The birds face high risk due to the consumption of high quantities of seed (United States Environmental Protection Agency, 2006). Lindane affects serum hormone level which is important in reproduction and metabolism. In ewes, concentrations of estradiol and insulin were significantly increased after administration of lindane, while concentrations of basal luteinizing hormone and thyroid levels decreased (Rawlings *et al*., 1998). The reduced hormone levels resulted in decreased egg production (Herbst and van Esch, 1991).

Effect on the hematological and immune system: Anaemia and decreased hemoglobin concentration have been documented after birds were exposed to lindane (Mandal *et al*., 1986). Suppression of T-cell mediated immunity in the wild Caspian terns and herring gulls were found to be associated with high perinatal exposure to OC compounds (Grasman *et al*., 1996). After

administration of 2 ppm endosulfan in chicks for 8 weeks, there was a significant decrease in the number of T and B lymphocytes and total leucocytes along with atrophy and decrease in size of the follicles and hemorrhages in the thymus (Garg *et al*., 2004).

ORGANOPHOSPHATES (OP) AND CARBAMATES (CM)

OPs and CMs are most commonly used pesticides throughout the world because of their low bioaccumulation properties in comparison to OCs. Since the early 1980s, both OPs and CMs have been used as pesticide. Both these insecticides inhibit acetylcholinesterase (AChE) at the postsynaptic membrane of cholinergic synapses (Bishop *et al*., 1998) in the central and peripheral nervous systems of all vertebrate species. OPs inhibit AChE by forming a phosphorylated enzyme derivative, making it more resistant to hydrolysis than the normal acetylated derivative (Taylor, 1990). Inhibition of AChE leads to accumulation of the neurotransmitter acetylcholine at the synaptic cleft in the sympathetic and parasympathetic nervous system and in neuromuscular junctions, thus disrupting transmission across cholinergic synapses (Pope *et al*., 1995). Irreversible inhibition of AChE results in continuous transmission and leads to seizures, respiratory failure and eventually death at high doses (Marrs, 1996). Birds appear to be more sensitive to acute exposure to anticholinesterase pesticides due to a reduced level of anticholinesterase detoxifying enzymes (Parker and Goldstein, 2000). Due to high activity of AChE in the brain of birds (Westlake *et al*., 1983), the rate of binding to OP and CM is more rapid than other vertebrates (Hill, 1992). Most OPs being the potent inhibitors, directly or indirectly (through the toxic metabolic byproduct) inhibit AChE (Extoxnet, 1994). Other alternative sites of phosphorylation and direct effects of OPs on signal transduction pathways are known (Richards *et al*., 1999), such as the inhibition of fatty acid amide hydrolase which affects limb immobility in OP-induced neuropathy (OPIDN) (Quistad *et al*., 2001). OPIDN is characterized by the demyelination of nerve fibers and paralysis which were observed 2-3 weeks after single or repeated exposure (Grue *et al*., 1997). Chloropyrifos, an organophosphate, inhibits AChE in a way that has cross-generational implications (Anway *et al*., 2005).

Inhibition of AChE by CMs either causes death within 30 min or is reversible with decarbamylation. While recovery from CMs usually occurs within 1-2 h, acute OP exposure causes avian mortality within 24 h (Hill, 1992). The metabolism of latent inhibitors in the brain, amount and frequency of exposure and the sensitivity of brain AChE to inhibition are three most important causes of OP toxicity (Hill, 1992). Mortality following exposure appears to be more often related to habitat preferences, physiological condition and /or foraging behavior than a species' ability to deal with actual toxic exposure (Mineau, 1991).

Acute toxicity of OP and CMs: The U.S. Department of Interior's National Wildlife Health Center reported that 50% of the documented cases of lethal poisoning of birds are caused by OPs and CMs (Madison, 1993). The possible route of exposure of these pesticides is the consumption of seeds or insects contaminated on their surface with lethal amounts of insecticide (Prosser and Hart, 2005). Organophosphates have been implicated in 335 separate mortality events causing the deaths of about 9,000 birds in the US between 1980 and 2000 (Fleischli *et al*., 2004). Worldwide, over 100,000 bird deaths caused by monocrotophos, a worst organophosphate, are documented (Hooper, 2002). Application of diazinon, another widely used OP pesticide, to lawns, golf courses and turf farms have killed thousands of birds in U.S (Tattersall, 1991). Diazinon predominantly affects herbivorous waterfowls like ducks and geese. Carbofuran, a CM pesticide alone is

responsible for most bird death in California followed by diazinon (United States Environmental Protection Agency, 1999). Analysis of brain AChE activity revealed that many birds have recovered from illness after sub lethal exposure to insecticides than die (Grue *et al*., 1991). Although several reports are available on the short-term changes of behavior in birds, after exposure to sub lethal doses (Grue *et al*., 1997) reports on the long-term changes in the behavior of birds appear to be few (Grue *et al*., 1991).

Sublethal toxicity of organophosphates and carbamates

Effect on feeding behaviors: OP and CM intoxication are often associated with anorexia and symptoms of gastrointestinal stress (Grue *et al*., 1991). Long-term effects of very small amount of OP affect the feeding behavior of breeding Red-winged Blackbirds (Nicolaus and Lee, 1999). Exposure to OPs and CMs interferes with the bird's ability to discriminate between contaminated and clean foods. Reduction in body weight following sub lethal exposure with an average weight loss of 14% was also noted. Such weight loss is correlated with 55-77% AChE inhibition in European Starlings after a single dose of dicrotophos (Grue and Shipley, 1984). Lesions in lateral hypothalamus due to pesticide exposure lead to food avoidance and cause a sharp reduction in body weight in birds (Kuenzel, 1994).

Effect on endocrine system and reproductive behavior: Alteration in the reproductive behaviour and gonadal development in birds (Kuenzel, 1994) have noticed following acute sub lethal exposure to OPs and CMs due to ventromedial hypothalamic lesions. Delayed development and degeneration of spermatogenic cells has occurred when domestic and semi-domestic birds were exposed to OP's. The decreased level of cholinesterase activity in testes and brains of adult male white-throated munia (*Lonchura malabarica*) is directly related to the increased number of degenerated germ cells in the seminiferous tubules, after exposure to methyl parathion (Maitra and Sarkar, 1996). In another experiment, exposure of adult male roseringed parakeets (*Psittacula krameri*) to the graded doses of methyl parathion resulted in impaired testicular function which may be due to an altered circulating milieu of LH and testosterone (Maitra and Mitra, 2008). When treated with two organophosphates such as methyl parathion and phosphamidon separately, the phosphamidon showed more potential effect and impaired gonadal functions even at very low sublethal doses in female spotted munia (Mitra and Maitra, 2004). The most disturbing sub lethal effect that can be linked to OPs and CMs is their affect on the endocrine system causing reproductive and developmental damage within offspring. Organophosphorus insecticides impaired reproductive function possibly by altering secretion of luteinizing hormone and progesterone (Rattner *et al*., 1984).

Alteration in the migratory behavior (Vyas *et al*., 1995), sexual behavior (Grue and Shipley, 1981; Hart, 1993), litter and clutch size (Bennett *et al*., 1991) and parental care (Grue, 1982), are due to reduced levels of reproductive hormones which results from pesticide exposure. Reduction in singing and displaying in European starling (Hart 1993) and increased aggression in both sexes (Grue *et al*., 1991) are strongly correlated with brain AChE inhibition. In OP exposed mallards, hatching success was reduced by 43% in comparison to controls due to abnormal incubation behavior including nest abandonment and extended time off nests (Bennett *et al*., 1991). OP and CMs reduce egg laying capacity. Reduction in food consumption alone is accounted for reductions in egg laying in Northern Bobwhites fed a diet contaminated with methamidophos for 15 days (Stromborg, 1986).

Alteration in the reproductive behaviour following ingestion of very-low concentrations of OP compounds may be endocrinological or pharmacological in origin. Studies suggest that OPs may influence reproductive functions in different vertebrates by reducing the brain AChE activity and monoamine levels and thus impairing hypothalamic or pituitary regulation on reproduction (Muller *et al*., 1977). In female bobwhite quail, significant decrease in plasma titers of LH, progesterone and corticosterone (Rattner *et al*., 1982) were noted following the short term ingestion of parathion. A variety of pharmacological agents that modify neurotransmitter levels would act at the level of hypothalamus to adversely affect the reproductive functions (McCann, 1982). Possibilities do exist that the insecticides may destroy hormonal homeostasis by suppressing GnRH release which may act directly on the gonadotropins to alter gonadotropin synthesis and secretion or indirectly by altering the pituitary cell responsiveness to GnRH through the actions of gonadal steroids resulting from alterations in FSH and LH by feed back mechanism (Stoker *et al*., 1993).

Effect on thermoregulation: OPs and CMs affect thermoregulation in birds. Acute sub lethal exposure to OP results in pronounced, short-lived hypothermia (Grue *et al*., 1991). OP and CM induced reductions in body temperatures in birds are often associated with decreases in AchE activity of more than 50% (Clement, 1991). The enhanced mortality in birds (i.e. *Falco brain sparverius*) is reported at sub lethal doses at thermo-neutral temperatures (Rattner and Franson, 1984). The interaction between low temperatures and pesticide toxicity appears to be the result of the impairment of thermoregulation, causing inability of birds to withstand the cold (Martin and Solomon, 1991).

Effect on hematological system and immune system response: Exposure to high doses of OPs can cause direct damage to cells and organs of the immune system and decrease the immune function. Histopathological changes in immune tissues and organs, cellular pathology, altered maturation, changes in lymphocytes and functional alterations to immunocompetent cells are documented after OP exposure (Voccia *et al*., 1999; Ambali *et al*., 2010). Other effects include direct damage to proteins and DNA (Videira *et al*., 2001). OPs interfere with immune system response in animals through both anticholinergic and non-cholinergic pathways (Barnett and Rodgers, 1994; Vial *et al*., 1996). Sublethal exposure to chloropyriphos and methidathion to young chickens results in reduction in WBC, neutrophils and lymphocyte count (Obaineh and Matthew, 2009).

CONCLUSION

Chemical pesticides cause serious sublethal effects during the reproductive stages of birds. Sublethal exposure may contribute to other causes of mortality such as trauma. Some bird species are more susceptible to pesticide in which breeding season coincide with the major application of pesticides. The preying birds like peregrine falcon, whooping crain and bald eagle are subjected to secondary poisoning when they consumed prey. Pesticides and their residues can affect birds and their young directly or indirectly by contaminating food sources. Exposure to pesticides during reproductive stages affects hatching success and fledging survival, as well as increases the chance of reproductive failure. Alteration of feeding behavior, compromised immune system and increased predation further reduces the ability of these birds to maintain healthy populations. As behaviour is the result of integration of many inputs, it is considered as a potentially sensitive indicator of pesticide toxicity (Warner *et al*., 1966). For OP, the behavioural effects has been studied in a review

while describing the effects of toxic chemicals on birds (Peakall, 1985), only the behavioural alterations are demonstrated at AChE inhibition (Rudolph *et al*., 1984).

In a recently published article, the impacts of POPs, policy level actions for combating the POP imposed problems, considering both holistic and reductionism approaches for ensuring sustainable development has been discussed (Mandal *et al*., 2007). For controlling the use of pesticides, farmers must be educated for judicious use of pesticides, use of biopesticides and use of pesticides derived from natural products should be promoted. For the human benefit, all uses and risks of pesticides must be considered to ensure conservation of various components of environment. Toxic release inventories and the community right to know will be an effective step in this direction and above all, since pesticides travel far and wide regardless of their point of use, a toxic gime must be established in the protected areas and other biodiversity hotspots. It is only through integrated pest management that is by combining the use of target specific pesticides of lower toxicity to birds with alternative methods that farmers and foresters and other pesticide users can use to reduce this affect. Legislation at the International and National level regarding the use of pesticide should be strictly followed involving the users. Indiscriminate use of pesticides is one of the many serious environmental problems which originate from the root cause of population growth, poverty, inequality in the distribution of wealth and modern unsustainable agricultural practices. Sustainable agricultural practices must be ensured for saving the entire life-support system on earth.

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