Chapter 3
Toxic Effects of Pesticides on Avian Fauna

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Abstract The best-selling book 'Silent Spring' (1962, by Rachel Carson) quivered the international awareness about the role of pesticides for fatally damaging the avian population, but, after more than five decades of such information and advent of various other functionally similar chemicals, still there are overwhelming recent reports of toxicity of pesticides. Globally, over 5 billion pounds of conventional pesticides are used annually for various purposes; these contaminants may act as silent killer of birds. Several studies suggest that the different populations of birds such as songbird, peregrine falcons, ospreys, and Swainson's hawks are unwitting victims of pesticide contamination.

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Popular organochlorine pesticides like DDT (dichlorodiphenyltrichloroethane) have been replaced by moderately toxic broad-spectrum insecticides, including organophosphates and carbamates and synthetic pyrethroids. Unfortunately, exposures to the organophosphates and carbamates still pose major threats to the different avian species. Having being important component of the ecosystem and their plentitude and sensitivity to direct and indirect effects of environment birds make themselves best indicators of early warning of any environmental problems and threats. Species of different shore, grassland, farmland, and migratory birds are directly exposed to lethal doses of these pesticides or through secondary poisoning.

Both organophosphates and carbamates are anti-cholinesterase chemicals, and intoxication emanates through the inhibition of acetyl cholinesterase, resulting in an accumulation of acetylcholine at synaptic junction following subsequent activation of cholinergic receptors which leads to respiratory damage and eventual death. Avian exposure to these pesticides occurs through dermal contact, inhalation, and predominantly the ingestion of contaminated foods such as seeds or insects. A number of factors intensify the likelihood of exposure of birds to pesticides such as cultivation practices, pest types, crop types, pesticide form, diet, and habitat preferences. The sublethal effects of organophosphates and carbamates on birds are manifolds, including malformed embryos, smaller broods, decreased parental diligence, reduced territorial defence, anorexia and weight loss, subdued immune response, lethargic behaviour, greater susceptibility to predation, interference in thermoregulation, endocrine disruption, and inefficiency to orient in the proper direction for migration. Thus, pesticide intoxication reduces the chance of survival and successful reproduction that ultimately perturb to flourish a healthy bird population. The present review tries to encompass the up-to-date information on the succession of the anthropogenic use of pesticide and their consequences on selected bird populations, emphasizing organophosphates and carbamates in particular.

Keywords Birds · Organophosphate · Carbamate · Lethal effect · Sublethal effect

3.1 Introduction

The human population of the world is predicted to increase approximately 10 billion by 2050 (Saravi and Shokrzadeh 2011). With the greater demand for food, intensification of agriculture becomes the primary objective for researchers and implementers. To produce more in the finite land mass, soil amendments (like use of fertilizers) and application of pesticides (for crop protection, as a whole) are common (Köhler and Triebskorn 2013; Gill and Garg 2014). Pesticides become indispensable in agriculture and are used for different purposes like crop protection, stored grain, floral gardens, and eradication of the pests transmitting infectious diseases in plants. Each year throughout the world, nearly 38 billion USD is spent (12.5 billion USD for the USA alone) on synthetic pesticides (US EPA 2011; Germany 2012).

During the early 1950s, organochlorines, the earliest generations of synthetic pesticides, were used in huge quantities in farms and forests. Organochlorines like dichlorodiphenyltrichloroethane (DDT), cyclodienes, and hexachlorocyclohexanes (HCHs) have the property to remain unchanged in the environment for long time. Due to their lipophilic nature or high octanol-water (Kow) and octanol-air partition (Koa) coefficients, warm-blooded organisms (i.e. birds and mammals) cannot excrete easily through, instead getting accumulated in adipose tissue and undergoing biological magnification (Odabasi and Cetin 2012). The book 'Silent Spring' authored by Rachel Carson (Carson 1962) raised the issue of the environmental risks of use of organochlorines especially DDT. The book elucidated publicly for the first time how indiscriminate application of pesticides and other chemicals is polluting water bodies, impairing birds and animals, and causing health problems in humans (Carson 1962). Although most toxic organochlorines (DDT and several cyclodiene compounds) were restricted and banned in many countries, gradually (since 1980), the second-generation, less persistent pesticides like organophosphates and carbamates and synthetic pyrethroids become the popular agriculture pesticides replacing DDT and cyclodienes. Both organophosphates and carbamates exert their effect to both invertebrates and vertebrates through inhibition of acetylcholinesterase at the cholinergic synapses in the nerve endings (Bishop et al. 1998). Accumulation of the neurotransmitter acetylcholine at nerve terminals and neuromuscular junctions leads to incessant transmission having consequences like seizures, respiratory failure, and, eventually, death (Pope et al. 1995; Marrs 1996; Testai et al. 2010) (Fig. 3.1). In human, organophosphate exposure at sublethal doses for longer duration can lead to cancer, diabetes, and neuronal disorders like Alzheimer's and Parkinson's disease (Amani et al. 2016). As organophosphate and carbamates are easily metabolised and readily excreted from the body, they were once considered safe to nontarget organisms. But unfortunately, a number of reports have shown an alarming decline of birds (namely, sparrow-hawk, mallard, and brown pelicans) in the last five decades due to pesticide toxicity (Mineau 1993; Mineau et al. 1999, 2005; Pain et al. 2004; Mineau and Palmer 2013). The second-generation pesticides have the potential to cause lethal effect in birds as birds are more sensitive to cholinesterase inhibitors than other vertebrates (Table 3.1) (predicted LD_{50} (lethal dose) values in sensitive birds are below 1 mg kg^{-1} body weight, whereas in rat this value is <10 mg kg^{-1} body weight) (Mineau et al. 2001; Health and welfare Canada 1987). The rate of binding of cholinesterase inhibitors like organophosphate and carbamates to acetylcholinesterase is more rapid than other vertebrates (Westlake et al. 1983; Hill 1992) because acetylcholinesterase has higher activity in the brains of bird. In different species of avian fauna brain acetylcholinesterase activity ranges from 7.4 to 19.8 μmol/min/g tissue (Shimshoni et al. 2012). As Mineau (2009) described, the abrupt deactivation of the critical cholinesterase in bird's brain and peripheral nervous system leads to 'short-circuiting' neural connections having a multitude of fatal consequences.

As birds play a central role in ecosystem functioning, healthy avian populations are symbolized for ecological stability because they are highly potential for rapid detection of environmental damages (Wayland et al. 2001; Smits and Fernie 2013).

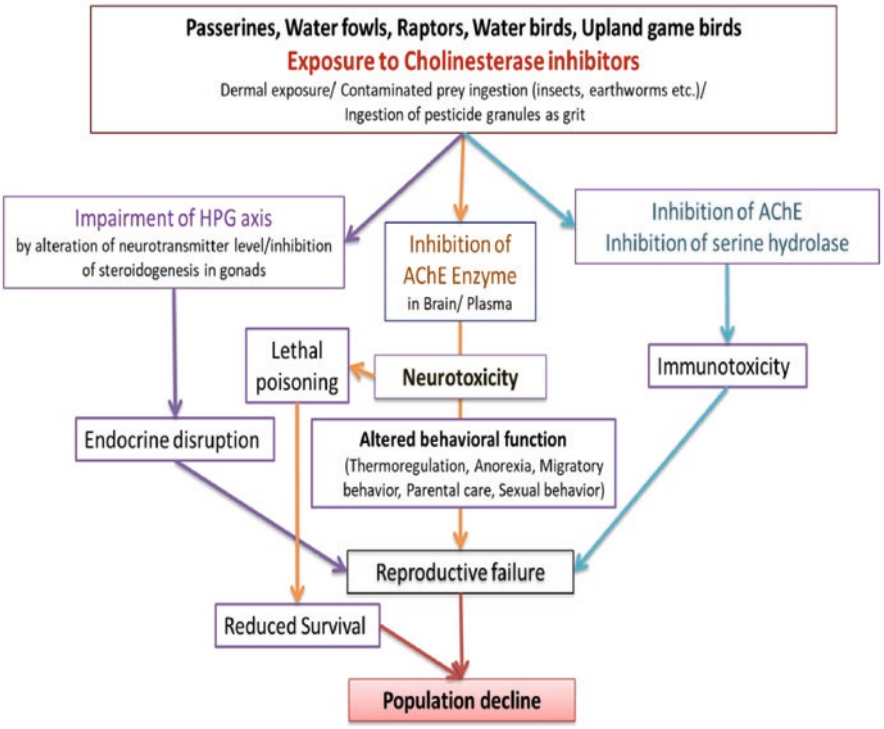

Fig. 3.1 Routes of exposure to cholinesterase inhibitors and their effects on avian fauna
Common routes of exposure of birds to organophosphate and carbamates include inhalation, absorption through the skin, consumption of treated seeds, vegetation with pesticide residues, contaminated insects, granular formulations as grit, food, carrion killed by a pesticide, food intentionally baited with pesticide, and water contaminated with pesticide from runoff or irrigation. Exposure to pesticide resulted in a significant inhibition of brain and plasma acetylcholinesterase (AChE) activity which consequences to death (lethal poisoning) and altered behavioural function. Exposure to such pesticides also leads to inhibition of serine hydrolase activity in immune system and impairment of hypothalamus-pituitary-gonadal axis which results in immunotoxicity and endocrine disruption; all these physiological and behavioural changes reduce the survival and reproductive fitness of the individual and ultimately lead to population decline

Birds accomplish several ecosystem services, like seed dispersal, pollination, controlling pests and bugs (playing a key role as biocontrolling agent), cleaning up carrion, and fertilizing plants, and reclamation of fragmented ecosystems (Sekercioglu et al. 2004; Alanna 2014, Gill and Garg 2014), therefore synchronizing ecosystem function. As they are sensitive and are also prone to contamination due to their food habits (Parker and Goldstein 2000), they are good indicators to assess the quality of the environment and management practices of any ecosystem, agricultural fields, wildlife, water bodies, etc. providing an early warning for environmental problems (Alanna 2014). Researchers stated that declining avian population is a sign of collapsing ecosystem (US FWS 2002; Kendall 2016).

Table 3.1 Acute toxicity of organophosphorus and carbamate pesticides to birds and rat

Pesticide	Class	Species LD_{50} mg Kg^{-1}				
		Mallard duck	Ring-necked pheasant	Red-winged blackbird	European starling	Rat
Aldicarb	Carbamates	3.4	5.34	1.78	4.22	0.8
Carbaryl	Carbamates	>2564	>2000	56	–	600
Carbofuran	Carbamates	0.480	4.15	0.42	–	11
Methiocarb	Carbamates	12.8	270	4.6	13	15–35
Azinphos-methyl	Organophosphate	136	75	8.5	27	13
Phorate	Organophosphate	0.616	7.5	1	7.5	3.7
Temephos	Organophosphate	80–100	31.5	42	>100	2000
Chlorpyrifos	Organophosphate	75.6	8.41	13	5	145
Parathion	Organophosphate	2.2	12.4	2.4	5.6	13
Fenthion	Organophosphate	5.9	17.8	1.8	5.3–17.8	235
Methyl-parathion	Organophosphate	10	8.21	10	7.5	14
Diazinon	Organophosphate	3.54	4.33	2.00	110	250
Malathion	Organophosphate	1485	167	400	>100	1375
Monocrotophos	Organophosphate	4.76	–	1.00	3.30	18
Fenamiphos	Organophosphate	1.68	0.5–1	–	–	8.1

Adopted from Tucker and Haegele (1971), Berg (1982), Walker (1983), Hudson et al. (1984), Schafer et al. (1983), Smith (1987). LD50 is the single oral dose of pesticide in milligrams per kilogram of bodyweight that is required to kill 50 percent of the experimental population

Mortality of birds due to pesticide exposure may occur through different routes, like dermal contact, ingestion of pesticide granules, treated seeds or contaminated water or prey (i.e. secondary poisoning), and poison baits (Fishel 2011; Guerrero et al. 2012). The familiar second-generation pesticides like carbofuran, monocrotophos, diazinon, parathion, and fenthion are also reported to be accountable for avian mortality (Mineau 2002). As for example, mass mortality of Swainson's hawks due to monocrotophos poisoning, in Argentina during 1995–1996, is a well-known phenomenon, after which this organophosphate compound was banned in Argentina (Goldstein et al. 1999). Data suggests that carbofuran (most toxic carbamate pesticide) has been the common cause of poisoning in birds of prey (Novotny et al. 2011; Vlcek and Pohanka 2012). Mineau et al. (2005) reported one of the largest mortality incidents in Canadian prairies with a death of nearly 800 horned larks (*Eremophila alpestris*) and 2000 lapland longspurs (*Calcarius lapponicus*) happened due to application of carbofuran granules in Canadian agriculture fields. Similarly, spraying of carbofuran for controlling grasshopper led to disappearance of burrowing owls in Canada (Fox et al. 1989). In 2011, DWHC reported remarkably high (830 mg kg^{-1} body weight) aldicarb in the stomach of the carcasses of male crow, male lesser-black-backed gull, and juvenile male and female buzzards.

According to a report of BLI, around 50 types of pesticides are involved for killing of different species of birds like songbirds, raptors, game birds, seabirds, and shorebirds (Birdlife International 2004; http://datazone.birdlife.

org/sowb/sowbpubs#SOWB2004). Studies support the activity of cholinesterase inhibitors inducing increased death of predatory and granivorous birds (such as Neotropical migrants, raptors, passerine, and crane) (Eisler 1985; Agriculture Canada 1993; Mineau 1993, 2005; Mineau et al. 1999; Pain et al. 2004; Mineau and Palmer 2013). Nonetheless, the study of pesticide-induced death of birds is difficult for the authors due to elusive nature of birds, as birds may be exposed and then migrate too far from the exposed areas and may die elsewhere or are consumed by scavengers (Hussain et al. 2011; Mitra et al. 2011).

Most current problems of avian acute toxicity due to pesticides stem from the illegal use of the banned pesticide (Mineau and Whiteside 2006) in many countries. The programme organized by FAO (The United Nations Food and Agriculture Organization) made aware the developing countries in the Middle East as well as Africa about the risks associated with outdated pesticide stocks and how they can be and what they can do about them; additionally, the Prevention and Disposal of Obsolete Pesticides programme organized by FAO also supports the states in discharging the stockpiles and hindering the reaccretion of banned pesticides (FAO 2013). As reported by Mansour (2009), most of the African countries have effectively or partly eradicated enormous quantities of persistent pesticides; still, Ethiopia authorizes one of the eminent reserves of banned pesticides (according to a report of FAO, 2013). In Yemen, up to 70% of pesticides are illegally imported for application on fruit and vegetable crops, specifically the qat tree (*Catha edulis*) (UN Office for the Coordination of Humanitarian Affairs 2007). Indeed, the highest amount of pesticides is sprayed in qat cropping areas in comparison to other vegetable crops promoting birds toward higher exposure and intoxication. This review aims to comprehend the up-to-date information on the succession of the anthropogenic use of pesticides and likelihood of the exposure to the avian fauna and their consequences on selected bird populations, emphasizing organophosphate and carbamates in particular.

3.2 Likelihood of Exposure

Different components like form of cultivation and nature of crop, types of pest, nature of pesticide and their forms, and food habits of birds are responsible for probable exposure pathways to pesticides. Probability of exposure to pesticides also depends on bird's ecology related to diet, foraging activities, habitat preferences, and migration behaviour (BLI 2013). Rather than the diet preferences or daily intake rate, foraging location is more important to influence exposure of birds to pesticides (Corson et al. 1998). Birds that search for food near agricultural areas are at higher risk for pesticide exposure rather than the birds that rummage more intermittently in grassland areas (usually, no insecticide is applied in that area) (BLI 2014).

3.2.1 Cultivation Practices May Influence Likelihood of Exposure

Probability of exposure to pesticides in birds is directly linked with the agricultural practices, like timing of pest control in a particular area for a seasonal crop (Narváez et al. 2016; Santos et al. 2016). A study by Osten et al. (2005) with black-bellied whistling duck (*Dendrocygna autumnalis*) reported the direct correlation during the period between pesticide application for specific agronomic purposes and significant inhibition of ChE activity due to exposure to anticholinesterase agents (organophosphate and carbamates). Further, secondary poisoning (like mortality of raptors due to consumption of contaminated songbird) is also evident due to pesticide application (Mineau et al. 1999). Therefore, coordinating time with pesticide applications and bird's annual cycle is necessary to avoid secondary poisoning. Though most of the second-generation pesticides are biodegradable, some of them may persist for a few months in the environment, water-logged fields, etc. making them available for exposure to birds (Mineau et al. 1999). Pesticide exposure rate to birds also depends upon its applications in croplands in different seasons. As for example, in Switzerland granular carbofuran was applied for seed protection in every spring which was linked to the declining of raptor population in spring (Dietrich et al. 1995). Similar findings have been reported by Mineau et al. (2005) in North American canola fields, where insecticides were applied from mid-May to early-June when migratory songbirds and geese were residing in the canola fields resulting in population reversal in those bird species. In some cropland, exposure risk reduces during growth period of the crops, because some birds prefer to spend time during seedling period of the crop rather than budding stages (Corson et al. 1998).

3.2.2 Types of Pest That Increase Risks of Pesticide Intoxication to Birds

Exposure of pesticides to birds depends upon the insect species they feed on, as pesticides are linked to pest control; therefore, birds feeding on insect pests of agriculture fields are more likely to be exposed to chemicals. Some insect species constitute a large fraction of a bird's aliment, which may be the target of particular pesticides. Rapid re-emergence of a pest or relative abundance in a crop field also prompts heavy pesticide applications (Cutler 2012; Qu et al. 2017). Pest resurgence may occur due to the application of less degradable and wide-ranging pesticides that terminate the native predators to a particular pest, or due to eradication of a primary pest that helps to flourish the secondary pests to emerge and become the major pest (Dhaliwal et al. 2006; Gill and Garg 2014). Pest outbreaks allure avian species and thereby unravel the strong possibility of vulnerability of birds to pesticides. Reports revealed the reappearance of bed bug (*Cimex lectularius*) (Davies et al. 2012), cotton bollworm (*Helicoverpa armigera*) (Mironidis et al.

2013), and brown plant hopper (*Nilaparvata lugens*) (Wang et al. 2010) population due to insecticide/pesticide resistance. To control locusts and grasshoppers, pesticides are sprayed on a large scale often in the habitats outside of the croplands; therefore, avian fauna that rummages ahead of cultivated land may also be prone to the toxicant (Dong et al. 2016). As for example, North American Grasshopper hawks (*Buteo swainsoni*) that prefer insects of Acrididae family (locusts, grasshoppers) as their food are more vulnerable to poisoning because of targeting pest outbreaks in agricultural crops (Mineau et al. 1999). Grasshopper control in Argentina by organophosphate and monocrotophos exterminated nearly 5000 grasshopper hawks during the arid summer of 1995–1996 (Goldstein et al. 1999). Pesticide application targeting pest outbreaks poses more risk to bird species than normal preventive use.

3.2.3 Crop Species Raising the Possibilities of Exposure

Cultivation of some specific crop species may hike the liabilities of pesticide exposure to birds (UNEP/CMS/SCC18/Inf.10.9.1, 2014). Pesticides of particular forms (granular/liquid form) and frequent or higher quantities of applications related to cultivation practices that are required for protection of some crops are more vulnerable for birds. Further, some crop types attract birds with higher foraging areas, food source, or other resources. Rice is one of the best examples that has the highest cumulative risk to birds than other crops. Parsons et al. (2010) reported that in the USA, pesticide treatments for rice cultivation cause death of water birds. Again, paddy field provides attractive winter sojourn and migratory halt sites for shorebirds, posing a high risk of exposure to organophosphate and carbamates. Eventually, the pesticide contamination probability to birds is increasing worldwide due to decrease in natural wetlands and ecosystems (Strum et al. 2008).

3.2.4 Formulation of Pesticides Increasing the Risk of Exposure

Particulate Forms

Granular formulations (grit types) of pesticides are more farmers' friendly (than liquid concentrates or powders) as they are easy to apply, highly concentrated, and target specific and have higher retention capacity in the fields (Best 1995). Consumption of natural grit is common to many bird species (especially for granivores and insectivores), as grit assists in grinding the hard foods such as seeds and hard animal parts in gizzard. Granivorous birds like sparrows, finches, and parrots searching for pebbles may consume granular pesticides primarily through incorporation along with

food (Best 1995). These birds are unable to distinguish granular form of pesticide because the carrier medium of pesticide granules is sand (silica) which is the ideal natural grit material (Mineau 2009). Therefore, particulate forms of pesticide may enter into the body of avian fauna either through intentional or unintentional ingestion and/or absorption through exposed area (Best and Fischer 1992). For example, waterfowl are exposed to granular insecticides when sieving for crop residues in pond and drenched soils or intake of grit (Best and Fischer 1992). Parsons et al. (2010) reported the pesticide contamination in water birds in some submerged crop (corn, wheat, and rice) fields in the USA and Canada. Granular forms of the pesticide differ in size, colour, shape, surface texture, carrier material composition, pesticide load per granule, and activity period in the field after application (Gionfriddo and Best 1996; Stafford et al. 1996). Besides primary poisoning, second-generation granular form of pesticides (such as carbofuran) causes secondary poisoning, for example, in raptors, consuming contaminated birds or other wild animals or been contaminated to such granules led to regional population declines (Sánchez-Bayo et al. 2013).

Seeds Coated with Insecticide

Seed treatment with organophosphate and carbamates is very common in developing countries but having been replaced by neonicotinoids (such as imidacloprid) and is no longer approved in some developed countries. Conventional seed treatment by applying pesticides for crop protection was widely used in the UK during the year between 1992 and 2002 (Garthwaite et al. 2003; Prosser and Hart 2005). In Spain, among 18 pesticides permitted for coating of seeds of cereals (MAGRAMA 2013), thiram and maneb are used as fungicides (among the 14), and the rest four are as insecticides. In India, carbofuran-treated seeds have been widely applied, and as a consequence, a number of birds are regularly exposed, though not being reported till date (Venkataramanan and Sreekumar 2012). These pesticide-coated seeds may serve as food source for granivorous birds, which may be fatal to flocks by wilful consumption and subsequent death of carnivorous birds for consumption of contaminated primary population (secondary poisoning) (Fletcher et al. 1996; Almeida et al. 2010). The incidence of avian poisoning due to seed treatment has been reported by Pain et al. (2004) where 15 sarus cranes (*Grus antigon*) and 3 common cranes (*Grus grus*) were intoxicated by monocrotophos at Bharatpur reserve forest in India. Intoxication from treated seeds depends on various factors like sowing area, lethality of pesticide and its concentration on the seed, density of the disclosed seed, and accessibility of alternative food materials (UNEP/CMS/SCC18/Inf.10.9.1, 2014). Again, the risk of exposure to the treated seeds will depend on how likely the birds are to consume them. Birds can avoid the toxic seeds due to the appearance and repugnance of the food or due to feeling of distress due to sublethal toxic effect through a mechanism of conditioned antipathy (Lopez-Antia et al. 2014).

Liquid Formulations and Flowable Pesticide

Liquid formulation is generally mixed with water, but sometimes, crop oil, diesel fuel, kerosene, or some other light oil may be used as a medium. Liquid formulation of pesticide usually contains the active ingredient, the carrier, and one or more other ingredients and may combine the features of emulsifiable concentrate and wettable powders. Although liquid formulations are easier to apply, they are efficiently absorbed through skin of human and other animals. Liquid pesticides lessen the risk of exposure to birds in comparison to the granular application because granular formulations are more attractive to birds (Mineau and Whiteside 2006). However, liquid form of pesticides can contaminate offsite soil, aquatic ecosystem terrestrial flora, and insects from drizzling droplet overflow and erosion after application, and some pesticides may disperse into the air, either becoming airborne during application or volatilizing from treated surfaces (Mackay et al. 2014). The most likely routes of exposure of flowable pesticides to birds are through the ingestion of residues in food items (contaminated vegetables/prey) and in drinking water (on-field puddles or other sources) or through inhalation, dermal contact, and preening (Moore et al. 2014). The risk of flowable pesticide will be greater to birds foraging on-field than off-field birds as greater exposure occurs in treated field. A study reported that mortality rates per day in the flocks of Pennsylvania mourning dove (*Zenaida macroura*) and American robin (*Turdus migratorius*) were significantly higher in the methomyl, oxamyl (carbamate and dimethoate organophosphate), and sprayed apple orchards than in non-treated orchards, and exposure of doves and robins to these insecticides in a frequent rate significantly lowered the reproductive fitness of these species (Fluetsch and Sparling 1994).

3.3 Lethal Effect of Organophosphate/Carbamate Exposure on Birds

The birds are subjected to be at risk of lethal or sublethal doses of any broad-spectrum pesticide like organophosphate and carbamates, if the time of foraging in the adjacent area coincides with the time of application, or shortly after that, or come in contiguity with the contaminated prey. According to the report of the United States (US) Department of Interior's National Wildlife Health Centre, organophosphate and carbamates are responsible for nearly about 50 percent of the registered cases of lethal poisoning of birds (Madison 1993) that occur due to ingestion of contaminated seeds or through secondary poisoning with the high concentration of insecticide (Prosser and Hart 2005). During the year 1980–2000, about 335 individual mortality cases of approximately 9000 birds in the USA happened due to organophosphate intoxication (Fleischli et al. 2004). The worst organophosphate monocrotophos has been reported to cause over 100,000 deaths of avifauna throughout the world (Hooper 2002). Spraying operation of diazinon to backyard,

playground, and grassland in the USA has reported to kill thousands of birds (Tattersall 1991). Waterfowls like ducks and geese are most sensitive to diazinon (LD_{50} value in Table 3.1), and carbofuran alone is responsible for massive mortality of birds in California followed by diazinon (US EPA 1998).

3.4 Sublethal Effect of Organophosphate/Carbamate Exposure on Birds

The effects of organophosphate and carbamate pesticides are multitude. Sublethal effects of cholinesterase inhibitors include production of smaller broods, changes in mobility, feeding and migratory behaviour, endocrine disruption, immunomodulation, and interference in reproduction (Pinkas et al. 2015). Thus, pesticide intoxication reduces the chance of survival and successful reproduction, ultimately perturbing to develop a healthy bird population (Pinkas et al. 2015; Eng et al. 2017). However, scanty information is available on the probable consequences of long-term exposure to organophosphate/carbamates at sublethal level on avian species.

3.4.1 Biochemical Biomarkers for Anticholinesterase Contaminants

Potential biomarkers used to monitor anticholinesterase exposure in birds include determination of cholinesterase (Santos et al. 2016) and carboxylesterase activity (Barr and Needham 2002), assessment of oxidative stress (Henry et al. 2015), and lactate dehydrogenase activity (Barata et al. 2010). However, application of suitable biochemical biomarkers depends on prior knowledge of species-specific and age-related reference and threshold values to identify stress-mediated responses from natural background variation (Domingues et al. 2015; Santos et al. 2019). A dose-dependent decrease in liver carboxylesterase activity was found following exposure to malathion, parathion, and trichlorfon in the range 0.125–2 mM for 48 hrs in Japanese quail (Abass 2014). Similar findings were observed in chlorpyrifos-treated Japanese quail by Narvaez and coworkers (2016). In a recent study, Ćupić Miladinović and coworkers (2018) observed that following chlorpyrifos (CPF) exposure, there was an increased accumulation of ROS in brain cells of Japanese quail (*Coturnix japonica*), supporting generation of oxidative stress. They have also reported the alteration of several oxidative stress-related parameters such as increased concentration of malondialdehyde (MDA), glutathione (GSH), nitrite (NO_2^-), and hydrogen peroxide (H_2O_2)] and increased activity of antioxidative enzymes like superoxide dismutase (SOD) and myeloperoxidase (MPO).

3.4.2 *Neurotoxic Effect*

As discussed, organophosphates and carbamates are well known for inducing cholinergic overstimulation (neurotoxic effect) by inhibiting acetylcholinesterase activity which leads to collection of acetylcholine at nerve terminals (Fig. 3.2) and neuromuscular juncture overstimulating nicotinic and muscarinic receptors (Walker and Thompson 1991; Walker 2003; Testai et al. 2010; Ivanović et al. 2016). Brain acetylcholinesterase is a potential biomarker for monitoring the degree of exposure to anticholinesterase agents and thus assessing the effect of these pesticides on bird population (Busby et al. 1983; Smith et al. 1995; Timchalk 2010; Villar et al. 2010). The anticholinesterase-degrading enzymes were found in reduced level in birds which may be responsible for higher sensitivity of birds to anticholinesterase pesticides than other vertebrates (Parker and Goldstein 2000). However, plasma acetylcholinesterase activity may also be considered as a promising indicator of the central nervous cholinergic status (Oropesa et al. 2013). Studies suggest that in brain of birds, greater than 50% inhibition of cholinesterase activity may lead to death (Ludke et al. 1975; Mineau et al. 2001; Mohammad et al. 2008). In contrast to organophosphate poisoning, post-mortem reactivation of cholinesterase may provide false-negative results in cases of carbamate poisoning (Smith et al. 1995).

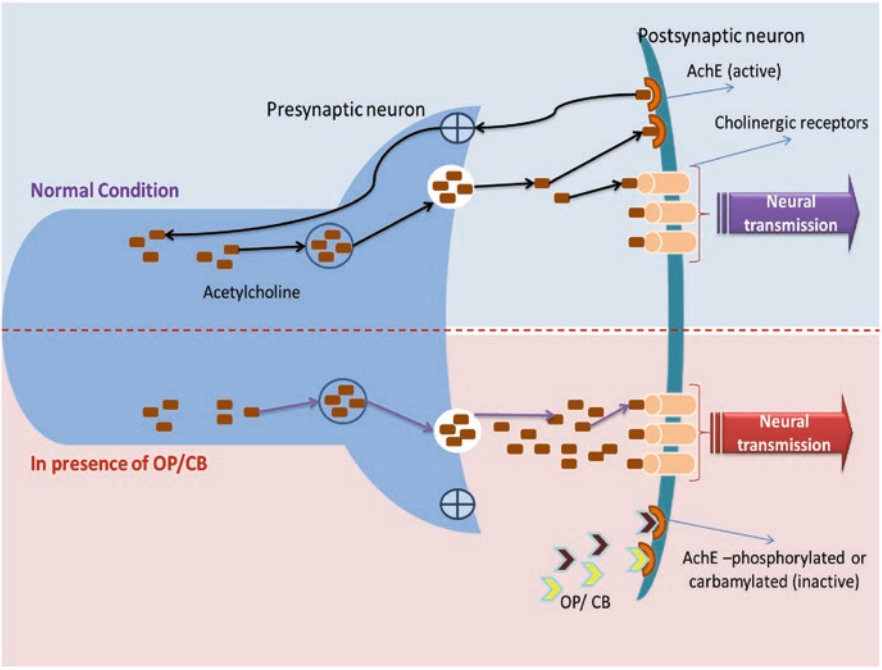

Fig. 3.2 Mechanism of inhibition of acetylcholinesterase (AChE) by organophosphates and carbamates

The reactivation of carbamylated cholinesterase is dependent on the duration and the temperature at which the carcass remained in the field and would therefore help in masking carbamate poisoning (Smith et al. 1995). Secondary symptoms may occur in some organophosphate poisoning called organophosphate-induced delayed neuropathy (OPIDN) in which the target enzyme is brain neuropathy target esterase (NTE), manifested by limb immobility of exposed individual (Lotti and Moretto 2005). OPIDN is symbolized by the demyelination of neurons and paralysis which can be noticed after 20–25 days following single or recurrent exposure(s) (Grue et al. 1997). Neurotoxic effect of organophosphate may also be mediated through oxidative damage by producing reactive nitrogen species [such as nitric oxide (NO•) and nitrogen dioxide (NO_2)]. Excess (NO•) acts as a neurotoxin-promoting neurodegenerative disorders (Di Meo et al. 2016).

Impairment of mitochondrial function plays an important role in the progress of many neurodegenerative disorders (like Parkinson's disease, Alzheimer's disease), which is related to the inhibition of complex I (CI) or ubiquinone oxidoreductase, member of the oxidative phosphorylation system located at mitochondrial inner membrane (Kulic et al. 2011). CI inhibitory effects were found in rats exposed to monocrotophos (Masoud et al. 2009) and dichlorvos (Binukumar et al. 2012), which include acute cholinergic conflict succeeded by possible intermediate syndrome and finally OPIDN. A study reported that hens treated with chlorpyrifos showed sign of delayed neuropathy like ataxia and locomotor disturbances with concomitant inhibition of NTE, mitochondrial CI, and decreased ATP (adenosine triphosphate) production, thereby supporting organophosphate-induced mitochondrial dysfunction in birds (Salama et al. 2014) which may be indirectly responsible for the changes in behaviour due to neurological complications.

Exposure to pesticide at sublethal dose emanated to a significant hindrance in brain cholinesterase activity in reproductively mature adults that can be correlated to an extent behavioural alteration such as limited mobility and interrupted incubating ability; all these changes ultimately lead to considerable lower production of fledged young (Busby et al. 1990). Studies have demonstrated that cholinesterase-inhibiting insecticides exclusively affect almost all physiological and behavioural functions (Greaves and Letcher 2017). Behavioural changes in response to toxic chemicals provide an insight to the population and ecological repercussion in remote future (Raley-Susman 2014). Organophosphate may induce behavioural alteration in avifauna by interfering thermoregulation, food consumption, sexual behaviour, clutch size, embryonic development, mobility, seasonal behaviour, territorial behaviour, and parental care (Grue et al. 1991, 1997). Such switch-over in physiological and behavioural pattern has the potential to reduce the survival and reproductive fitness of individuals, which ultimately affects the population up to local extinction of several bird species (Grue et al. 1997). A perceptible change in a population of white-throated sparrows (*Zonotrichia albicollis*) in Canada took place after forestry spraying operations with fenitrothion (an OP) (Busby et al. 1990). Lethargic behaviour of birds due to sublethal exposure of organophosphate and carbamates can increase the susceptibility for predation of house sparrows (*Passer domesticus*) and bobwhite quails (*Colinus virginianus*) (Hunt et al. 1992; Hawkes

et al. 1996). Red-winged blackbirds (*Agelaius phoeniceus*) exposed to sublethal dose of organophosphate for longer duration was found to affect the feeding behaviour of the species (Nicolaus and Lee 1999). As sublethal exposure of these pesticides is often associated with anorexia, a common consequence of exposure is reduction in the body weight (Grue et al. 1991; Maitra and Mitra 2008; Moye and Pritsos 2010). A single dose of dicrotophos leads to 55–77% inhibition of brain acetylcholinesterase in European Starlings that can be correlated with weight loss (Grue and Shipley 1984). According to Kuenzel (1994), pesticide-induced lesion in lateral hypothalamus leads to food avoidance causing significant body weight reduction in birds. Pigeons exposed experimentally to sublethal dose of chlorpyrifos and aldicarb showed abnormal flight and improper navigation (Moye and Pritsos 2010). Mc Carty et al. (2009) reported that buff-breasted sandpiper (*Tryngites subruficollis*), a species of migratory shorebird that sojourn in spring at the agricultural fields near Rainwater Basin area of Nebraska, is subjected to extensive exposure to pesticide that led the bird to debilitate social and courtship behaviour (Mc Carty et al. 2009).

Organophosphates and carbamates affect thermoregulatory ability in birds causing inability to withstand in cold seasons (Martin and Solomon 1991). Acute exposure of organophosphate at sublethal level showed pronounced but transient hypothermia (Grue et al. 1991). Anticholinesterase-directed hypothermia in birds is often coupled with more than 50% inhibition in brain acetylcholinesterase activity (Clement 1991). The higher mortality rate in American kestrels (*Falco sparverius*) exposed to cold temperature was due to poor thermoregulatory ability (Rattner and Franson 1983).

3.4.3 Effects on Endocrine System and Reproduction

Organophosphates and carbamates are among the endocrine-disrupting chemicals (EDCs) that can cause altered patterns of behaviour by mimicking the action of hormones. Although effects of exposure to EDCs vary from species to species, the bird species at the top of the food chain are particularly vulnerable (Carere et al. 2010). EDCs can interrupt redox homeostasis causing oxidative stress and imbalance between pro-oxidants and antioxidants. Redox balance shifted toward more oxidant condition instigates oxidative damage, anticipating several degenerative pathologies (Abdollahi et al. 2004).

Damstra et al. (2002) suggested that some unique characteristics of birds make them more vulnerable to potential endocrine-disrupting chemicals including consumption of large amount of food and higher metabolic rates, periods of starvation that mobilize lipid reserves, hormone-dependent behaviours, developmental scheme, and regulation of sexual differentiation. Physiological and metabolic processes of a bird can be considerably affected by very delicate changes in the balance of the endocrine system (Damstra et al. 2002; Fernie et al. 2015).

Although organochlorines (prochloraz, endosulfan, dieldrin) are well known for their estrogenic properties, some of organophosphates and carbamates (toclofos-methyl, chlorpyrifos, quinalphos) are also reported to have estrogenic potential (Andersen et al. 2002; Kitamura et al. 2010). Differentiation of avian reproductive system is estrogen dependent; therefore, differentiation of the accessory male and female genital ducts was found to be altered by estrogen exposure in a dose-dependent fashion (Fry 1995). Chatterjee et al. (1992) reported estrogen-like action of quinalphos exposure of which induces vaginal cornification increased uterine wet weight in rat as established by uterotrophic assay. Gonadal impairment may occur due to alteration in steroidogenesis in testis or ovaries of the birds treated with anticholinesterase agents (Ray et al. 1987). Sublethal exposure of organophosphates (methyl parathion/phosphamidon/quinalphos) to wild female of white-throated munia (*Lonchura malabarica*) showed significant reduction in the activity of two important steroidogenic enzymes of growing follicle, namely, Δ53β-hydroxysteroid dehydrogenase (3βHSD) and 17β-hydroxysteroid dehydrogenase (17βHSD) in a dose-dependent manner (Mitra 2006). These two steroidogenic enzymes (3βHSD and 17βHSD) are the key player in the production of oestrogen and progesterone, respectively (Civinini et al. 2001). Degenerative changes at histological level in ovary of the treated birds include reduced thickness of membrane granulosa layer, vacuolation, and exfoliation of granulosa cells of mature follicles (Mitra 2006). Arrested developmental process and degeneration of spermatogenic cells are observed in domestic and semi-domestic birds after exposure to organophosphates (Mitra et al. 2011). Increased number of degenerated germ cells in the seminiferous tubules was found in the testis of adult male white-throated munia (*L. malabarica*), after exposure to sublethal dose of methyl parathion (Maitra and Sarkar 1996). Male rose ringed parakeets (*Psittacula krameri*) when ingested to graded doses of methyl parathion showed subsided testicular function which may be caused by an upset circulating context of LH and testosterone (Maitra and Mitra 2008). Thus, sublethal exposures of xenobiotics adversely effect on reproduction of avian fauna that may not be related to mortality but through a direct effect on population level. Alternative pathway of organophosphate-induced reproductive impairment in different vertebrates may occur by modifying neurotransmitter levels and thus debilitating hypothalamic and/or pituitary monitoring on reproduction (Muller et al. 1977). Possibilities also exist by suppressing GnRH release, which may act directly by modifying gonadotropin synthesis and secretion or indirectly by changing the pituitary cell sensitiveness to GnRH through the mediation of gonadal steroids resulting from adjustment of FSH and LH level by feedback mechanism (Stoker et al. 1993). According to Rattner et al. (1984), organophosphorus insecticides depreciate reproductive function possibly by modulating secretion of luteinizing hormone and progesterone. Rattner et al. (1982) also reported that significant decline in plasma titre of LH, progesterone, and corticosterone was noted in female bobwhite quail following the short-term exposure of parathion.

3.4.4 Effects on Immune System

The immune system is predisposed to any external insults including xenobiotics (Blanco 2011). Impairment of immune system of vertebrates by organophosphate/carbamates has been evidenced by a number of research works in the past decades (Wong et al. 1992; Barnett and Rodgers 1994; Vial et al. 1996; Zhuang et al. 2015). Decreased humoral and cell-mediated response and nonspecific immunity, along with increment in hypersensitivity and autoimmunity, are some immunotoxic effects induced by organophosphate agents (Shahzad et al. 2015). Normal functioning of immune system is impeded through anticholinergic as well as non-cholinergic pathways by organophosphate (Barnett and Rodgers 1994; Vial et al. 1996). Anticholinesterase-induced oxidative stress and immunomodulation are well established in mammalian models (Cabello et al. 2001; Galloway and Handy 2003; Abdollahi et al. 2004; Polláková et al. 2012; Watanabe et al. 2013). Unfortunately, very little information is available about organophosphate-/carbamate-induced immunotoxicity in non-mammalian models like birds. Cupic Miladinovic et al. (2018) have suggested that chloropyriphos-induced oxidative stress in Japanese quail may be responsible for inflammatory responses. Immunosuppressive effects like reduced lymphocyte proliferation and reduced functional status of phagocytic cells have been found in carbaryl-treated chicken (Singh et al. 2007). Young chicks when exposed to sublethal dose of chloropyriphos and methidathion resulted in reduction of total count of WBC, neutrophils, and lymphocyte (Obaineh and Matthew 2009). In the study of Shahzad et al. (2015), there was increased proliferation of interfollicular connective tissue, cytoplasmic vacuolation, oedema, and appearance of pyknotic and fragmented nuclei (marker for degeneration that depleted the frequency of lymphoid follicles in bursa of Fabricius in chlorpyrifos-treated chicks at sublethal dose). Similar degenerative pathologies were also found in spleen and thymus of the treated chickens.

3.5 Pesticides and Birds

In northern Europe and North America, many grassland or farmland bird species are known to undergone population declines in the past five decades (Mineau and Whiteside 2013). Reports from various survey analyses indicated that grassland birds of North America as a group are declining faster than birds from other biomes (Dunn et al. 2000; Sauer et al. 2000; Bird Life International 2013). During 1971–1975 after introduction of organophosphate in the UK, a series of incidents involving mortality of birds have followed after application of herbicides and insecticides for agricultural intensification in UK, primarily via indirect, food-mediated effects (Campbell and Cooke 1997; Potts 1997). Following consumption of coated seeds with carbofenothion resulted in mass mortality of greylag geese (*Anser anser*) and pink-footed geese (*Anser brachyrhynchus*) in the UK, assumed to be around 1%

of the world population of this species (Greig-Smith 1994). This upshot in the UK led to restrictions of the use of carbofenothion and replacement by chlorfenvinphos; however, chlorfenvinphos poses a greater risk to pigeon than to geese. The increased mortality of pigeon leads to replacement of chlorfenvinphos by fonofos during the middle of 1980s (Greig-Smith 1994).

A study to discover out the prime factors for grassland bird declines in Europe and North America based on the data for 23-year period (from 1980 to 2003) was organized by the toxicologist Pierre Mineau and others (Mineau and Whiteside 2013). The study analyses the five potential sources of grassland bird decrement besides fatal pesticide menaces. These are change in agricultural farming such as hay or alfalfa production, farming intensity, or the percentage of actively cropped agricultural pasture, the use of herbicides and insecticide, and change in permanent pasture and rangeland. This study recognizes that the foremost cause of the extensive decline in grassland bird numbers in the USA is acutely toxic pesticides and focuses on the degree to which lethal pesticides, like organophosphates and carbamates, are accountable for the decrement in grassland bird populations. This finding challenges the most likely assumption that change in crop pasture, the chief factor for habitat loss, was the primary cause of those population declines (Mineau and Whiteside 2013).

Pesticides are recognized as one of the cause of the frequent declines of species of Neotropical migrants. From a report of American Bird Conservancy (2009), among the 341 species of Neotropical migrants (includes plovers, terns, hawks, cranes, warblers, and sparrows), 127 bird species were known to decline. Sixty species were in severe decline mode (population decrease of 45% or more), of which 29 were different species of songbird. Due to destruction of natural swampland and grasslands (Knopf 1994; Skagen 2006), shorebirds are enforced to inhabit alternative places like paddy fields and turf grass farms as migratory stopover during their annual journey between breeding and non-breeding territory (Twedt et al. 1998; Corder 2005; Blanco et al. 2006; Robbins 2007). Although application of highly toxic organophosphate and carbamates is regulated in many countries, less toxic organophosphate and carbamate compounds are still recommended to be used in agriculture for controlling pest of the crops including rice and turf grass in various countries of North and South America (Merchant 2005; Blanco et al. 2006; Way and Cockrell 2007). Poisoning of migratory birds in South America has been documented to potential exposure to cholinesterase arresters while utilizing resources in agricultural habitats (Goldstein et al. 1999). Feet of dead birds may be used to monitor the short- and long-term external (dermal) exposure to pesticides beside the traditional method using chemical analysis of pesticide present in the gastrointestinal tract, liver, eggs, and muscle (Alharbi et al. 2016). In addition to mortality (Pain et al. 2004; Wobeser et al. 2004; Renfrew et al. 2006), sublethal exposure to organophosphate and carbamates can elicit a number of behavioural changes such as loss of migratory orientation and slower flight speed due to obstruction in physiological process (Vyas et al. 1995; Grue et al. 1997; Brasel et al. 2005).

Migrants belonging to Nearctic-Neotropical shorebird can be categorized into upland and wetland shorebirds depending on their habitat requirements. Upland

shorebirds like American golden-plover (*Pluvialis dominica*), upland sandpiper (*Bartramia longicauda*), and buff-breasted sandpiper (*Tryngites subruficollis*) prefer dry habitats having low vegetation (Myers and Myers 1979; Isacch and Martínez 2003). The aforesaid species regularly utilize crop pasture migratory sojourn (Strum et al. 2008) and devour a variety of agricultural pests, whose emergence time coincide with the migration period of birds and, thereby, may come into direct contact with organophosphate and carbamates (Houston and Bowen 2001; Nagoshi and Meagher 2004; Isacch et al. 2005). On the other hand, wetland species, such as least sandpiper (*Calidris minutilla*), pectoral sandpiper (*C. melanotos*), and white-rumped sandpiper (*C. fuscicollis*), prefer habitats with standing water and intermittently visit rice fields and other agricultural areas where organophosphate and carbamates are widely used (Hands et al. 1991; Skagen and Knopf 1993; Twedt et al. 1998; Skagen et al. 2005; Blanco et al. 2006). Carcasses of few shorebirds have been found in rice fields shortly after carbofuran (a potent anticholinesterase) application, as reported from a number of field survey (Flickinger et al. 1980, 1986; Littrell 1998).

3.6 Conclusion

Application of synthetic pesticides was initially aimed to intensify agricultural productivity and food availability; however, their negative effects have overweighed their welfares. From the past decades, prevalence of pesticide poisoning of birds and the hazardous effect due to direct and indirect poisoning of synthetic pesticides cannot be overlooked; second-generation insecticides, organophosphates, and carbamates are used in routine agricultural practices, and their effects on avian fauna are varied depending on the plausibility of exposure and the degree of toxic level exposure of the pesticide. However, the likelihood of exposure and related lethal and sublethal effects of the pesticides are formidable to study at the field level. Conversely, it can be stated that the broad-spectrum nature of organophosphates and carbamates makes birds more liable to of exposure, when they are present in the neighbouring areas at the time of applying pesticide, or immediately after that, or getting contact with a pesticide contaminated prey. Migratory birds are more prone to sublethal effects of pesticides having cholinesterase inhibitors that can have significant effects on migratory behaviour of the birds.

The above discussion tries to encompass the severe consequences of indiscriminate pesticide application on avian population. To understand the range to which birds are exposed to pesticides, the foremost requirement is to find out the factors influencing exposure and to figure out preventive measures that might be applied to minimize the exposure. Best management practices integrating control in agricultural practices, use of pest resistant cultivars of plants, and rational use of synthetic pesticide could reduce hazards of pesticide application. Further, advanced approaches in biotechnology and nanotechnology may facilitate the development of pesticides with nominal adverse effects. Community development and extension

programs that could train and motivate the farmers to take up the contemporary integrated pest management (IPM) strategies may reduce the negative impact of pesticides to our environment which may lead to discontinuing our anthropogenic legacy of silent spring.

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