Hormonal Changes Associated with Insecticide Exposure in Albino Rats

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ABSTRACT:
Insecticide an organic chemical used in agriculture, medicine, industries and household has bio-pharmacological effects such as inducing stress, hormonal imbalance and/or physiological effects and reproductive toxicity. This study therefore investigated the possible effects of exposure to insecticides on hormonal responses in rats. Albino rats were exposed to insecticides through drinking water for continues twenty one days and the following hormone concentrations determined using standard ELISA methods: thyrotrpin, total thyroid, total thyroxine, follicle stimulating, progesterone, prolactin and luteinizing. The results obtained showed that the concentration of thyrotrpin, total thyroid and follicle stimulating hormones significantly decreased (p<0.05) while total thyroxine, prolactin and progesterone concentrations significantly increased (p<0.05) compared to the control. The change in the concentration of luteinizing hormone was insignificant (p>0.05) in the group of rats exposed to insecticide when compared to the unexposed group. In conclusion, the exposure of rats to insecticides resulted in adverse changes in their hormonal concentrations

KEYWORDS: hormones, hormonal changes, insecticide exposure and albino rats

INTRODUCTION:
A hormone is a chemical substance produced by an endocrine gland, secreted into the blood and delivered to target organ on which it will act and elicit an appropriate physiological response (Crisp et al., 1998). The activities of hormones just like organs and tissues in the body can be altered which brings about changes in the activities of the hormones. Hormonal changes may be due to stress or as a result of induced toxicants like insecticides/pesticides (Shettler et al., 2003).

Insecticides are agricultural chemicals used for controlling pests on plants or animals. Problems associated with pesticide hazards to man and environment are not confined to the developing countries, but extended to developed nations and most countries are still facing some problems in certain locations (Nuckols et al., 2007). The severity of insecticide hazards is much pronounced in third world countries. A number of long persistent organochlorines, which have been banned or severely restricted are still marketed and used in many developing countries (Hajjo et al., 2007). The ideal insecticide is an insecticide which be effective only against the pests and be harmless to people, animals and environment. However, they have some side / non-target effects that may show undesired actions later (El-Kashoury et al., 2005).
A large number of chemicals occurring in our environment may have the potential to interfere with the endocrine system of animals (Dalsenter et al., 1997). Many of these chemicals can disrupt development of the endocrine system and of the organs that respond to endocrine signals in organisms indirectly exposed during prenatal and/or early postnatal life; effects of exposure during development are permanent and irreversible (Colborn et al., 1993). There is much concern that exposure to such environmental contaminants causes decreased sperm counts, impairment of sperm motility, reduced fertilization ability, producing abnormal sperm in men and wildlife (Alm et al., 1996). Pesticides have been shown to cause overproduction of reactive oxygen species (ROS) in both intra- and extracellular spaces, resulting in a decline of sperm count and infertility in wildlife and human (Sharpe and Skakkebaek, 1993). The antioxidant system plays an effective role in protecting testes and other biological tissues below a critical threshold of ROS thus preventing testicular dysfunction (Oschendorf, 1999). ROS has been shown to damage macromolecules, including membrane bound polyunsaturated fatty acid (PUFA), causing impairment of cellular function (Lenzi, 2000). For this reason, the present study was undertaken to evaluate the hormonal changes associated with the exposure of rats to insecticides.

MATERIALS AND METHODS:
Experimental Design:
Twenty adult albino rats purchased from the Veterinary department of the University of Nigeria Nsukka and acclimatized under laboratory conditions with food and water ad libitum for one week. The rats were allocated into two groups of ten rats and treated as follows:
- Group 1: Rats received tap water and food only throughout the experimental period (control group)
- Group 2: Rats were exposed to insecticide through drinking water for 21 days (experimental group)

The experimental work on rats were performed according to the guidance for care and use of laboratory animals (NRC, 1996).

Blood Sample Collection:
At the end of the experimental period (21 days), blood samples were collected from the retro-orbital venous plexus of anesthetized rats in centrifuge bottles for 8 minutes at a speed of 3000rpm. The clear serum was then obtained and stored at -20°C until used for hormonal assay.

Determination of Thyrotropin Concentration:
Thyrotropin concentration was determined using the method of Ekins (1993) as contained in the DS – EIA – THYROID – TSH kit (Immunospec Cooperation). The absorbance was measured at 450nm.

Determination of Total Thyroid Concentration:
Total thyroid concentration was determined using the method of Nelson and Wilcox (1996) as contained in the DS – EIA – THYROID – T3 – TOTAL kit (Immunospec Cooperation). The absorbance was read at 450nm.

Determination of Total Thyroxine Concentration:
This test was carried out using the method of Kozwich et al., 1991 as contained in the DS – EIA – THYROID – T3 – TOTAL kit (Immunospec Cooperation). The absorbance was read at 450nm.

Determination of Follicle Stimulating Hormone Concentration:
Follicle stimulating hormone concentration was determined using the method of Pierce and Pearsons (1981) as contained in the DS – EIA – GONADOTROPIN – FSH kit (Immunospec Cooperation). The absorbance was read at 450nm.

Determination of Progesterone Concentration:
This test was carried out using the method of Check (1995) as contained in the DS – EIA – STEROID – PROGESTERONE kit (Immunospec Cooperation). The absorbance was read at 450nm.

Determination of Prolactin Concentration:
Prolactin concentration was determined using the method of Hansen (1989) as contained in the DS – EIA – PROLACTIN kit (Immunospec Cooperation). The absorbance was read at 450nm.

Determination of Luteinizing Hormone Concentration:
Luteinizing hormone concentration was determined using the method of Uotila et al. (1981) as contained in the DS – EIA – GONADOTROPIN – LH kit (Immunospec Cooperation). The absorbance was read at 450nm.

Statistical Analysis:
Data obtained was analyzed using SPSS version 17.0 and the results represented as mean ± SD. Significant difference was performed using the student’s t-test at 95% confidence interval.

RESULTS:
The hormone immunoassay was conducted to show the changes in hormonal activity by comparing the means of exposed and unexposed control. Table 1 shows that the concentrations of thyrotropin, total thyroid and follicle stimulating hormones in the experimental group significantly decreased (p<0.05) compared to the control group while the concentrations of the hormones total thyroxine, prolactin and progesterone significantly increased (p<0.05) in the exposed group compared to the unexposed. There was no significant difference (p>0.05) in the concentration of luteinizing hormone of the experimental group compared to the control group.
Table 1: Changes in hormonal changes observed in the two groups of rats (exposed and unexposed)

<table>
<thead>
<tr>
<th>Hormones</th>
<th>Mean Change in Hormonal Concentration of the Exposed Group (mg/kg)</th>
<th>Mean Change in Hormonal Concentration of the Unexposed Group (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyrotropin</td>
<td>0.2 ± 0.001*</td>
<td>0.4 ± 0.007</td>
</tr>
<tr>
<td>Total thyroid</td>
<td>60 ± 1.22</td>
<td>160 ± 2.11</td>
</tr>
<tr>
<td>Total thyroxine</td>
<td>0.9 ± 0.005*</td>
<td>0.3 ± 0.001</td>
</tr>
<tr>
<td>Follicle stimulating hormone</td>
<td>6 ± 0.024*</td>
<td>10 ± 0.021</td>
</tr>
<tr>
<td>Progesterone</td>
<td>140 ± 3.33</td>
<td>60 ± 2.083</td>
</tr>
<tr>
<td>Prolactin</td>
<td>55 ± 1.341*</td>
<td>40 ± 1.121</td>
</tr>
<tr>
<td>Luteinizing</td>
<td>24 ± 1.201</td>
<td>25 ± 2.101</td>
</tr>
</tbody>
</table>

Values represent mean ± SD, n = 20
*Significant difference at p<0.05 (student’s t - test)

DISCUSSION:
Hormonal changes of human exposure to insecticides remain a worldwide concern due to the fact that these insecticides are mutagenically linked to the development of cancers (Leiss and Saltz, 1995). Also, insecticides have serious hazards in reducing fertility and in causing damage of reproductive organs of animals (Whorton et al., 1977). The significant decrease (p<0.05) in the concentrations of follicle stimulating hormone, total thyroid hormone and thyrotropin hormone in the rats exposed to insecticides confirms the findings of Desaulniers et al., 1999 and Lafuente et al., 2000 who investigated the toxicological influences of PCB (126 and 153) and methoxychlor at different concentrations in male rats. This may be due to the fact that many insecticides are able to block or activate the steroid hormone receptor and affect the concentrations of certain hormones (Vinggaard et al., 2000). Also in accordance with the findings of the present study, Hatz et al., 1993 proposed a mechanism for pesticide induced toxicity to be due to their ability to deplete stores of vitamin A and thyroid hormones from the body by 30 – 50% through interactions with a common plasma protein carrier called transthyretin (TTR). Another support to the interaction of pesticides with TTRs was established by Van den Brey et al., 1991 who mentioned that hydroxylated PCBs and a number of halogenated industrial chemicals, mainly pesticides may decrease thyroid hormone levels in rats through interference with hormone transport carriers (TTRs). The decrease in the concentration of FSH observed in this study may be due to an elevation in circulating levels of inhibin, a glycoprotein of primarily serotol cell origin which inhibits FSH synthesis and secretion of pituitary (Caroll et al., 1991).

Organochlorines pesticides have some estrogenic properties and may modify the feedback mechanism of steroids on the hypothalamus and pituitary (Lafuente et al., 2000). Exogenous estrogens (natural and synthetic) elicit all the pharmacological responses usually produced by endogenous estrogens. The present study revealed a significant increase (p<0.05) in prolactin and progesterone in the experimental group treated with insecticide. This result is similar to the work of Tag El – Din et al., 2003 who suggested that pesticides mimic estrogenic activity which may have a direct effect on the testes or indirectly through the hypothalohypophyseal testicular axis or by desensitizing the testes to gonadotropins. Since many pesticides have been shown to block or activate the steroid hormone receptors, this study emphasizes the importance of screening pesticides for a wide range of hormone mimicking effects.

Luteinizing hormone is a glycoprotein released from the anterior pituitary which stimulates testosterone production by Leydig cells of the testes in males. The result of this study revealed an insignificant difference (p>0.05) in the luteinizing hormone concentration of the exposed rats when compared to the unexposed. It was proved that certain pesticides did not alter luteinizing levels when administered into rats at different doses for short – term intervals such as TCPM (Foster et al., 1999), DDT (Krause, 1977) and PCB 28 and 77 (Desaulniers et al., 1997). This study was conducted within a short –term (21 days) which may explain the insignificant difference observed.

The overall findings of the present study support the fact that there are hormonal changes associated with insecticide exposure in rats. We therefore strongly suggest that insecticides should be evaluated for toxic hazards even recommended as 'safe' to prevent certain effects on its users.

REFERENCES:


