Toxico-pathological impact of sub-acute exposure to acephate on health biomarkers of broiler chicks

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Abstract

The present study aimed to investigate the immuno-toxicopathologic effects of environmental contaminant acephate (Ace) in experimentally exposed one day old White Leghorn broiler chicks (n = 150). The Ace was reconstituted in groundnut oil as vehicle (1 ml/kg) to obtain a final concentration of a single dose to the birds 21.3, 28.4 and 42.6 mg/kg body weight (BW) for twenty eight days of the experiment through the stomach tube. The chicks in the vehicle control group was given groundnut oil 1 ml/kg only while the chicks of plane control group had received only ad lib standard feed and water. Birds exposed to high dose (42.6 mg/kg BW) showed signs of toxicity (salivation, lacrimation, gasping, convulsions, frequent defecation and tremors). The birds exposed to low dose Ace showed marked increase in the body weight of chicks while medium and high doses (28.4 and 42.6 mg/kg) showed significantly (P \leq 0.05) decreased body weight. Non-significantly ($P \ge 0.05$) decreased TEC, Hb concentration; PCV and TLC were observed in the high dosed group as compared to control and other low dosed fed birds. Initially a nonsignificant compensatory increase followed by significant decrease in serum protein was observed during the study period. Serum albumin showed a significant ($P \le 0.05$) decrease in high dosed Ace fed birds on 28th day of study. Non-significant increase and significant decrease in serum on 14th and 28th day of study was observed respectively. The AChE activity was significantly ($P \le 0.05$) decreased in blood, serum and plasma in Ace fed birds compared to control birds. we found significantly $(P \le 0.05)$ higher levels of serum ALT and AST in Ace fed birds as compared to control. During the experimentation Ace had showed dose dependent immunosuppressive effect on humoral immune response of birds from 28th day of experimentation. The Bursa of Fabricius in treated birds showed increased inter-follicular connective tissue proliferation, severe moderate cytoplasmic vacuolation, edema, and degenerative changes such as pyknosis and fragmentation of nuclei that depleted the follicles of lymphoid cells. In the spleen, disorganization of follicular patterns, severe congestion, cytoplasmic vacuolation, degenerative changes, and hyperplasia of reticular cells were noted. The thymus in treated birds exhibited congestion, hyper-cellularity, and a presence of immature monocytes in the medullary region, as well as myoid cell necrosis. In conclusion, these studies clearly demonstrated that Ace could induce immuno-toxicopathological effects on health biomarkers of broiler chicks.

Introduction

The present study aimed to investigate the immuno-toxicopathologic effects of environmental contaminant acephate (Ace) in experimentally exposed one day old White Leghorn broiler chicks (n = 150). Decreased immunocompetence in animals and birds due to environmental contaminants may lead to increased vulnerability, recurrent infections,

disease epidemics, and vaccine failure. Today, the use of pesticides has become inevitable in agriculture in order to increase agricultural production. However, it is now recognised that the unsystematic and negligent use of pesticides could jeopardise the ability of humans and other animals to combat disease, including poultry. Organophosphates (OPs) which have replaced the use of organochlorines (OCs) are known for their pesticidal potency. The findings support their harmful effects on the immune, endocrine and nervous systems. Acephate is one of the top 10 organophosphate insecticides marketed worldwide. It is a watersoluble insecticide belonging to a phosphoramidothioate group of organophosphate insecticides and is known to be non-phytotoxic in many crop plants (Worthing 1987). Since the presence of insecticide residues in agricultural products is harmful to both animal and human health, this study was conducted to investigate the effects of repeated oral administration of acephate on the male white leghorn (WLH) cockerel chick immune system (Tripathi et al., 2007; Tripathi et al., 2011).

Materials and Methods

The Ace was reconstituted in groundnut oil as vehicle (1 ml/kg) to obtain a final concentration of a single dose to the birds 21.3, 28.4 and 42.6 mg/kg body weight (BW) for twenty eight days of the experiment through the stomach tube. The chicks in the vehicle control group was given groundnut oil 1 ml/kg only while the chicks of plane control group had received only ad lib standard feed and water.

Results and discussion

Birds exposed to high dose (42.6 mg/kg BW) showed signs of toxicity (salivation, lacrimation, gasping, convulsions, frequent defecation and tremors). The birds exposed to low dose Ace showed marked increase in the body weight of chicks while medium and high doses (28.4 and 42.6 mg/kg) showed significantly (P ≤ 0.05) decreased body weight. Nonsignificantly ($P \ge 0.05$) decreased TEC, Hb concentration, PCV and TLC were observed in the high dosed group as compared to control and other low dosed fed birds. Initially a non-significant compensatory increase followed by significant decrease in serum protein was observed during the study period. Serum albumin showed a significant (P ≤ 0.05) decrease in high dosed Ace fed birds on 28th day of study. Nonsignificant increase and significant decrease in serum on 14th and 28th day of study was observed respectively. The AChE activity was significantly ($P \le 0.05$) decreased in blood, serum and plasma in Ace fed birds compared to control birds. we found significantly ($P \le 0.05$) higher levels of serum ALT and AST in Ace fed birds as compared to control. During the experimentation Ace had showed dose dependant immunosuppressive effect on humoral immune response of birds from 28th day of experimentation. The Bursa of Fabricius in treated birds showed increased inter-follicular connective tissue proliferation, severe moderate cytoplasmic vacuolation, edema, and degenerative changes

such as pyknosis and fragmentation of nuclei that depleted the follicles of lymphoid cells. In the spleen, disorganization of follicular patterns, severe congestion, cytoplasmic vacuolation, degenerative changes, and hyperplasia of reticular cells were noted. The thymus in treated birds exhibited congestion, hyper-cellularity, and a presence of immature monocytes in the medullary region, as well as myoid cell necrosis. The present research was performed on one day old WLH cockerels. Due to continuous proliferation and differentiation, the immune system is one of the most susceptible pesticide targets. The immune system of these chicks was more vulnerable to toxicity as the lymphoid organs in the chickens were in growing phase On the basis of these results, it can therefore be inferred that acephate is immunotoxic and that activation of apoptosis is one of the forms in which immunotoxicity is achieved. Consistency of findings from all studies supports the point that this pesticide triggers apoptosis and causes immunotoxicity. There is no assay that can finally detect and quantify immunosuppression and apoptosis or necrosis in the cells. It is therefore important to analyse multiple parameters, as done in this study, before drawing conclusions, particularly as apoptosis is a very rapid event and both modes of cell death are likely to occur simultaneously (Cocoran et al., 1994).



Figure 1: Gross pathological examination of experimental chicken

Conclusion

The findings suggested that acephate has been found to be Immunotoxic at the dose level tested. The immune system is one of the most sensitive pesticide targets. Continuous proliferation and differentiation of acephates at this exposure level may be considered immunotoxic. In conclusion, these studies clearly demonstrated that Ace could induce immuno-toxicopathological effects on health biomarkers of broiler chicks.

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