being significantly more reactive than controls at 7, 14, and 21 days post exposure. Thirty days after exposure all control animals passively avoided after a single training trial (1 mA shock for 1 sec). In contrast, there was high variability in the response latencies of treated rats on *both* training and testing trials. Histological analysis of these rats 35–40 days after exposure showed a consistent pattern of severe neuronal degeneration and necrosis in the hippocampus, amygdala, pyriform cortex, and septum of treated rats; no control rat showed any neuropathology. Thus, the behavioral measures of reactivity to handling, reactivity to novel environments, return to pre-injection body weight, and overt acute symptoms are indicative of neuropathology in rats that otherwise appear to be fully recovered from soman poisoning.

DRL-20 DEFICITS IN SURVIVORS OF THE NERVE AGENT SOMAN. Robert F. Smith, Department of Psychology, George Mason University, Fairfax, VA 22030 and John H. McDonough and Catherine D. Smith, U. S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, MD 21010.

A near-lethal dose of the nerve agent soman produces diffuse neuropathy and punctate lesions, particularly prominent in limbic system. To determine whether soman also produces lasting behavioral changes, 45 male Edgewood rats (Sprague-Dawley derived) were first pretrained to bar press for liquid reinforcement and then dosed with 110 micrograms/kg soman (24 animals) or saline (21 animals). Survivors were then reshaped to bar press, given three days of continuous reinforcement, and 45 days of DRL-20 sec training. Six of 15 soman survivors could not be reshaped, but all controls reshaped ($p=0.003$, Fisher's exact test). ANOVA revealed that soman treated animals received significantly fewer reinforcements on the DRL schedule; on the final day of testing, the controls received a mean of 44.0 reinforcements, while the soman group received a mean of 20.3. These data indicate that near-lethal soman administration in rats produces behavioral deficits lasting at least 75 days after dosing.

SELECTIVE EFFECTS OF NEONATAL EXPOSURE TO CADMIUM CHLORIDE ON ADULT OPERANT BEHAVIOR IN RATS. M. C. Newland, W. W. Ng, R. K. Miller, R. B. Baggs and B. Weiss. Divisions of Toxicology and Laboratory Animal Medicine, Departments of Radiation Biology and Biophysics, Obstetrics/Gynecology, and Pharmacology, University of Rochester, Rochester, NY.

In Long Evans rats, neonatal exposure to cadmium chloride (0, 1, 3 and 6 mg/kg, SC on postnatal day 1) resulted in short and long term behavioral, neurological, and morphological alterations. Dysfunctions in several preweanling assessments, which included suckling, location of home bedding, cliff avoidance, and gait, were associated with hydrocephalus. Alterations in the acquisition and maintenance of fixed-ratio (FR) 75 performance were noted in non-hydrocephalic adult males which performed normally on the preweanling assessments. These alterations in operant behavior were also unrelated to body weight changes. The effect on FR performance was related to dose in an inverted U manner: overall rates increased at 3 mg/kg and decreased at 6 mg/kg. Post-reinforcement pausing was unaffected at these doses. The highest dose of cadmium eliminated the cohesiveness of FR performance by disrupting the running rate while having no effect on post-reinforcement pausing. Thus, selective changes in adult operant behavior were seen in animals which appeared normal as determined by the preweanling assessments. (Funded by ES 01248 and ES 07026).

PRE- PLUS POST-NATAL METHYLMERCURY EXPOSURE IN MONKEYS PRODUCES DEFICITS IN SPATAIL VISION. Deborah C. Rice and Steven G. Gilbert. Toxicology Research Division, Bureau of Chemical Safety, Health and Welfare Canada.

Monkeys (*Macaca fascicularis*) were exposed to methylmercury during the entire period of gestation; from birth to four years of age they received the same dose that their mothers had received. Immediately thereafter, spatial visual function was determined using a forced-choice psychophysical procedure. The monkey faced two oscilloscopes, one displaying a vertical sine wave grating, and the other displaying a blank field of equal average luminance. The monkey was required to press the button corresponding to the oscilloscope displaying the grating in order to be reinforced. For each of a number of spatial frequencies, the contrast at which the monkey responded with 70% correct choices was determined and considered the threshold for that frequency. Three of six treated monkeys exhibited impaired spatial visual function relative to controls under both high and low luminance conditions.

THE ROLE OF ATTENTIONAL PROCESSES IN POSTNATAL LEAD NEUROTOXICITY. Lloyd Hastings and True-Jenn Sun. Department of Environmental Health, University of Cincinnati, College of Medicine, Cincinnati, OH 45267.

The objective of the present study was to investigate the effects of early exposure to low-levels of lead on attentional processes using a rat model. Attentional processes were assessed by looking at two tasks involving the blocking paradigm. Rat pups were exposed to 0 or 1090 ppm (0.2%) lead acetate via dams' milk and weaned onto the same solution as their dams had received. Flinch-jump thresholds were obtained on day 91. The rats were then evaluated using a two-way avoidance task, followed by testing on a conditioned suppression task. No differences were found in flinch-jump thresholds. Analysis of the avoidance task data revealed that there was neither a significant lead effect, nor a significant blocking effect. Analysis of the suppression ratios from the conditioned suppression task revealed that while there was not a significant lead effect, there was a significant blocking effect for both groups.

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