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Review Article

Pesticide Toxicity in wild Life with Special Reference to Avian: A Review

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ABSTRACT

Pesticide manufacturing companies have neither cared to register their pesticide nor provided their pesticide toxicity data. In Oregon, aldrin treated seed grain killed thousand of wild geese and other wildlife in 1973. In Canada 36 geese were killed in 1988 as a result of application of carbofuron and disulfan in Idaho. Phorate was involved in the death of hundreds of waterfowl and several bald and golden eagles in South Dakota in 1989. Many of the incidence involved wildlife kills result from misuse or illegal application. One such incidence occurred in 1990 when an applicator in North Dakota illegally applied carbofuran to carcase for predator control, he was found guilty of killing several forms of wildlife. Pesticides are used for preventing, destroying, repelling and mitigating any pest. Reducing the risk of pesticide exposure to non-target organisms requires applicators to incorporate crop scouting and integrated pest management (IPM) with knowledge of wild life cycle and habitat in developing a farm pesticide applicator plan. Development of such a plan will insure not only most cost effective means for controlling crop pest situations, but also result in the greatest reduction of risk of pesticide exposure to wild life. Pesticide when used with good judgment and care benefit for agriculture and environment to be reviewed in this paper.

Key words: Pesticide toxicity, Avian, Wild life, Pesticide residue

INTRODUCTION

India has achieved self-sufficiency in food production mainly due to introduction of modern agricultural practices, chemical fertilizers and pesticides. Now that we have archived self-sufficiency in food production we must strive for safe food, both for man and animal.

We are consuming at around 80,000 tons, of chemical pesticide per year and over 400 different pesticides are available in the Indian market, only 111 are registered as per requirement of insecticide act. But the other pesticide manufacturing companies have neither cared to register their pesticide [1]. In addition, our farming community in general is not properly educated about the hazard associated with the use of these chemical pesticides. Even the elementary precautions such as covering the nose and mouth during field application are rarely observed. Similarly pesticide are applied on animal body either injudiously or without caring for then necessary precautions. This results in accidental poisoning or pollution of our environment. Some of the pesticides do not decay in nature such as chlorinated hydrocarbon compound, which pose greater threat to our ecosystem [2]. The level of different organochlorine such as DDT, endosulfan, hepatochlor and Iinde along the madras coast and reported a high concentration of these pesticides in the sample indicating pollution load along the madras coast was estimated [3]. Abuse of chemical pesticide is a worldwide problem [4]. These are ever-growing incidents of pesticide poisoning in the developing countries of the world.

The diversity of wildlife habitat throughout our country is surprising in rural areas fenscerows, field, pasture, pond, wetland and woodland comprise important habitat for many species of wildlife. Lands that provide habitat for wildlife also may bring them into contact with human activities. Wildlife species living adjacent to farm land may benefit from the crop grown but may be inadvertently exposd to pesticide used to reduce insect, weed and diese pests of those crops [5]. There is much documentation showing that wildlife can be harmed by particular pesticide. The documentation includes laboratory toxicity studies on various type of wildlife, field trials that must be performed in order to register the pesticide and reports of incidence of wildlife poisoning.

In Oregon, aldrin treated seed grain killed thousand of wild geese and other wildlife in 1973. In Canada 36 geese were killed in 1988 as a result of application of carbofuron and disulfan in Idaho. Phorate was involved in the death of hundreds of waterfowl and several bald and golden eagles in South Dakota in 1989. Many of the incidence involved wildlife kills result from misuse or illegal application. One such incidence occurred in 1990 when an applicator in North Dakota illegally applied carbofuran to carcase for predator control, he was found guilty of killing several forms of wildlife.

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Table 1 provides toxicity comparisons for commonly used pesticides these comparisons are give information to applicator needed to reduce the risk of pesticide toxicity to wild life

S.No.	Trade Name	Family	Toxicity class	Documented effects on wild life
1	Accephate	Organophosphate	I-Fish/aquatic insects III-Birds, mammals	Moderate to slight acute oral toxicity to birds and mammals.
2.	Aldicarb	Carbamate	I-Fish, birds, mammals and aquatic insects	Birds and mammals motility reported after ingestion of exposed granules. It is aquatic organism.
3.	Azinphosmethyl	Organophosphate	Mammals Fish/aquatic insects II- Birds	Extremely toxic mammals in formulation containing high percentage of active ingredients. Highly toxic to fish and aquatic insects.
4.	Carbofuran	Carbomate	I- All	Highly toxic to all forms of wild life even when applied lowest recommended dose.
5.	Chlorpyriphos	Organophosphate	Birds/fish Mammals	Highly toxic to mammals and birds through oral. Extremely toxic to Fish and aquatic insects.
6.	Diazinon	Organophosphate	I. Birds, fish	Extremely toxic to birds, fish, toxic to bees.
7.	Dimethoate	Organophosphate	I-Birds, fish and aquatic insects III-Mammals	Extremely toxic to birds, fish and other aquatic organisms. Pheasants are particularly sensitive to this parasite.
8.	Disulphan	Organophosphate	I- All	Extremely toxic to birds, mammal, fish, bee and aquatic organism.
9.	Endosulphan	Chlorinated hydrocarbon	Fish and aquatic insect Birds. I-II, mammals	Fish kills associated with contaminated with agricultural runoff and cause sufficient mortality.
10.	Esfenvalerate	Pyrethroid	Fish IV -Birds and Mammals	Highly toxic to fish.
11.	Parathion	Organophosphate	I – All	Parathion extremely toxic to birds, mammals through out both oral and dermal exposure at recommended application rate.
12.	Fenofos	Organophosphate	I-All	Highly toxic to mammals birds and fish.
13.	Malathion	Organophosphate	1 Fish, bees III Birds, Mammals	It is toxic to bees, fish and other aquatic insect. No toxic to wild birds and mammals when used as recommended dose.
14.	Phorate	Organophosphate	I- All	Extremely high oral and dermal toxicity has been reported in bird and mammals.

I - Extreme Toxic, II - Moderate toxic III - Less toxic

Pesticide and their classification- Pesticide: Pesticide is any substance or mixture of substance that is use for preventing, destroying, repelling and mitigating any pest.

Pest: Pests are living organisms that occur where they are not wanted or that cause damage to crops or human or other animal. Example includes insects, mice and other animal, unwanted plants (weeds), fungi and micro organism such as bacteria and virus.

Some household products are pesticide all of these common products are considered pesticide: Cockroach spray and baits, Insect repellents for personal use, Rat and other rodent's poison, Kitchen, laundry and bath disinfectants and sanitizer, Products that kills mold and mildew, Some lawn and garden products such as weed killer and some swimming pool chemicals.

Currently 400 members of 4 groups of pesticide are used in India. The first generation pesticides are:

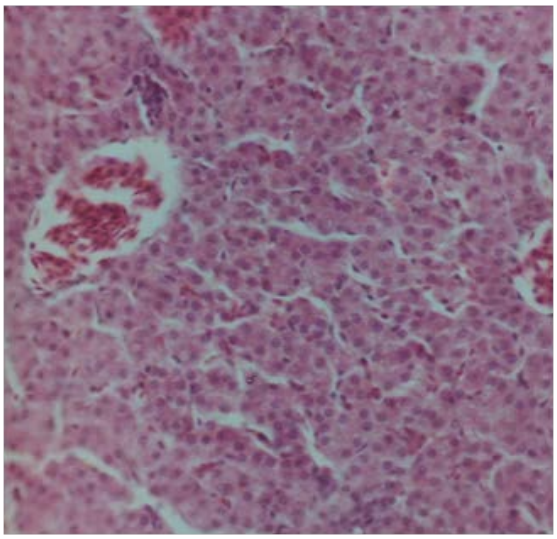
Chlorinated hydrocarbon compound: These are the first generation pesticide. In this group most commonly used pesticide are included: DDT, Endosulfon, Heptachlor, Aldrin, Isodrin and Dieldrin etc.

Table 2: Due to long exposure of wild mammals to pesticide following system of body and symptoms

Category	System affected	Common symptoms
Respiratory	Nose, trachea, lungs	Irritation, coughing, choking, tight chest
Gastro intestinal	Stomach, intestine	Nausea, diarrhea, vomiting
Renal	Kidney	Back pain, urination, more or less than usual.
Dermatological	Skin, eye	Rashes, itching, redness, swelling
Reproductive	Ovaries, testes	Fetus infertility, miscarriage
Neurological	Brain, spinal cord	Behavior confusion, depression, dizziness, convulsion, coma.

Source: Hudson et al [34]

Figure 1 & 2 shows the main symptoms of honeybee pesticide killing large number of dead bees in front of the hives and the second more deadly way is when the bees comes in contact with insecticide and transport it back to the colony, either as contaminated pollen or nectar or on its body, respectively.



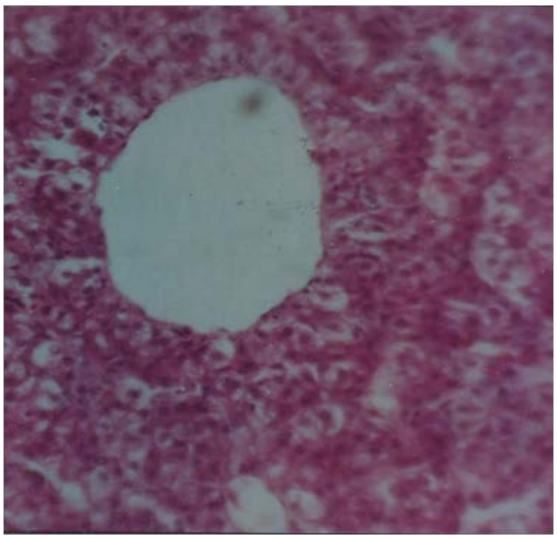


Figure 3 & 4 show histopathological section of liver from fenvalerate intoxicated bird showing vacuolar degeneration of hepatocytes. Congestion of sinusoids and central vein were observed.

Organophosphate: The second generation pesticide includes the following: Quinalfos, Monocrotofos, Malathion, Parathion, Trichlorophan, Fenchlorofos, Phoxim, Abate, Dichlorovos, Fenthion and Haloxon etc.

Carbomates: 3rd generation pesticide, in this group most commonly used pesticides are: Aldicarb, Carbafuron, Carbaryl and Aminocarb etc.

Synthetic Pyrethroid: These are fourth generation pesticide. In this group most commonly used pesticides are: Cypermethrin, Deltamethrin, Permethrin and Cismethrin etc.

Blood chemistry during pesticide toxicity: Blood Glucose: Thr marked elevation of blood sugar level in parathion-intoxicated rabbit was stated [6]. The hyperglycemia in geese after oral administration of Malathion was observed [7]. A significant increase in blood glucose level after organophosphate (dimethoate) exposure of Rat for 15 and 30 days was observed [8].

Blood urea nitrogen: The biochemical changes associated with Malathion toxicity in immature avian species was studied [9]. They found an increase in total protein content of blood plasma. The significant increase in blood urea level in fenvlerate toxicity in goat was recorded [10]. A highly significant elevation of urea in organophosphate intoxicated canines was observed [11].

Acetyl cholinesterase activity (AchE): A decreased in plasma cholinesterase activity in malathion fed male quails was reported [12]. A decreased brain AchE activity in goslings intoxicated with organophosphate (trizophos) pesticide was observed [13]. The AchE activity decrease in rabbit intoxicated with dieldrin was found [14].

Haematology:

They observed reduced number of circulating

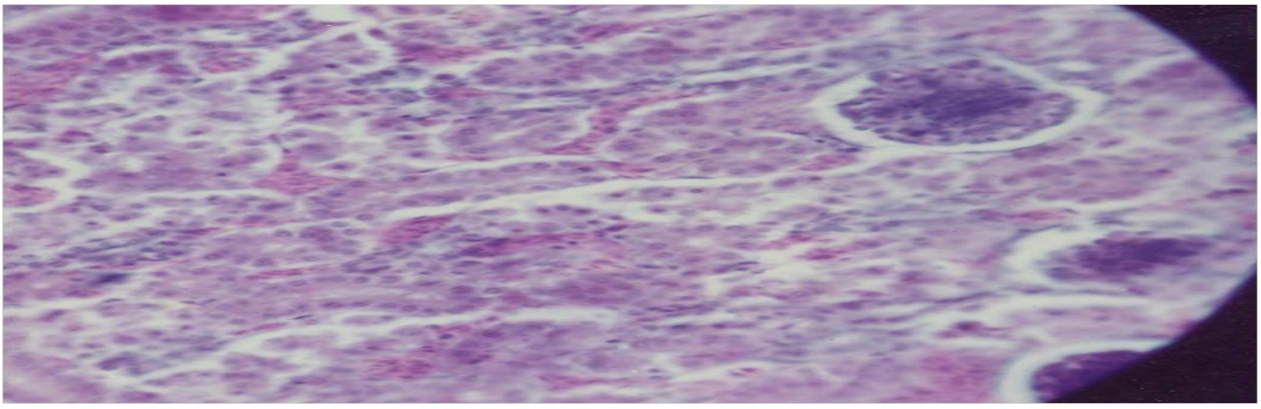


Figure 6 shows section of kideny from Endosulfan toxiciated birds showing congestion and haemorrhages in the intertubular space.

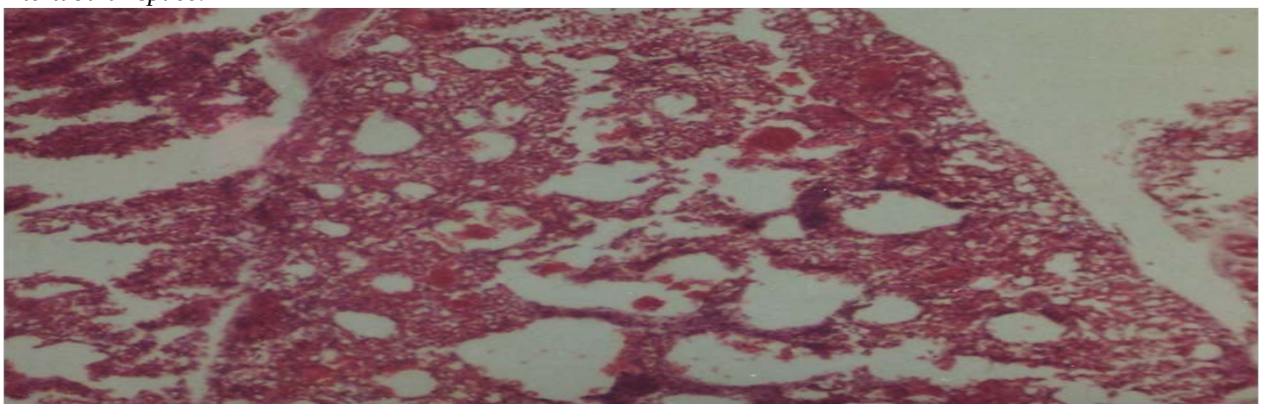


Figure 7 shows histopathological section of lung from fenvalerate intoxicated bird showing severe congestion,hemorrhages and polymorph nuclear cells in and around para bronchus

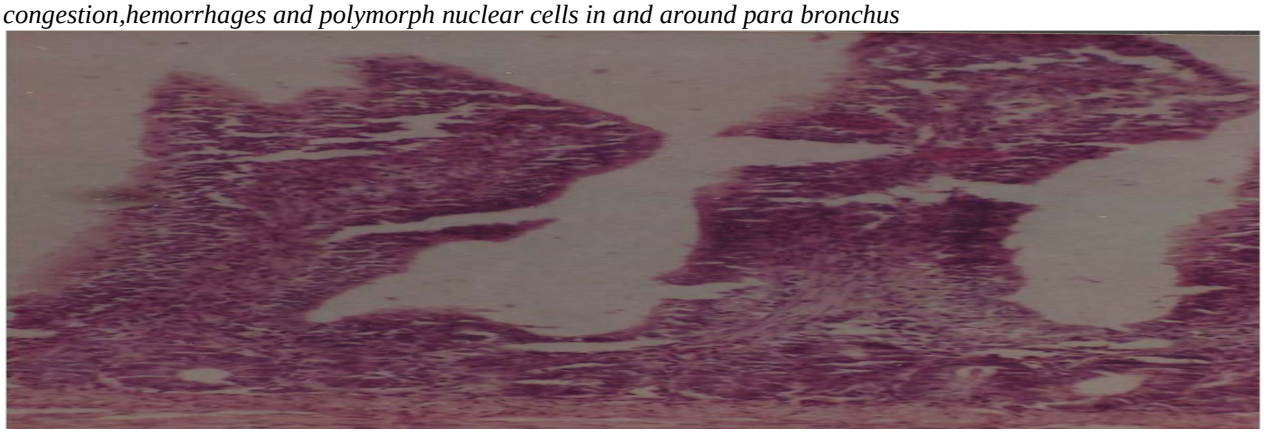
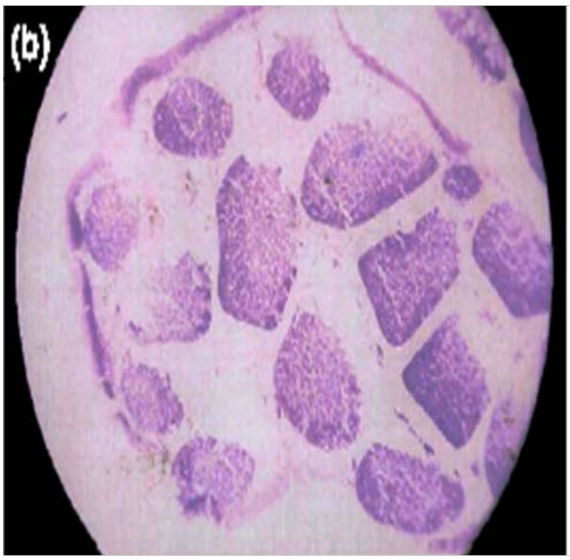
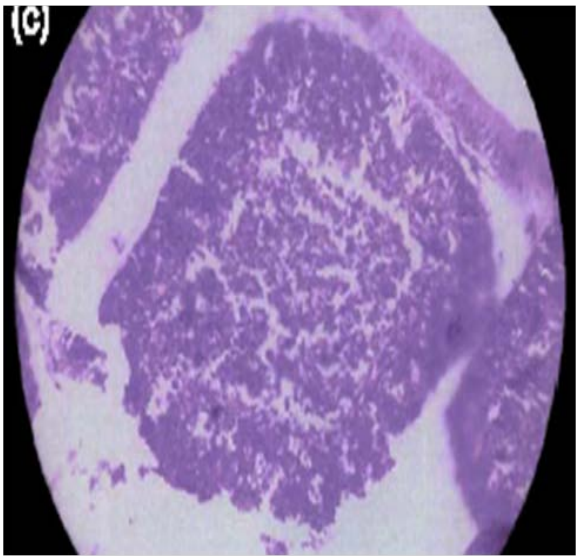


Figure 8 shows histopathological section of Aldrin intoxicated birds showed marked decreased in the height of villi but the width of villous increased.





Figures 9 & 10 : In malathion intoxicated birds showed hypoplasia of bursal follicle and endosulfan intoxicated bird showed mild depletion of lymphocytes in medullary and cortical areas of bursa, respectively.

erythrocytes, neutrophilic leucocytosis, eosinopenia and monocytosis after oral administration of phorate to quails [15]. The effects of various chlorinated hydrocarbons on avian species was studied and concluded that continuous administration of DDT in feed- Reduced haemoglobin concentration in blood and reduced haematocrit value [16]. He also observed increase in the haematocrit and haemoglobin value during chronic aldrin toxicity. The haematological changes in rabbit due to Malathion toxicity was recorded - Decreased haemoglobin concentration in blood, Decreased count of erythrocyte, Leucocytosis and Lymphocytosis [17].

Immunology: It is now being realized throughout the world that long term effect of pesticide adversely affects the ecosystem of the planet and endanger the living being. Thus comprehensive account of immunological status of the pesticide-exposed animal is being presented here.

Organophosphate: Contradictory reports are available to the effect of organophosphate pesticide on the immune function of the experimental animals. Parathion and malathion, suppressed the primary IgM response was reported [18]. They further conclude that organophosphate induced immunosuppression due to direct action of acetylcholinesterase upon immune system. They further reported that parathion suppressed humoral immunity in rabbit [18]. Malathion decrease the globulin producing cells in the popliteal lymph node of the rabbits was reported [19].

Chlorinated hydrocarbon pesticide: The rats exposed to aldrin or lindnane, functional disorder thymus and adrenal gland was concluded [20]. The immunological parameters of rabbit exposed to chlordane for 14 days and reported significance dose related increase in total lymphocyte count due to lymphocytosis was evaluated [21].

Synthetic Pyrethroid pesticide: On the basis of *in vitro* effect of pyrethroid on lymphocyte mitogenic responsiveness. The possibility of immunosuppression by synthetic pyrethroids was reported [22]. The mitotic arrest induced by fenvalerate in human lymphocytic culture was observed [23]. The suppression of cell mediated immunity and antibody forming ability of lymphocyte due to chronic cypermethrin intoxicated in rabbit and mice was observed [24].

Pesticide Toxicity- Pathology: The endosulphan treated birds showed degenerative change in liver and necrosis of lymphoid follicle of spleen a with hyperplastic changes was reported [25]. The following pathological changes in waterfowl due to some organophosphate pesticide toxicity were found [26].

GIT = Food in proventriculus may be colored with dye used to indicate pesticide. Intestinal wall may be congested and hemorrhages seen.

Lungs = congestion, excess fluid.

Kidney = congestion.

They reported that goslings died due to trizophos toxicity and showed oedema of lung during autopsy [13].

They observed that the cats regularly treated with flea powders and strays showed hyperthyroidism and enlarged lymph glands. Enlargement of thyroid was also seen in fish and in rodents fed on these fish [22].

Insecticides associated with bladder cancer in some canine. The risk of bladder cancer was increased in canine, which are living in that area where insecticide regularly spread for mosquitoes [5].

Gills of sport fish damage due to Malathion: Scientists placed blue fish in test tanks that had received small doses of Malathion equate to the amount that have found to be occur in environment after routine spraying. After 24 hours mild degenerative changes seen in gills of fish. After 48 hours damaged is more pronounced and after 96 hours there are severe damage resulting a condition known as “bulbing”. They also said due to

low level Malathion heart defects, blood clot, oscillating blood in heart and pericardial oedema [27]. The lizards exposed to malathion at very low level (1 milligram/kg body weight) cause damage in liver, kidney and intestine was reported [28]. The birds die due to DDT toxicity on necropsy showing petechial and ecchymotic hemorrhages seen in heart and myocardium appears whitish in color. Lungs appear congested and darkened in colour and in some case blood tinged exudates in the bronchioles. The liver may be affected with fatty degeneration and focal necrosis was reported [29].

The organochlorines (DDT, dieldrin and Lindale) toxicity was found in vultures. They are collected 42 sample of vulture that was freshly dead and suitable for detailed analysis. Diagnostic testing includes histopathology, toxicology and electron microscopic of the 14 vultures, which are showing enteritis, arteriolitis, tracheitis, and visceral gout. Organochlorine were detected in fat samples some vultures which indicated exposure of orlganochlorine [30].

Clinical Signs in wild life due to different pesticide: Pesticide toxicity in wild birds: Pesticide can kill birds directly, poison them without killing them directly, or affect them by reducing their food or habitats resources.

Lethal poisoning: Insects and vegetation sprayed with insecticides can contain sufficient residues to kill hungry birds. Organophosphate or carbonates insecticides kill small birds. As well, sufficient quantities of pesticide residues remain in the stomach of poisoned bird and mammals to kill predators and scavengers such as eagles and crows.

Sub lethal poisoning: Not all poisonings result in immediate death.

Due to toxicity of organochlorine in wild life (Avian species) the main symptoms are:

Thickness of egg cell

Decrease in reproductive success

Spasmodic twitching of muscle (including eye lids)

Incardination, which lead to convulsions and blind staggers.

The clinical signs of DDT in avian include: Ataxia, Wing drop, Jerkiness in gait, when symptoms are prolonged for few days, acute toxic tubular nephrosis, Enteritis will be present and result in dehydration was stated [29].

The clinical signs of monocrotofos (organophosphate) toxicity in geese such as: Flaccid paralysis, profuse salivation and heavy mortality were explained [31].

The organophosphate toxicity reported in goslings. In coordination and death was only observed by them. Cholinesterase activity of the brain was observed to be low [13].

The toxic symptoms of moncroptophos toxicity in avian species and reported: Depression, Zig zag movement, Ataxia, Frequent diarrhea, Hypersalivation, Lacrymation, and Paralysis of leg were observed [32].

The clinical sign in waterfowl due to carbamates and organophosphate toxicity was reported such as: Hyperexicitability and restlessness may seen initially, Depression, excessive lachrymator and salivation, Diarrhoea and urination, Dyspnoea with open-mouthed breathing, Cyanosis, lethargy, tremors and convulsions [29].

The fenvalerate toxicosis in cat and symptoms observed: Hypersalivation, Ataxia, Vomiting, Depression and seizures was noted [33].

Toxicity of pesticide in wild mammals: Toxicity in wild mammals mostly referred in two types – acute and chronic. Acute toxicity due to short term exposure and happens within a short term of time, whereas, chronic toxicity is due to long-term exposure and happens over a longer period.

Pesticide kills bees: Unfortunately, honeybees are insects and greatly affected by insecticides when bees direct, contact with insecticides during it is foraging in the field. The bee immediately ties and does not return to the hive. In this case the queen, brood and nurse bees are not contaminated and colony survives. The second more deadly way is when the bee comes in contact with insecticide and transport it back to the colony, either as contaminated pollen or nectar or on the its body. Pesticide kills bees, the main symptoms of honeybee is large number of dead bees in front of the hives. Another symptom is sudden loss of the colony's field force. After honey bee pesticide loss the colony may suffer additionally from blood disease and chilled brood (Barber *et al.,* 1996).

Reducing pesticide exposure risk to wild life: If the selected pesticide posses high threat to wild life, the applicator would beware of the risk and can take the steps to minimize any potential threads by following recommendation (Smith, 1987).

Read the Pesticide label: Certain pesticides pose a risk to wild life. Some products are classified as "RESTRICTED USE PESTICIDE" because of environmental hazard. Restricted use pesticide should be only applied by certified applicator that has been properly trained.

If a particular pesticide is especially hazardous to wild life, it will be stated on the label.

For example –

This product is highly toxic to bees

This product is toxic to fish

This product is toxic to birds and other wild life.

General Environmental statements: General environmental statements appear on nearly every pesticide label as reminders of common sense action needed to avoid contaminating the environment.

Examples of general environmental statements include:

Do not apply pesticide when runoff is likely to occur

Do not apply when weather condition favors drift from treated areas

Do not contaminate water when cleaning equipment or disposing of wastes

Do not allow drift on desirable plants or trees

Do not apply when bees are likely to be in area

Do not apply where the water table and close to the surface.

Use the lowest effective rate: Many times the label will allow a range of rates to control a particular pest. Often pesticide use as in lower rate when pest are in sensitive growth stages, at the lower populations and the weather and growing conditions are favorable.

Use Buffer Zones: An area between the area sprayed and sensitive area is called a buffer zone. This area can be a grass strip or may even be part of the crop that is not treated. This buffer zone will help traps pesticide and prevent them from entering sensitive area by spray drift or by runoff.

Spot Spraying: Many times a pest is located only in a portion of field. Spraying only the area when the pest found leaving the rest of the field untreated and reduce potential risk.

Begin spraying in the middle of the field: Most wild life will be present near the edges of a field. Spraying the field by starting in the middle of the field will allow wild life time move out of the field area.

Check weather conditions:

A good application always check the weather condition before spraying, weather conditions can greatly affect the pesticide exposure through spray drift or runoff. Do not apply pesticide just before rains because pesticide could runoff the treated field with excess rain water and potentially contaminate sensitive areas.

Avoid spray drift:

Spray drift can cause damage to wild life or wild life habitat. The following measures are available to greatly reduce drift:

Avoid Spraying on Windy days: Check the wind speed and direction. If condition could cause spray drift into sensitive areas, do not spray. Another weather condition to avoid is a temperature inversion. Temperature inversions occur when cooler air is near the ground and is beneath warmer air. Very small spray droplets will remain suspended in the air and can move some distance.

Use of nonvolatile formulation: Some pesticide are volatile and can form vapors, usually or warmer days, (greater than 70^0F) which can drift into susceptible areas. If there is an alternative pesticide or formulation, that is not volatile and will control the pesticide.

Increase Droplet size: Spray nozzles produce spray droplets of many different sizes. Larger droplets size will reduce drift, such as increasing nozzle size or water volume, reducing spray pressure.

Increase Water volume: Increasing the water volume will decrease drift because water droplets will be larger and will tend to drift less.

Reduce Spray Boom Height: Set spray booms at the lowest height that will give uniform coverage the closer the boom is to the spray target the less chance there is for drift.

Use a Shielded sprayer: Using a shielded sprayer will help reduce spray drift by protecting the spray from wind. More information on spray drift can be found at your states cooperative extension service county office.

Histopathology: Liver: In endosulfan intoxicated group of bird's liver showed focal lymphocytes infiltration around the blood vessels. There was rarefaction of the cytoplasm of hepatocytes and sinusoids and vein of the liver packed with erythrocyte.

Kidney: Mild nephrosis in the kidney of fenvalerate intoxicated bird, which is characterized by rarefaction, vacuolation and desquamation of the epithelial cells. There was mild congestion of blood vessels in the intertubular space. The glomerular tufts were showing proliferation and Bowman's space was almost filled with it.

Endosulfan toxiciated birds show intertubular blood vessels of cortical region and medullary blood vessels were highly congested. There was mild haemorrhage in the intertubular space.

Lungs: The histopathological examination of lungs of intoxicated group, showed marked changes. All the blood vessels severely congested and some Para bronchi containing varying amount of erythrocytes. The epithelial cells lining of the parabronchi were compressed and at places desquamated. A few mononuclear cells were also seen infiltrating the lamina propria of the perabronchus.

Intestine: Histopathology of Aldrin intoxicated birds showed marked decreased in the height of villi but the width of villous increased. There was serve mononuclear cell infiltration in the lamina propria of intestine.

Bursa: Histopathological picture of bursa varied with type of pesticide. In malathion intoxicated birds showed hypoplasia of bursal follicle. The bursa of endosulfan intoxicated bird showed mild depletion of lymphocytes.

Brain: The cerebrum of DDT intoxicated birds showed satellitosis, neuronophagia and perineuronal oedema. The blood vessel s was congested and had perivascular oedema.

SUMMARY

The responsibility to prevent or minimize of the effect of pesticide applications on nontarget organisms rests with every pesticide applicator. Information concerning the proper use and application of a pesticide can be found on the product label. Reducing the risk of pesticide exposure to non-target organisms requires applicators to incorporate crop scouting and integrated pest management (IPM) with knowledge of wild life cycle and habitat in developing a farm pesticide applicator plan. Development of such a plan will insure not only most cost effective means for controlling crop pest situations, but also result in the greatest reduction of risk of pesticide exposure to wild life. Pesticide when used with good judgment and care benefit for agriculture and environment. Proper use ensures that food and wild life production objective can both be realized.

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