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A review on pesticides and their effects on reproductive system in animals

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Abstract
Toxic pesticides are chemical which do not degrades easily in the environment and get deposited in body tissues through biomagnification. The presence of pesticides in the environment is highly toxic to humans, animals and birds. As they play an important role in increasing the production of food and improve human health by reducing vector borne diseases but they are harmful also in same manner. The residue of environmental pesticides remains in soil, water, air, feed and fodder items for a longer period and contaminate them. This article emphasizes the effects of pesticides on the reproductive system mainly in animals and chickens. Excess pesticides in the environment lead to respiratory distress, increased stress levels, immunosuppression, behavioural changes which ultimately result into decreased production in the form of egg, meat and milk. The pesticides also have detrimental effect on human heath as their concentration increases in body fat depots when their occurrence is higher in environment.

Keywords: Pesticides, reproduction, ovary, testes

Introduction
The use of pesticide is increasing day by day particularly in developing countries [15]. India uses approximately 85,000 tons of pesticides annually and an increase of 8% is expected every year. The residue of such environmental pollutants remains in soil, water, air, feed & fodder items for a longer period, to contaminate them [17, 53]. Since domestic animals with men is here or not share immediate environment they are equally exposed to these pollutant [27, 28]. Chicken are especially vulnerable to pesticides toxicity because poultry houses are dusted with pesticides. The logic that there is general resemblance between man and animal with regard to toxicity profile, suggests that animal population can best be utilized as bio-indicator of pollution.

The potential advantage of domestic animal as means of obtaining early warning of possible effect of increase environmental on human health and screenings from government depots that are sold as poultry feed, after being declared as unfit for human consumption, may be contaminated with pesticides [51]. Exposure of poultry to chemical pesticides causes health consequences to poultry contributing in great economic loss, while also posing a potential threat to public health due to presence of pesticides in poultry meat, ample evidences exist to suggest that the use of pesticides on crop, in store houses, in poultry houses, the no judicious application for spraying or in dipping solution to prevent ectoparasites, leaves behind in residue causing serious health effect [31, 32, 33, 34]. Chronic exposure of chicks to small amount of OPP leads to deleterious effect on metabolism, immune system and reproductive system of birds [16, 27]. In fact, dairy cattle rearing on drinking water contaminated with sewage reduced their reproductive performance [25].

The exposure of males to pesticides can adversely affect pregnancy outcome through a direct genetic or epigenetic effect of their residues on the male germ cells either during spermatogenesis in the testis or sperm maturation in epididymis or by the direct exposure of oocyte during fertilization to the pesticide residues in the seminal plasma [8, 12, 27]. There is growing evidence regarding the adverse impact of certain pesticide residues on reproductive system, and such pesticide residue are known as “reproductive toxicants” or “endocrine disrupters”. These toxicants modulate and or disrupt reproductive hormone milieu by acting at a variety of sites including hypothalamus, pituitary and reproductive organs [17, 47]. Thus, fertility is reported to be declining day by day due to pesticide exposure in world.

Impact on hypothalamic-pituitary-axis

Majority studies have found the sensitive windows *viz*. Development, gestation and lactation for the irreversible impact of pesticide residues on hypothalamic-pituitary axis [19] [35]. On reproductive system, the endocrine disrupting effects of pesticide residues could be through their binding to estrogens, androgen or other receptors. Thus, at hypothalamus, pituitary and at reproductive organs, various endocrine disruptors may act either like endogenous steroids [27]. These pesticides residues can cause imbalance between estrogens and androgens, which ultimately influences the hypothalamic-pituitary-gonadal axis [35].

In females, an extensive damage to ovarian follicles disrupts the endocrine balance leading to a reduction in circulating estrogens and progesterone and an increase in follicle stimulating hormone (FSH) and luteinizing hormone (LH). In an *in vitro* study, progesterone and estrogens release was decrease after the exposure of bovine granulosa cells to OCPs at 0.0001 to 1.0 parts per billion (ppb) [14]. Various endocrine-disrupting pesticide residues can indirectly change the balance of feedback control of hypothalamus-pituitary-ovarian system [35] [39]. Also, the secretion of oxytocin from granulose and luteal cells can be increased by the PCBs [26] [27].

In female

Accumulation of lipophilic residues in ovary/ follicular fluid is detrimental for follicular wall, reduces blastocyst rate and quality, alters neuroendocrine control of ovulation, alters signalling pathway necessary for folliculogenesis, CL Function, estrogens, progesterone, PGF2α+, PGE2 and Oxytocin biosynthesis [35, 54] and for pre-implantation of embryo and Embryo-maternal signaling, stimulates oviduct contraction and impairs fertilization [35, 37, 41].

Leads to embryotoxic effect, chromosomal abnormalities, early embryonic death, foetal death and foetal resorption. Maternal and foetal compartments lead to pesticide deposition in the lipid-rich tissue of the foetus, where poor enzymatic activity increases the accumulation of lipophilic pesticide [50]. Also, the metabolism of pesticide residues in the foetus is particularly low due to their poor binding affinity for the sex hormone binding globulin [24]. Moreover the neonates have a higher percentage of total body water and less body fat to serve as storage site for lipophilic pesticides [29]. Thus, less deposition of pesticide residues leads to their higher circulating levels in the newborn. Additionally, low clearance rates or hepatic metabolism and result in greater toxicity in foetus and neonate [5, 27]. In fact, offspring at birth had dioxins concentrations that were 25% higher than those circulating in the mother [39]. Much of the current evidence suggested that the mammalian reproductive system is susceptible to an alarming impact of detrimental EDCs during the foetal neonatal life than in the adulthood [23]. Following a low dose exposure to pesticides throughout the life of mother, there is accumulation of lipophilic pesticides in the stores [27]. These fat stores are rapidly mobilized during the pregnancy and in particular lactation, and exert endocrine disrupting effects in late pregnancy and post-natal life [6]. During 2-3 weeks post-partum, an increase in PCB concentration observed in milk samples from high producing dairy cows indicated the role of massive lipolysis in liberating and thereby increasing EDC concentrations in milk [17, 37]. The slow metabolism or excretion of unusually high concentrations of pesticides during foetal and post-natal life leads to underdevelopment of reproductive and immune system. Moreover, these underdeveloped systems in the developing foetus are more prone to adverse impact of pesticides [10] [17]. The risk of pesticide exposure through feed for young animals is different from the adult because they consume milk and owing to the high fat content of milk the rates of pesticide exposure are relatively high [37]. Studies have shown that neonatal calves are able to absorb EDCs added to the colostrum even by 1 hr post-partum [22].

The developmental stage at which damage occurs determines the impact that the exposure to pesticide residues on reproduction [19] [48]. In fact, In utero disruption of gametogenesis and gonadal differentiation can have trans-generational consequences [4].

Impact on ovary and oocytes

Chronic exposure to environmental pollutants leads to their accumulation in body fat, blood serum and consequently in follicular fluid [35] [52]. In fact, the follicle basement membrane is permeable to low and high molecular weight substances that can diffuse into follicular fluid [13] [35]. However, the kind of contaminants in follicular fluid may vary according to the geographic area.

In males

An inverse correlation exist between the presence of OCP and OPP residues in the blood or seminal plasma on one hand and blood testosterone concentrations or semen characteristics on the other [27]. Pesticides like chloropyrifos and piperophos have inhibitory effect on testosterone biosynthesis in leyding cells by altering the expression of crucial steroidogenic enzymes [49]. In a study, the exposure of pesticides during foetal life inhibited mid-gestational rise in gonadotropin secretion, and hence about 40% reductions in testis size was observed due to associated decrease in sertoli cell number at birth in sheep [17, 45]. Pesticide exposure associated estrogenic effects may attenuate leydig cell function, fertility and leads to obesity in males [27, 45]. Pesticide-exposed greenhouse workers exhibited high risk of delivering a male with cryptorchidism, smaller penis and testicles, lower serum testosterone, and inhibition-B, higher serum FSH, and higher LH: testosterone ratio [3, 11, 17, 42].

Impact on testis and spermatozoa

Pesticide residues can be detrimental to male reproductive system by causing toxicity to sperm plasma membrane as many lipophilic OCP and PCB residues have the ability to concentrate in seminal plasma and were detected in the seminal plasma of farm animals [20, 27]. Their concentrations were variable among the farm animals due to differences in animal feed, permeability of the blood-testis and blood-epididymis barrier, and the ability of the reproductive system to metabolize and excrete pollutants [39].

The presence of pesticide residues in the fluids surrounding spermatozoa have negative influence on spermatozoa cell function, *viz*. Spermatozoa density, motility and morphology [1, 21]. Organophosphorus pesticide residues can alter spermatozoa chromatin structure and DNA quality at different stages of spermatogenesis, and by disrupting the hypothalamic-pituitary-gonadal axis [27, 40]. In fact, spermatozoa are more prone to negative effects of pesticide residues due to their exceptional large surface D volume ratio (>50:1). However, *in vitro* results are conflicting regarding the impact of pollutants on motility, mitochondrial membrane potential and acrosome reaction of spermatozoa [38].

Endosulfan exposure has the capacity to impair testicular functions by causing alterations in enzymes pathways associated with spermatogenesis [46]. The exposure of buffalo spermatozoa to pesticides (chloropyrifos and endosulfan) negatively affected the spermatozoa plasma membrane integrity, mitochondrial membrane potential and fertilization competence [43]. Although these pesticides caused spermatozoa dysfunctions at a higher concentration on male infertility in farm animals could not be ruled out owing to their cumulative effect or their chronic exposure [43].

Epididymal toxicant, a class of pesticides residues, has impact on the time required for sperm transport through the epididymis [27]. Moreover, higher concentrations of OCP residues were observed in the epididymis than in the testis [9]. During the course of foetal or early neonatal life, any disruption in the differentiation/ multiplication of sertoli cells in fetal testis by the environmental estrogens in detrimental for the adult to produce sperm is determined by the sertoli cells [7, 39, 43, 44]. Even a transient exposure of pesticides during foetal life was associated with abnormal sertoli cell development and poor sperm production in the adult sheep [47]. In utero and lactational exposure of male rats to an environmentally relevant mixture of OCPs can exert anti-androgenic impact during testicular development [2]. Thus, suggesting a potential reproductive hazard for humans and other species. The ewes reared on pastures fertilized with sewage sludge gave birth to offspring that exhibited a decrease in foetal blood testosterone levels as well as reduction in leydig, sertoli and germs cell numbers [36]. Main pathogenic mechanism of toxic effect of pesticide residues on testes is damage of sertoli/leydig cells that leads to disturbed spermatogenesis and testosterone inhibition [17, 30].

Conclusion

Continuous use of pesticides decreases the reproductive performance & reproduction of animals. To come up from this problem, we have to screen natural flora and fauna. As we know these herbs and herb-derived medicines have played a crucial role in health and disease management for many centuries. The global demand for herbal medicinal products has increased significantly in recent years. It is estimated that, the world's population will be more than 7.5 billion in the next 10 to 15 years. This increase in population will occur mostly in the southern hemisphere, where approximately 80% of the population still relies on a traditional system of medicine based on herbal drugs for primary healthcare. Pesticides are capable of persisting in the environment and bio magnifications and pose adverse effects to animal, human health and environment.

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