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ACUTE NOISE-INDUCED ALTERATIONS IN THE IMMUNE STATUS OF ALBINO RATS

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Abstract : The effect of acute noise induced changes on the immune functions of albino rats was studied. Cell mediated immunity was assessed by Leukocyte migration inhibition index (LMI) and humoral immunity by estimating antibody titre. The organ weight of spleen, thymus, adrenal and lymph node was noted, the cell count of spleen and thymus was enumerated and plasma corticosterone level was estimated. A significant increase in the plasma corticosterone level, thymus weight and cell count along with significant decrease in the antibody titre, spleen weight and cell count was observed in noise stressed animals. No significant changes were observed in the LMI and organ weight of adrenal and lymph node in these animals. Our study shows acute noise to be a potent stressor causing definite alterations in the immune functions of the albino rats.

Key words : acute noise corticosterone immunity

INTRODUCTION

Noise is the most commonly encountered stressor in today's environment. Extensive studies have been done on the extra auditory effects of noise. If this noise exposure is temporary, the body system usually returns to a normal state within minutes. If the noise stimulation is sustained or consistently repeated, persistent changes may develop in the neurosensory, endocrine, (1) and digestive systems (2) and also leads to developmental abnormalities (3).

Though it is clear that noise affects the different systems of our body in various ways, very little literature evidence exists on the specific effect of noise stress on the immune system. An increase in the serum IgG levels and an increase in the B and T lymphocytes, were found in workers exposed to industrial noise of 88–96dB (4). However, suppression of *in-vitro* activation of B cells in mice exposed to sound stress (5) has also been reported. As previous studies have thrown up contradictory results and no clear conclusion could be drawn so far based on these reports, our main objective of this investigation was to study the changes in the immune function in albino rats after acute noise stress exposure.

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METHODS

Wistar strain male albino rats weighing 150–200 g were used for the study. Two groups of animals comprising of 15 animals each were used. The animals were maintained under standard laboratory conditions and provided with food and water *ad libitum*. Group 1 were control animals. Group 2 animals were subjected to broad band noise at 100dB intensity for 4 hours and sacrificed after 30 min. The sacrificial time was kept constant between 8.00–9.00 a.m. to avoid variations due to circadian rhythm.

Noise stress procedure:

Broad band noise (White noise) was produced by a white noise generator and amplified by an amplifier which was connected to a loudspeaker located 30 cm above the animal cage. The intensity of the sound was measured by a sound level meter (Cygnet-D 2023) and maintained at 100dB intensity. The background noise level inside the room was 42 ± 3 dB.

The animals were anaesthetised with ether and blood was collected from jugular vein to obtain stress free samples (6). The plasma was used for the estimation of the corticosterone level by spectrofluorimetric method (7). Humoral immunity was assessed by the antibody titre level. The serum was separated and the titre was estimated by the direct haemagglutination method. The titre value was expressed as mean log2 value. The spleen, thymus, adrenal and lymphnode were removed and the organ weight was noted and expressed with respect to body weight. The organs spleen and thymus were teased separately in Minimum Essential Medium (AT 045, Hi Media) to give a single cell suspension, made upto a known volume and the cell count was done (8). The cell mediated immunity was assessed by the Leukocyte migration inhibition (LMI) test (9). Sensitized T lymphocytes in the presence of the corresponding antigen produce migration inhibition factor (MIF) which inhibit the migration of leukocytes from capillary tubes. Immunodefecient lymphocytes fail to release MIF. The migration inhibition system has also been used to study the various tissue antigens that stimulate the sensitized lymphocytes to produce mediator substances. Thymocytes were used as a migrating population to study the effect of migration inhibition factor released by the lymphocytes of spleen in the presence of sheep red blood cells.

Statistical analysis was done using Student's 't' test. $P<0.05$ was considered statistically significant.

RESULTS

The plasma corticosterone level was significantly increased ($P<0.001$) in the acute noise stress group. No variation was observed in the organ weights of the lymph node and adrenal in the stress group (Table I).

TABLE I: Plasma corticosterone and organ weight of spleen, thymus, adrenal and lymph node in acute noise stress.

Parameter	*Control*	*Noise stress*
Corticosterone (µg/dl)	38.8±4.7	98.9±4.6*
Organ Weight Body Weight of Spleen	4.5±0.2	2.5±0.3*
Thymus	0.9±0.08	1.7±0.2*
Adrenal	0.21±0.01	0.20±0.01
Lymph node	0.20±0.02	0.19±0.002

Values are expressed as mean ± SD
Significance *P<0.001.

The antibody titre was decreased ($P<0.001$) in the stress group when compared with the control animals. No difference was noted in the LMI index of the stressed animals. A significant increase ($P<0.001$) in the thymus weight and count and a significant decrease ($P<0.001$) in the spleen weight and count was observed in the animals subjected to noise stress (Table II).

TABLE II: LMI, Antibody titre and Cell count of Spleen and Thymus in acute noise stress.

Parameter	*Control*	*Noise stress*
Antibody titre	1.4±0.5	0.1±0.3*
LMI Index	0.87±0.06	0.9±0.06
Spleen count ($x10^8$)	4.7±0.3	2.4±0.5*
Thymus count ($x10^8$)	4.2±0.3	7.4±0.7*

Values are expressed as mean ± SD
Significance *$P<0.001$.

DISCUSSION

The significant elevation in the plasma corticosterone level observed in the acute noise stress group could be due to the norepinephrine acting at CRF neurons in the paraventricular nucleus to directly stimulate CRF release and subsequently ACTH and corticosterone secretion, in noise exposure (10). Thus our findings are in agreement with the previous study of Sandi et al (11) who have shown enhanced ACTH and corticosterone levels in rats exposed to noise stress.

A reduction in the antibody titre observed in these acute noise stressed animals was well in agreement with previous reports (12). Studies (5) have shown that *in-vitro* activation of B cells was suppressed in mice exposed to sound stress for 20 days, but animals exposed over extended periods had enhanced B cell activity. In the present study, the decrease in the antibody titre could be due to the increased clearance of the non-specific circulating antibodies by the reticuloendothelial system, because in these animals the phagocytic activity of neutrophils was significantly increased (in Press). Increased phagocytic index was also reported in animals exposed to noise stress for twenty one days (13). No difference was noted in the LMI index of the stressed animals. The release of migration stimulation factor (MSF) from the T suppressor cells and migration inhibition factor (MIF) from the T-helper cells was observed in a mixed cell population obtained from the spleen of mice (14). A normal migration depends on the balance of these two opposing factors. Probably these factors could not have altered by this level of noise stress and duration.

No variation in the organ weights of the adrenal and lymph node was observed in the noise stress group. In spite of the increase in the corticosterone level no appreciable changes observed in the organ weight might be due to the duration of four hours which may not have been sufficient to cause hypertrophy or hyperplasia and this is well in agreement with previous reports (15).

The thymus weight and the thymus count were significantly increased in the noise stress group and it is in agreement with the studies of Folch et al (16) who have shown that noise stress produces an increase in the weight of the thymus and thymocyte number. A significant reduction

in the spleen weight and spleen count was noted in the animals subjected to noise stress. In any stress, the activation of sympathetic nervous system alongwith adrenocortical system is well known. In lower animals, spleen is a blood reservoir and sympathetically induced splanchnic vasoconstriction is known to shift the blood to peripheral circulation and this could be the possible cause for decrease in spleen cell count and weight observed.

To conclude, our study has clearly shown, acute noise to be a potent stressor producing definite alterations in the immune system. The alteration seen may be due to the resultant autonomic stimulation and change in the neuronal, humoral and hormonal factors. However, whether these change are reversible and adaptation is possible in chronic stress or further alteration will occur in immune system can be answered only with further in depth study.

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