Mercuric chloride influence on active-avoidance acquisition in rats

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Sublethal doses of mercuric chloride were administered for 14 days into 25-day-old female rats. Although there was no impairment in either general appearance (body weight, food intake, etc.) or escape behavior, mercury poisoning produced significant deficits in active-avoidance learning. Results indicate that behavioral impairments can occur as a consequence of low doses of mercury administration in the absence of gross physical abnormalities.

Considerable evidence (see Putman & Madden, 1972) indicates that high amounts of mercury in various forms (inorganic as well as organic mercury) can lead to tremors, motor disability, intellectual impairment, blindness, deafness, and loss of muscle control. All of these symptoms indicate the susceptibility of the nervous system to mercury poisoning. Until recently, however, the physiological and psychological effects of low doses of mercury have gone unnoticed.

An experiment by Spyker, Sparber, & Goldberg (1972) demonstrated that small amounts of mercury could cause subtle behavioral changes without causing any gross physiological changes. They injected .16 mg/20 g body weight of methyl mercury decyandramide into pregnant mice and found no difference between groups in terms of general appearance, body weight, or amount of brain protein, choline acetyltransferase level, or cholinesterase activity. However, although the offspring of these mice appeared normal, they were significantly more emotional in an open field than the offspring of nontreated mice. In addition, mercury treatment disrupted normal swimming patterns in mice.

The purpose of the present experiment was to evaluate the effect of low doses of inorganic mercury on the acquisition of an active-avoidance response. Learning deficits in animals exposed to continuous low doses of inorganic mercury would indicate behavioral impairment due to the effects of mercury in organisms not exhibiting gross physiological deficits.

METHOD

Subjects

Twenty-nine experimentally naive female rats, purchased from Sprague-Dawley, Inc. (Madison, Wis.), served as Ss in this experiment. All rats were 25 days old at the beginning of the experiment.

Apparatus

The apparatus was a two-chamber black and white avoidance apparatus, identical to that previously described by Klein & Spear (1969). A 1.6-mA footshock was used during active-avoidance training.

Procedure

All animals were assigned to one of three groups. Ss in Group I (N = 6) received a high dosage of mercuric chloride (7 mg/kg) daily for 2 weeks prior to active-avoidance training. Group II rats (N = 5) received a low dosage of mercuric chloride (5 mg/kg) for 2 weeks before training, while Ss in Group III (N = 18) received no mercuric chloride prior to active-avoidance acquisition. The lethal dose of mercuric chloride is 37 mg/kg.¹ Individual weights of each S were taken every 4 days and dosages altered according to weight gain.

Crystalline mercuric chloride was administered to each S daily, mixed with approximately 10 g dry food mixed with water and ¼ tsp of sugar. This was the only food given to insure that all of the mercuric chloride was ingested. The controls received only the mash and sugar. Water was presented ad lib to each S.

All Ss were given one-way active avoidance to a criterion of five successive avoidances. In order to avoid shock, the rat had to cross from the white chamber into the black chamber before the 5-sec CS-US interval elapsed. In an avoidance procedure similar to that described in Klein & Spear (1969), rats received escapable but not avoidance footshock treatment during the first two trials, with an avoidable shock condition instituted on the third trial. The rat was detained in the black chamber approximately 5 sec after trial termination and spent the 30-sec intertrial interval in a holding cage.

RESULTS

As in the Spyker et al study, there were no significant differences between treated and nontreated rats in terms of either general appearance or body weight. All rats weighed between 60 and 75 g at the beginning of the

Fig. 1. Mean number of trials to learn the active-avoidance responses as a function of dose level of mercury administered.

Fig. 2. Mean amount of shock received during active-avoidance acquisition for experimental and control-group animals.

experiment. Both treated and nontreated Ss weighed between 95 and 110 g at the end of the 2 weeks of mercury administration.

Figure 1 presents mean trials to learn the active avoidance for experimental and control rats. An analysis of variance showed a significant group effect ($F = 3.82$, $df = 2/26$, $p < .05$). A modified Tukey test for unequal Ns showed that the two experimental groups did not differ significantly in terms of number of trials to acquire the active-avoidance response. The control-group rats learned the active-avoidance response in significantly fewer trials than did Ss in either experimental group.

Analysis of total shock received during acquisition (see Fig. 2) indicated a significant effect of experimental manipulation ($F = 9.11$, $df = 2/26$, $p < .01$). A modified

Tukey test demonstrated that both experimental groups received more shock during acquisition than did control-group rats. This difference in amount of shock received during training was not due to performance differences between groups. An additional analysis of variance conducted on mean escape latencies indicated no significant differences in escape latencies between experimental and control-group rats. It would appear that mercuric chloride administration significantly impaired active-avoidance acquisition without influencing either escape latencies or general physiological condition (as indicated by identical weights).

DISCUSSION

In replication of Spyker et al, the experimental results indicate that sublethal amounts of mercury can cause subtle behavioral impairments. In addition, prolonged exposure to mercuric chloride produced detrimental effects like those due to methyl mercury poisoning. These results should provide alarm, as most indexes of harmful pollution effects are physiological in nature and it is possible that portions of our human population are being exposed to low mercury pollution. Further research is obviously necessary to delineate magnitude of psychological impairment to mercury poisoning in a number of experimental situations.

REFERENCES

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NOTE

1. Lethal dose of orally injected mercuric chloride in rats is 37 mg/kg Merck Index, 8th ed., page 659.