Changes in Plasma Corticosteroid Concentrations of Rats Following Exposure to Noise

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(Received January 14, 1969)

In previous papers[1] We reported on the decreased concentrations of adrenal ascorbic acid (AAA) in rats after short peroids of exposure to noise and that the decreased levels of AAA returned to normal approximately six hours after the end of exposure to noise. Eosinipil counts in the perpheral blood of rats were also decreased by short exposure to noise.[2]

In our last paper[3] we reported that the AAA concentrations of rats were decreased by short exposure to noise but that repeated exposure to noise for periods of 10, 20 and 30 days did not decrease the concentrations of AAA, indicating the possibility of adaptation to noise through certain physiological responses to noise.

Observations from our previous experiments indicated that ACTH release from the anterior pituitary lobe is increased in response to noise and that the adrenal cortex is stimulated by the increased plasma ACTH. However, the concentration of AAA is an indirect indicator for estimating ACTH release. Therefore, in an attempt to observe more precisely the change in ACTH release as a result of exposure to noise, experiments were designed to measure the plasma corticosteroid concentrations in rats after exposure to noise.

MATERIALS AND METHODS

Experiments were designed to measure plasma corticosteroid concentrations following exposure to noise for both a short period and after regular repeated exposure over more extended periods of time.

Male rats of the Wistar strain weighing approximately 200g apiece were used.

Experiment I: Short exposure to noise.

In this experiment there were 51 rats in the control group and 50 rats in the experimental group.

The rats were exposed for 25 minutes to noise of 1,000 cps, 100 $\pm$ 5 db.

30 minutes after the end of the noise exposure, the rats were sacrificed and the plasma corticosteroid concentrations were determined.

Experiment II: Regular repeated exposure to noise for periods of 10 days, 20 days and 30 days.

In this experiment rats were exposed to noise of 1,000 cps, 100 ± 5 db. for 2 hours a day for periods of 10 days, 20 days and 30 days.

There were 50 rats in the control group for this experiment. There were 15 rats in the group which underwent exposure for 10 days and 20 rats which underwent exposure for 20 days. The rats which underwent exposure for 30 days were sub-divided into 2 groups: Group A, which had 15 rats and Group B which had 18 rats.

In the case of the groups which underwent exposure for 10 days and 20 days, the rats were sacrificed 30 minutes after the final noise exposure for each group and the plasma corticosteroid concentrations were determined. In the case of the rats which underwent exposure for 30 days, those in Group A were sacrificed 30 minutes after the final noise exposure and the plasma corticosteroid concentrations determined and those in Group B were sacrificed 12 hours after the final noise exposure and the plasma corticosteroid concentrations determined.

In the case of the control group, an appropriate number was sacrificed each time those in an experimental group were sacrificed and their plasma corticosteroid concentrations determined.

The rats were sacrificed by decapitation without anesthesia and blood from the trunks was collected in heparinized beakers. The blood samples were centrifuged and the separated plasma was treated according to the procedure of Steenburg and Thomasson.[4)]

Finally, corticosteroids in chloroform were extracted directly into the fluorescence reagent (sulphuric acid reagent). The acid extract was transferred to a quartz cell and read in an Aminco-Bowman spectrophotofluorometer at an activation wave length of 467 mμ and a fluorescence setting of 525 mμ.

RESULTS

Experiment I:

Basal or resting corticosteroid concentrations were determined from blood samples of the 51 control rats. The mean value of the concentrations was 8.25 ± 0.58 μg/100 ml (see Table 1).

The mean value of the plasma corticosteroid concentrations of the experimental rats was 20.50 ± 1.61 μg/100 ml (see Table 1), a significant increase (Pr. <0.01) over the control group.

Tab!e 1. Plasma corticosteroid concentrations of rats 30 minutes after exposure to noise one period of 25 minutes.

	Number of Rats	Mean (μg./100 ml.)	Standard Deviation	Standard Error
Control	51	8.25	4.12	0.58
Noise Exposure	50	20.58**	11.40	1.61

**: Significant increase (Pr.$<$0.01)

Experiment II:

For the control group the mean value of basal concentrations was 8.40 $\pm$ 0.65 μg/100 ml.

For the rats in the 10 day exposure group the mean value of the plasma corticosteroid concentrations was 12.87 $\pm$ 1.99 μg/100 ml, a significant increase (Pr. $<$0.05) over the control group.

For the rats in the 20 day exposure group the mean value of the plasma corticosteroid concentrations was 9.43 $\pm$ 0.77 μg/100 ml.

For Group A of the rats in the 30 day exposure group the mean value of the plasma corticosteroid concentrations was 8.03 $\pm$ 0.64 μg/100 ml, and for Group B (sacrificed 12 hours after final noise exposure) the mean value of the plasma corticosteroid concentrations was 7.11 $\pm$ 0.55 μg/100 ml (see Table 2).

Table 2. Plasma corticosteroid concentrations of rats after extended exposure to noise.

			Number of Rats	Mean (μg./100 ml.)	Standard Deviation	Standard Error
Control			50	8.40	4.63	0.65
Noise Exposure	10 days		15	12.87*	7.70	1.99
	20 days		20	9.43	3.43	0.77
	30 days	A	15	8.03	2.48	0.64
		B	18	7.11	2.34	0.55

*: Significant increase (Pr.$<$0.05)
A: Sacrificed 30 minutes after final noise exposure.
B: Sacrificed 12 hours after final noise exposure.

Thus, the rats in the 20 day exposure group showed no significant increase and both groups exposed for 30 days showed slight decreases in comaprison to the control group.

DISCUSSION

In our previous papers we reported that the concentration of AAA in rats was decreased by exposure to noise for a short period of time[1] but the concentration of AAA of rats exposed for extended periods of time either remained relatively unchanged or else showed an increase.[3] These findings may be interpreted as indicating an adaptation to noise by the rats.

From reports of various other investigations it is presumed that the decrease in AAA concentrations indicates an increased concentration of plasma ACTH and the return to normal levels of AAA suggests a restortion of the ACTH level.[5][6]

Accordingly, it seems reasonalbe to assume that an increased release of ACTH from the anterior pituitary lobe occurs prior to the decrease of AAA in response to a short exposure to noise but in the case of extended exposure to noise there will be no appreciable increase in ACTH release.

Although the concentration of AAA is an excellent index for the estimation of ACTH release, it is indirect. Therefore, in an attempt to study more precisely the relationship between ACTH release and noise we carried out experiments to determine the plasma corticosteroid levels of rats following exposure to noise for various periods of time. The data of Table 1 and Table 2 show that the plasma corticosteroid concentrations were significantly increased by a single exposure to noise and after regular repeated exposure to noise for a period of 10 days there was still a significant increase in the plasma corticosteroid concentrations. After 20 days of regular repeated exposure to noise there was only a slight increase in the concentrations of the plasma corticosteroid concentrations, but the increase was not sufficient to be considered significant. After 30 days of exposure to noise there was a slight, but not significant decrease in the plasma corticosteroid concentrations. However, this decrease is the subject of further study.

According to Sayers et al.,[5] the administration of pure ACTH diminishes both the ascorbic acid and cholesterol contents of the adrenal glands. Further, Guillemin et al.[6] reported that stress and the administration of ACTH elevate the plasma corticosteroid concentrations and lower the level of AAA.

These reports and our findings indicate that a single exposure to noise or regular repeated exposure to noise for periods of up to 10 days results in an elevation of ACTHrelease from the anterior pituitary lobe which results in an elevation of corticosteroid concentrations. As noted previously, the slight decrease in plasma corticosteroid concentrations for the groups which underwent noise exposure for 30 days is still under study.

SUMMARY

In an attempt to observe the response of the hypothalamo-hypophyseal adrenal axis to noise, the plasma corticosteroid concentrations of rats were determined

after various periods of exposure to noise.

A single exposure and regulai repeated exposure to noise for 10 days resulted in significant increases in the plasma corticosteroid concentrations ; regular repeated exposure to noise for 20 days resulted in a slight but not significant increase. Regular repeated exposure to noise for 30 days resulted in a slight but not significant decrease in the plasma corticosteroid concentrations. This decrease is the subject of further study.

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