Evaluation of liver lipid peroxidation and antioxidant profile in broiler chicken fed with mixture of T-2 toxin and endosulfan

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ABSTRACT


The combined feeding of 0.5 ppm T-2 toxin and 20 ppm of endosulfan for four weeks significantly (P<0.05) increased the liver lipid peroxidation in the T-2 toxin+endosulfan treated group compared to the control in experimental broiler birds. The endosulfan treated group showed significant (P<0.05) increase in the GST values compared to the control. The values of catalase decreased significantly (P<0.05) in all toxin treated groups compared to the control. No significant differences were observed for GSH, GPx and SOD between group showed significant (P<0.05) increase in the GST values compared to the control. The values of catalase decreased significantly (P<0.05) in all toxin treated groups compared to the control. No significant differences were observed for GSH, GPx and SOD between control and the toxin treated birds. The present study indicated that T-2 toxin and endosulfan combination caused considerable damage in the broiler birds.

Keywords: Antioxidant, chicken, endosulfan toxicity, lipid peroxidation, T-2, toxicosis

The worldwide occurrence of mycotoxins and pesticides contamination of feedstuffs and the severity of the diseases caused by these xenobiotics in farm animals have increased in recent years. Many factors contribute to this increase such as the global climatic change, increased and indiscriminate use of pesticides and increased international trading of feed stuffs from different geographical origins. Xenobiocits are well known for the induction of oxidative stress. Oxidative stress is defined as a disruption of the pro-oxidant -antioxidant balance and occur as a result of an increase in reactive oxygen species (ROS). Although, the individual toxic effects of T-2 toxin, a cytotoxic trichothecene mycotoxin produced by various species of Fusarium and endosulfan, a cyclodiene organochlorine insecticde in poultry are well documented, however, there is no evidence of what health effects result when they are in mixtures. Hence, the present work was planned to study the role of T-2 toxin and endosulfan on liver lipid peroxidation and antioxidant profile in broiler chicken.

The T-2 toxin was produced on wheat culture by growing Fusarium sporotrichioides var sporotrichioides MTCC 1894 culture. The T-2 toxin from the ground wheat culture was quantified by using thin layer chromatography. Technical grade endosulfan (96.4%) was obtained from M/S Hyderabad Chemicals, Hyderabad, India. Powdered culture material containing known amounts of T-2 toxin and endosulfan were incorporated individually and in combination into the mycotoxin free diet, so that the diets contained T-2 toxin (0.5 ppm), endosulfan (20 ppm) and T-2 toxin and endosulfan (0.5+20 ppm). A toxin free diet was also kept as control. Forty, day-old commercial broiler chickens (Vencob, India) were weighed, wing-banded and randomly allotted to four treatment groups of 10 chicks each. The birds were raised on battery brooders with ad libitum supply of feed and water. The diets were fed for 28 days. The birds were sacrificed at the termination of the experiment following ethical procedures. Liver tissue samples were collected from all control and toxin fed birds at the end of the 28 days of trial and stored at -20°C till the biochemical assays were carried out. Liver homogenate samples were used to determine the lipid peroxidation, Glutathione peroxidase (GPx), glutathione-S-transferase (GST), catalase (CAT) and superoxide dismutase (SOD). The non-enzymatic antioxidant reduced glutathione (GSH) was estimated by the method of Moron et al. The data generated were subjected to one-way analysis of variance using SPSS version 10.0 software for windows.

Mean lipid peroxidation (TBARs) and antioxidant status in liver homogenates in broiler chicken fed T-2 toxin and endosulfan are presented in the Table. A significant (P<0.01) increase in the liver lipid peroxidation values were observed in the T-2 toxin+endosulfan treated group when compared to the control. The T-2 toxin and endosulfan fed groups showed a numerical increase in the liver lipid peroxidation values. Endosulfan treatment caused increased liver lipid peroxidation (59.48%). Similarly, earlier workers reported increased lipid peroxidation of erythrocyte cell membrane in broiler chicks fed 30 ppm endosulfan for 60 days. There was numerical increase in the liver lipid peroxidation in T-2 toxin (18.12%) fed birds. Similar increases in liver lipid peroxidation were reported in experimental T-2 toxicses
in chicken\(^1\) and broiler chicks\(^{11,13}\). The study suggested that membrane damage occurred in the intoxicated hepatocytes. Liver lipid peroxidation is one of the responsible factors for the damage and necrosis of liver, induced by chemical compounds like T-2 toxins. Because biological membranes are rich in unsaturated fatty acids, the susceptibility of membranes to peroxidative attack is not rare as was also opined by earlier workers\(^{13}\). T-2 toxin and endosulfan when given alone caused a numerical increase in the liver lipid peroxidation while coexposure caused a significant increase in the lipid peroxidation (68.49%). Because their coexposure induced lipid peroxidation was higher from that induced by either of the agents, it seems that there was appreciable interaction between them. The mycotoxin-pesticide induced increase in lipid peroxidation suggests generation of free radicals and subsequent oxidative stress mediated structural and functional changes in the hepatocytes.

T-2 toxin and endosulfan and their co-exposure decreased the GSH levels by 18.38\%, 27.85\% and 21.07\%, respectively. Similarly, earlier workers\(^{10}\) reported that feeding 30 ppm endosulfan to broiler chicks for 60 days caused significant decrease in the reduced glutathione in erythrocyte cell membrane. Moderate to significant decrease in the GSH levels have also been reported in T-2 toxin\(^{11}\) and T-2 toxin+endosulfan when given alone caused a numerical increase in the liver lipid peroxidation while coexposure caused a significant increase in the lipid peroxidation (68.49%). Because their coexposure induced lipid peroxidation was higher from that induced by either of the agents, it seems that there was appreciable interaction between them. The mycotoxin-pesticide induced increase in lipid peroxidation suggests generation of free radicals and subsequent oxidative stress mediated structural and functional changes in the hepatocytes.

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<table>
<thead>
<tr>
<th>Groups</th>
<th>MDA (TBARS)</th>
<th>Non-enzymatic antioxidant</th>
<th>Enzymatic antioxidants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mmol/g tissue</td>
<td>GSH</td>
<td>GPx</td>
</tr>
<tr>
<td>Control</td>
<td>443.18(^a)</td>
<td>577.24</td>
<td>627.20</td>
</tr>
<tr>
<td>T-2 toxin</td>
<td>523.48(^b)</td>
<td>471.16</td>
<td>592.29</td>
</tr>
<tr>
<td>(0.5 ppm)</td>
<td>±62.96</td>
<td>±48.22</td>
<td>±5.94</td>
</tr>
<tr>
<td>Endosulfan</td>
<td>706.78(^b)</td>
<td>416.46</td>
<td>605.02</td>
</tr>
<tr>
<td>(20 ppm)</td>
<td>±103.44</td>
<td>±53.51</td>
<td>±30.98</td>
</tr>
<tr>
<td>T-2 toxin +</td>
<td>746.53(^b)</td>
<td>455.60</td>
<td>608.13</td>
</tr>
<tr>
<td>endosulfan</td>
<td>±119.21</td>
<td>±60.22</td>
<td>±8.46</td>
</tr>
<tr>
<td>(0.5 ppm + 20 ppm)</td>
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</tbody>
</table>

Means bearing the same superscript within a column do not differ from each other (P>0.05); TBARS- mmol of MDA/g of tissue, GSH- μm CDNB-GSH conjugate formed/min/mg protein, GPX- μg of glutathione utilized/min/mg protein, SOD- Enzyme required to inhibit 50 per cent pyrogallol autooxidation/unit/g of tissue and CAT - μm of H₂O₂ decomposed/unit/g of tissue.

The results of the study induced indicated that T-2 toxin and endosulfan and their coexposure did not affect the values of GPx and SOD. However, significant increase in the SOD and GPx has been reported in broiler chicks fed 30 ppm endosulfan for 60 days\(^{10}\). Similar to the present observation, T-2 toxin did not alter the values of GPx in chicken fed 0.012 and 0.2 mg T-2 toxin/kg of feed for 10 days\(^{11}\), in broiler chicks fed 1.5 mg T-2 toxin/kg BW/day from 7 to 28 days of age\(^{13}\) and in broiler chickens fed with 0, 0.5, 1.5, 4.5 and 13.5 ppm of T-2 toxin for 17 days\(^{14}\). Significant increase in the levels of GST in the endosulfan group (21.03\%) and numerical increase in the T-2 toxin (15\%) and T-2 toxin+endosulfan (12.06\%) groups were observed. Similar observations were made in broiler chicks fed 30 ppm of endosulfan for 60 days\(^{10}\) and 1.5 mg T-2 toxin/kg BW/day from 7 to 28 days of age\(^{13}\).

The results of the study induced indicated that T-2 toxin and endosulfan combination caused considerable hepatic damage that could affect the health and performance of the broiler chicken.

REFERENCES


