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Prenatal and Early Postnatal Effects of Lead Ions on Motor Activity According to Gender Differences in Rats

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ABSTRACT

BACKGROUND. Lead exposure in the early stages of pregnancy and the prenatal period is extremely dangerous and could cause substantial health problems in the future. The majority of epidemiological studies have considered gender as a confounding factor when evaluating the neurodevelopmental toxicity of lead exposure. This paper examines the effects of prenatal lead exposure on locomotor activity in relation to motor cortex morphology. The results are discussed in female and male rats.

OBJECTIVES. Offspring of Wistar rats that, during pregnancy, received lead acetate at a) 0%, b) 0.05%, c) 0.2% with water were used for experiments.

METHODS. Blood lead levels were analyzed by atomic absorption spectrophotometry. The overall degree of locomotor activity was evaluated using Calvin S. Hall's Open Field Test. Histological analysis of motor cortex neuronal density was conducted using a light microscope.

RESULTS. During the critical days of neurogenesis (PD7, PD14, PD21, PD28), a statistically significant decrease in the number of neurons in the motor cortex of experimental pups was observed compared with the control group. Additionally, there was a statistically significant difference even at low doses. The second series of experiments was conducted on PD61 offspring or young adult rats. Behavioral tests showed significant differences between female and male rats. Male rats exposed prenatally to lead ions showed reduced locomotor activity, as measured by the number of lines crossed in the open field test; in the high-dose group, this difference was statistically significant. Unlike male rats, female rats showed increased motor activity. There was a trend toward decreased motor cortex neurons in offspring of both sexes at both low and high doses, but the decrease was statistically significant in male rats at the high dose.

CONCLUSIONS. Our results show that lead exposure during pregnancy significantly reduces the density of motor cortex neurons. However, this effect is gender-dependent: it was statistically significant in male offspring and later expressed in their motor activity. We can speculate that the motor area of the brain of developing male rats is more sensitive to lead ion exposure than that of female rats.

KEYWORDS. Female rat; Lead prenatal exposure; Locomotor activity; Male rat; Motor cortex

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BACKGROUND

It has long been known that lead is a neurotoxicant that impairs brain development and, as a result, impairs function. Therefore, exposure to lead remains a major public health problem, particularly in the modern world, where lead ions can enter the body from a variety of sources.

Lead exposure in the early stages of pregnancy and the prenatal period is extremely dangerous and could cause substantial health problems in the future. [1-4] By decreasing brain plasticity, lead acts as a neurotoxic agent and affects neural stem cells (NSCs) at a crucial stage of neurodevelopment. [5,6] The placenta and the developing fetal blood–brain barrier (BBB) allow lead to pass freely. [7]

Through processes such as synaptic pruning and trimming, neuronal migration, and the development of neuron-glia contacts, lead exposure can affect brain development and contribute to a range of neurological illnesses, including Alzheimer's disease (AD) and Parkinson's disease (PD). [6] A lower mental development index (MDI) at age two has also been linked to prenatal lead exposure, according to studies. In another study, children who were exposed to lead before age seven had lower IQ scores between ages eleven and thirteen. [8] Lead has been shown to impact NSC proliferation in both in vitro and in vivo studies. A study on rats exposed to lead revealed that dendrites in NSCs that developed into neurons had changed form. Lead alters nitric oxide synthase activity, which affects the brain vasculature and, in turn, the

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serotonergic system. This can cause increased aggressive behavior along with depression. [6]

Lead exposure during brain development can cause long-lasting alterations in gene expression, which may contribute to the emergence of adult neurological problems, according to a study on prenatal lead exposure. The detrimental effects of early-life lead exposure on behavior, cognitive functions, and brain networks further underscore its long-term impacts. [4]

Some studies have examined the relationship between lead exposure and neuronal changes in motor areas, but these studies have mainly focused on chronic exposure during childhood. [9]

Despite active research into the effects of lead exposure, the issue remains relevant because there is significant conflicting data. The majority of epidemiological studies have considered gender as a confounding factor when evaluating the neurodevelopmental toxicity of lead exposure, despite reports of differences between the sexes in terms of exposure patterns, chemical absorption in the gastrointestinal tract, metabolic traits, and detoxification capacity. [10-12]

The disparities in lead exposure across genders have been the subject of several articles. Chronic lead exposure in postnatal rats has been demonstrated to cause more serious health effects in both sexes, such as hypertension, anxiety, and reactive astrogliosis. However, whereas females are more prone to chemoreflex hypersensitivity, males have more behavioral, cognitive, and respiratory abnormalities. On the other hand, both sexes experience reactive astrogliosis and hypertension when exposed to lead intermittently. Males exhibit more reactive astrocytes in the hippocampus, while females are more vulnerable to chemoreflex hypersensitivity, elevated respiratory rate, and cognitive impairment. [13-15]

There is no clear and consistent picture of the consequences of prenatal lead exposure on different sexes, even though gender has been studied in many animal studies as a modifier of the effects of prenatal lead exposure. Males but not females in adult mice exposed to lead from day 8 of gestation to day 21 postnatally showed extremely hostile behavior toward their progeny. [16] According to exploratory behavior analysis, the same study found that females had higher levels of anxiety than males. [16]

While female rats increased their time in the pool during a forced swim test, [17] studies have shown that perinatal exposure in adult male rats increased emotionality. This may be an adaptive outcome, but it is linked to neural circuits underlying neurobehavioral disorders, such as depression. [18]

Only males with blood lead levels ranging from 15.38 to 28.97 µg/dL at weaning exhibited associative memory deficits when tested using a trace fear conditioning paradigm in research using Long Evans (LE) rats exposed perinatally (during gestation/lactation) to lead. [19]

Male rats did not exhibit impaired reference memory in the Morris water maze, but female rats exposed to lead during pregnancy did (hippocampal lead levels were tested at 1.73±0.19 µg/g wet weight on day 21). [20]

Therefore, it may be necessary to examine the sex-specific consequences of lead exposure in relation to lead-induced alterations of brain circuits involved in the typical sexually dimorphic expression of behavioral and cognitive functions, to gain a deeper understanding of the neurodevelopmental consequences of lead's sex-specific effects.

This paper examines the effects of prenatal lead exposure on locomotor activity in relation to motor cortex morphology. The results are discussed in female and male animals.

METHODS

Animals

Wistar rats were used for experiments. There were two series of experiments (**FIG.1**). The experimental animals received a low dose of lead (0.05% lead acetate in drinking water) in the first series and a high dose (0.2% lead acetate in drinking water) in the second (lead acetate trihydrate was purchased from Sigma-Aldrich Cat. N 107375).

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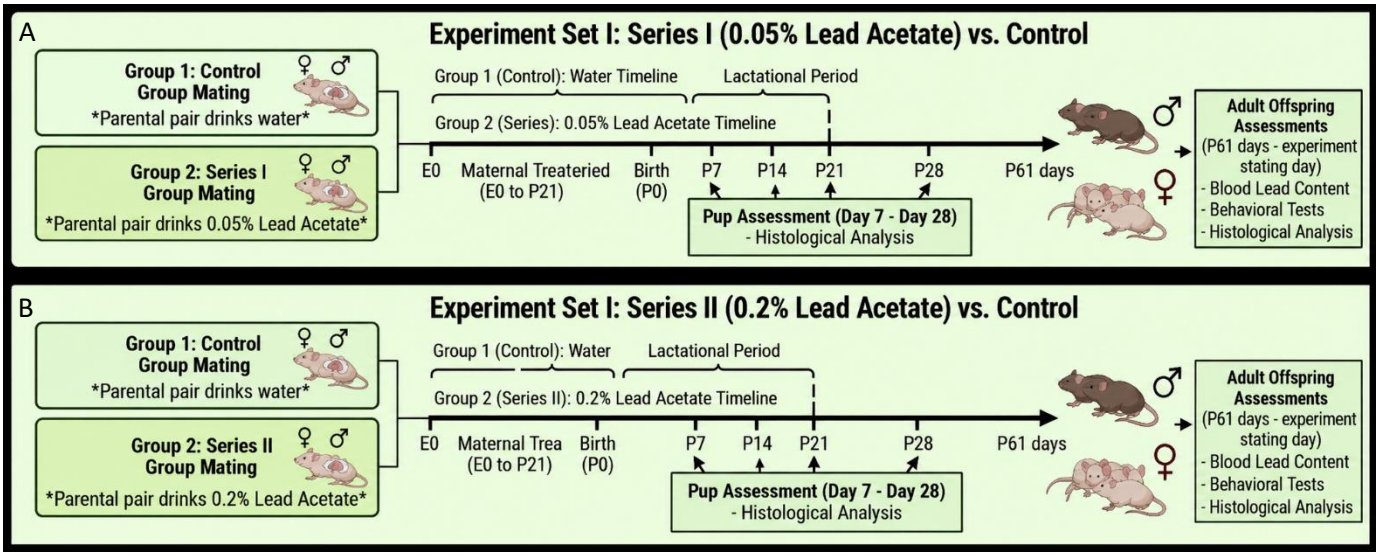
FIGURE 1. Experimental design



Explanations: A. The experimental animals received a low dose of lead (0.05% lead acetate in drinking water); B. The experimental animals received a high dose of lead (0.2% lead acetate in drinking water).

The control groups in each series received ordinary drinking water. Every animal had unrestricted access to food and water. Once a week, body weight and fluid intake were recorded. Pregnant rats were kept on the same drinking solution until the delivery of their young and housed in separate cages with wood shavings for nesting.

For each series of experiments, 6 families were assigned to the control group and 6 to the experimental group. There were two female rats and one male rat in each cage. We received approximately 9-12 pups from each family.

6-6 newborns were randomly collected from different cages each day for morphological analysis performed on days P7, P14, P21, and P28. For P61, 16-16 females and 16-16 males from the control and Experimental groups underwent behavioral and histological analyses.

Blood lead content

Blood samples from pups were analyzed for lead content using atomic absorption spectrophotometry as part of the Trace Metals Analysis. The mean blood lead levels for each litter and for each treatment group were calculated.

Open-field test

The overall degree of locomotor activity was evaluated using Calvin S. Hall's Open Field Test (OFT). The open field test (OFT) apparatus for rats was a square arena that measured locomotor activity-the frequency at which a rodent uses all four paws to cross a grid of lines- and exploration and anxiety-like behavior. It was divided into equal squares, measuring 100 by 100 cm and 40 cm high. A 75 W lightbulb, positioned 75 cm above the arena, illuminated the center of the arena during the test, which took place in a poorly lit room. Each animal was placed in the center of the field and was observed for 5 min.

All experimental procedures were carried out during the light phase of the light: dark cycle (10:00–16:00 hr).

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Histological analysis

Brain slices from experimental and control animals were fixed in paraffin for morphological examination. Sections were cut to a thickness of 10 µm and stained with a cresyl violet solution (Sigma-Aldrich, Cat. No. C5042). Six rats from each group had their motor cortex neurons counted in columns. Every sixth section was collected and placed on poly-L-lysine-coated glass slides. Blind cell counting and a systematic random selection procedure were performed.

A 400x magnification with a 2D counting grid (250 µm × 250 µm) was used. Principal (small and internal pyramidal) neurons with well-identified nuclei were counted. For both experimental and control animals, a total of 30 columns were calculated from each case.

Statistical analysis

Statistical analysis was performed using GraphPad Prism 10. Behavioral data were analyzed using a two-way ANOVA followed by a Tukey post hoc test. Statistical analysis was performed based on the factors of gender and lead-ion dose. The level of significance is set at $p < 0.05$. All data will be presented as a mean ± standard error of the mean (SEM).

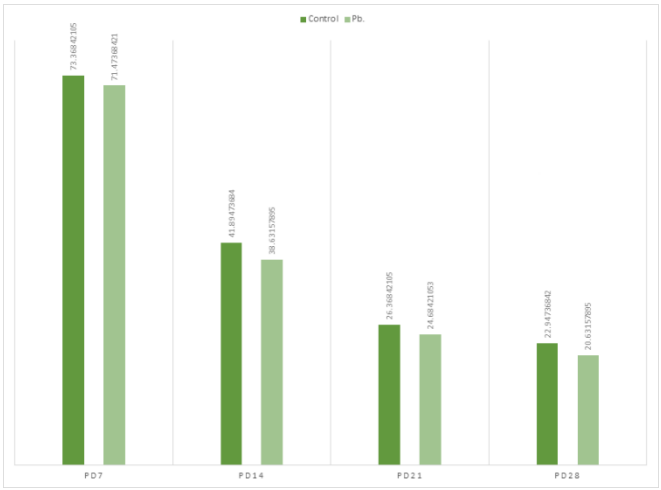
Every technique and experiment was authorized and conducted in compliance with the EC Ethical Directives and the recommendations established by the Ivane Beritashvili Center of Experimental Medicine on Animal Care (protocol# N 7/10.11.2025).

RESULTS

In our experiments, morphological analysis of the offspring's brain areas, including the motor cortex of interest to us, was performed on days PD7, PD14, PD21, and PD28, during the critical period of neurogenesis.

It was found that across all periods, the number of neurons in the motor cortex of experimental pups' brains decreased significantly compared with the control group. Additionally, there was a statistically significant difference even at a low dose (**FIG.2**).

FIGURE 2. Neuronal density in the motor cortex during critical periods of rodent brain development. Postnatal days 7, 14, 21 and 28.



Abbreviations: PD, postnatal days; Pb, lead

The data obtained are as follows: PD7 - cont.=73.30±1.072; exp.=71.40±1.9; PD14 - cont.=41.85±0.85; exp.=38.70 ±.0.66 ($P<0.05$); PD21 - cont.=26.45±1.85; exp=24.60±0.95; PD28 - cont.=22.90±1.10; exp.=19.80±0.76 ($P<0.05$).

At high dose, the following results were obtained: PD7 - cont.=72.30±1.067; exp.=70.40±0.2; PD14 - cont.=41.62±0.85; exp.=36.70±.0.66; PD21 - cont.=26.33±0.91; exp=23.50±1.1; PD28 - cont.=20.88±0.9; exp.=18.62 ±.0.56 ($P<0.05$).

At PD14 and PD28, we observed a statistically significant difference in neuronal density in the motor cortex of control pups and those exposed to a 0.05% lead dose.

The next experiments were started on PD61 offspring or young adult rats. The experiments were conducted separately on female and male rats.

To measure blood lead (Pb) concentration in PD61 rats by Atomic Absorption Spectrophotometry (AAS), a direct dilution method using Graphite Furnace AAS (GFAAS) was performed (wavelength: 280 nm; slit width: 0.7 nm). The method was validated using Certified Reference Material (NIST SRM 955c), yielding a recovery rate of 98±3%. The Limit of Detection (LOD) and Limit of Quantitation (LOQ) were 0.5 µg/Land 1.6

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µg/L, respectively. Blood lead concentrations are expressed in µg/dL to align with clinical toxicology standards for neurotoxicity assessment. Regardless of sex, the lead-exposed groups exhibited a significant, dose-dependent increase in BLLs compared to the control groups ($p<0.05$).

In the control group, the blood lead level was about 0.1 mg/dL. In the experimental groups, it was 5.8±0.3 mg/dL (Female), 6.1±0.4 mg/dL (Male) at the low dose (0.05%) ($P<0.01$) and 17.1±0.6 mg/dL (Female), 18.1±0.6 mg/dL (Male) at the high dose (0.2%) ($P<0.01$).

Behavioral tests in an open field were conducted on these offspring and revealed differences between female and male rats (**TAB.1** and **TAB.2**).

TABLE 1. Results of Open Field tests at the low dose (0.05%)

Parameter	Male control	Male Experimental	Female control	Female experimental
Ambulation	24.20±2.035	19.71±0.944	20.00±1.210	26.00±2.786
Entry into the center	1.50±0.32	1.08±0.38	0.81±0.35	0.92±0.27
Rearing	14.39±2.0	15.00±2.1	12.14±1.4	14.18±1.5
Grooming	4.50±1.6	4.05±1.7	6.0±1.8	7.2±2.5
Hole reflex	8.28±0.89	6.50±1.5	7.0±0.78	5.03±1.68
Bolus	1.33±0.24	2.08±0.79	1.57±0.81	2.17±0.41

TABLE 2. Results of Open Field tests at the high dose

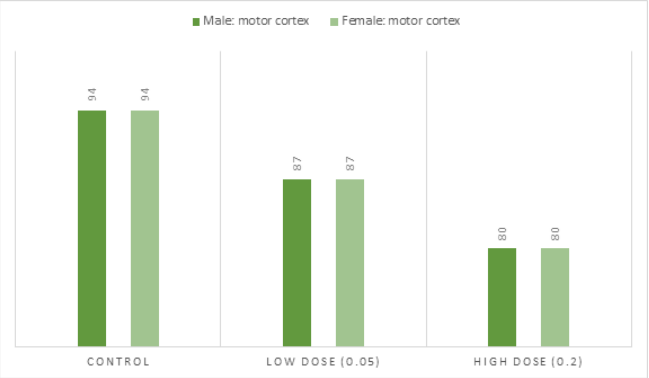
Parameter	Male control	Male experimental	Female control	Female experimental
Ambulation	32.17±2.455	12.75±1.373	30.00±1.732	37.67±2.333
Entry into the center	1.0±0.32	0.57±0.13	1.71±0.44	2.01±0.14
Rearing	12.31±1.8	10.00±1.6	14.11±1.4	16.17±2.4
Grooming	4.23±1.2	6.25±1.9	5.0±1.8	7.87±1.6
Hole reflex	6.24±0.79	4.50±1.5	7.57±1.08	5.79±1.39
Bolus	0.33±0.12	1.06±0.69	1.64±0.54	2.63±0.83

Male rats exposed pre- and early postnatal to lead ions exhibited lower locomotor activity than control rats, as measured by the number of lines crossed in the open field test. This decrease was dose-dependent, with a trend toward a decrease at low dose but not statistically significant: cont.=24.20±2.035; exp.=19.71±0.944 ($P=0.58$), while a statistically significant decrease occurred at high dose, as shown by the crossed lines cont.=32.17±2.455; exp.=12.75±1.373 ($P<0.01$).

Completely different results were obtained in female rats. Unlike male rats, they showed increased motor activity. At both doses, the experimental rats exceeded the control rats in the number of lines crossed in the low dose: cont.=20.00±1.210; exp.=26.00±2.786 ($P=0.26$); in the high dose: cont.=30.00±1.732; exp.=37.67±2.333 ($P=0.49$).

Histological analysis of the motor cortex of these rats' brains is as follows: Male rats – cont.=105.5±2.291; exp.=91.05±3.919 (low dose), 66.56±1.858 (high dose). Female rats - cont.=97.67±2.963; exp.=85.67±3.381 (low dose), 80.67±1.993 (high dose) (**FIG.3**).

FIGURE 3. Number of motor cortex neurons in a column of male and female rats



There is a trend toward a decrease in motor cortex neurons in the offspring of both sexes at both low and high doses, but this decrease is statistically significant in male rats at the high dose ($P<0.01$). The number of neurons in the motor cortex of male rats prenatally exposed to 0.2% lead acetate was reduced by 35% compared to control values (**FIG.4**).

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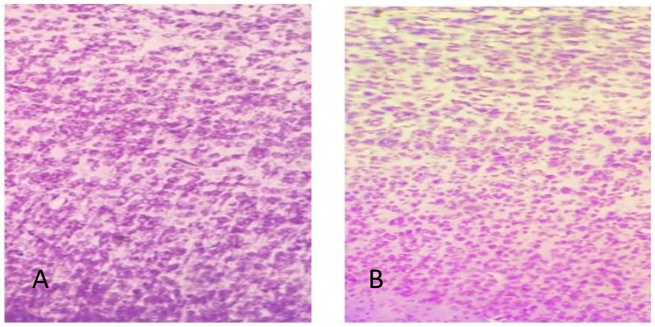
FIGURE 4. Motor cortex sections from a control male rat and a male rat exposed to 0.2% lead acetate



Explanation: A. Motor cortex sections from a control male; B. Motor cortex sections from a male rat exposed to 0.2% lead acetate. x400. Stained with cresyl violet.

DISCUSSION

The significance of lead ion exposure during the developmental period with respect to gender is discussed in the study presented. Despite the placenta's ability to shield the fetus from several toxins and extraneous substances, lead can freely pass through it, making prenatal exposure to the metal conceivable.

Numerous proteins in placental cells help detoxify chemicals that enter the placenta. However, research on the presence of harmful substances like lead in maternal and fetal blood samples has shown that the placenta cannot totally stop these toxicants from passing through. [21]

According to studies, because the blood-tissue and blood-brain barriers are still developing, lead uptake and accumulation in the fetus's brain and other tissues may increase as pregnancy progresses and organs develop. According to a study by Neda et al., the amount of lead in the fetus is determined by the mother's environmental exposure and by its transfer through the umbilical cord. [22] The work presented demonstrates that lead has a substantial effect on brain development at both low and high doses.

This is clearly shown during crucial stages of neurogenesis. Rats' postnatal development, which occurs between PD7 and PD28, is one of the main stages of brain maturation. It is characterized by rapid neurodevelopmental, metabolic, and physical changes, with an emphasis on hippocampal, cortical, and sensory development. The density of neurons in the cerebral cortex of 14-day-old and 28-day-old pups differs significantly from that of control and experimental animals. The brain is actively developing between PD7 and PD14, particularly in interneurons and neuronal networks. By PD21, numerous neural pathways have formed (PD28), and the brain is actively undergoing gliogenesis.

Sex hormones affect sexually dimorphic brain development, and gender-specific neurotoxicity is linked to differences in the density and distribution of estrogen receptors between males and females. [23] By controlling the expression of NADPH oxidases and antioxidant enzymes, estrogens play a significant role in the regulation of neuronal structure and the defense of the brain against oxidative damage. [24] Brain development depends on thyroid hormone, and the capacity of thyroid homeostasis to be disrupted varies by gender; females exhibited different thyroid-stimulating hormone levels in relation to blood lead levels. [25]

This study has demonstrated that male and female rats exhibit different outcomes when exposed to the same lead-based toxicant, suggesting that hormonal influences substantially impact motor cortex development.

Furthermore, our findings suggest that males are more vulnerable to prenatal exposure than females, with males typically having higher blood lead levels and greater risk of respiratory, neurobehavioral, and perinatal harm.

In summary, the study focuses on sex-related differences in neurotoxicity, with an emphasis on behavioral abnormalities related to motor activity and morphology. As mentioned in the introduction, the data available in the literature are mixed, and the present study will help clarify this issue.

CONCLUSIONS

Our results show that lead exposure during pregnancy and lactation reduces the density of motor cortex neurons. However, this effect is gender-dependent: it was statistically significant in male offspring and later expressed in their motor activity. We speculate that the motor area of the developing male rat brain is more sensitive to lead exposure than that of the female rat brain.

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