American Journal of Medicine and Medical Sciences 2024, 14(6): 1670-1672
DOI: 10.5923/j.ajmms.20241406.43

# Delay of Postnatal Growth and Development of Rat Offspring Caused by Mother's Chemical Stress

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**Abstract** This article investigates the effect of pre- and postnatal exposure to the organophosphorus pesticide chlorpyrifos on growth and development of laboratory rats in postnatal pathogenesis. The present study revealed that chlorpyrifos exposure of pregnant female rats resulted in offspring with lower birth weight and higher neonatal mortality rates, lagging behind in development in contrast to intact peers. The time of prenatal stress exposure on mortality and biometric indices of offspring is also of importance. Thus, early exposure to chlorpyrifos had a more significant effect than exposure in the second half of pregnancy.

**Keywords** Ontogenesis, Pesticides, Stress, Chlorpyrifos

## 1. Relevance of the Problem

Widespread use of pesticides contributes to the expansion of human and animal contact with toxic substances, occurrence of poisoning cases and increased morbidity of the population in areas of intensive use of pesticides. The prenatal (intrauterine) period of development is the most vulnerable, as the environment strongly influences the formation and physiology of the fetus. Factors acting during critical periods of fetal development can trigger a number of maladaptive mechanisms that have a major impact on the structure and function of tissues, with consequences that can have long-term effects, ultimately affecting their subsequent offspring [3,6].

**The aim of the study** - to investigate the effect of organophosphate pesticides (OPP) on the offspring of animals poisoned with chlorpyrifos at different stages of pregnancy.

## 2. Materials and Methods

The study was conducted on mixed-breed white rats weighing 180-200 g and their offspring at different stages of postnatal development. The first day of pregnancy was considered the day when spermatozoa were detected in vaginal swabs of female rats. The rats were then placed in separate cages. The animals were divided into 3 groups containing five pregnant females. The first group consisted of unaffected animals. In the 2nd group, the rats were poisoned with chlorpyrifos at a dose of 1/50 LD50 throughout the gestation period. In the third group, rats were poisoned with chlorpyrifos at a dose of 1/50 LD50 during the second half of pregnancy until birth. The gestation period, litter size, neonatal mortality, body weight gain, and maturation time of the rats were recorded in each group.

Death of newborn pups (excluding stillbirths) within three days of birth was recorded as neonatal mortality. Body weight of newborn pups in all groups was measured on days 1 and 30 after birth. Body weight of experimental rats was compared with that of control rats.

The day of eye opening (eyelids) was recorded for each animal in each group.

## 3. Results Obtained

**Neonatal mortality.** Neonatal mortality was observed in all groups of stressed animals. However, the mortality rate was higher in group 2 (18%) than in group 3 (9.8%). No neonatal mortality was observed in the control group.

**Body weight gain of newborn pups.**

The body weight of newborn pups at 1 day of age differed significantly among experimental groups. The body weight of newborn pups in group 2 (4.7±0.1 g, p=0.001) was significantly lower than that in the control group (5.9±0.30 g, p=0.003) or in group 2 (5.2±0.2 g, p=0.005). The body weight of the offspring on day 30 of postnatal life was significantly lower in group 1 (45.68±1.25 g, p=0.02) than in the control group (70.15±4.89 g, p=0.005) and in group 2 (61.22±0.005).

Eye (eyelid) opening in rats.

Analysis of the results shows a statistically significant difference in understanding between the experimental groups and the control group. Eyelid opening in group 3 (15.16 ± 0.30 days, p = 02) showed a significant delay

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Received: Mar. 25, 2024; Accepted: Apr. 17, 2024; Published: Jun. 21, 2024
Published online at http://journal.sapub.org/ajmms

compared to the control group (13.1 ± 0.70 days, p = 0.004), but still less than in group 2 (17.5 ± 0.2 days, p = 0.02). Experimental groups 2 and 3 took significantly longer to open their eyelids than the control group.

## 4. Discussion

In the present study, a positive correlation was observed between weight gain and pesticide exposure. The chemically stressed groups showed a significant decrease in maternal weight gain during pregnancy compared to the control group. Between day 1 and day 10 of gestation, a decrease in weight gain was observed in group 2.

Neonatal mortality was associated with maternal chlorpyrifos exposure during pregnancy. Taking this into account, we observed that neonatal mortality was observed in groups 2 and 3, which may be related to intoxication in the intrauterine period with subsequent disruption of organogenesis, which is a critical period for fetal development. Adverse conditions in the prenatal period may lead to persistent metabolic changes that increase the risk of postnatal disease. A significant decrease in offspring birth weight was observed in chlorpyrifos-exposed groups, which may also be related to maternal metabolic disturbances that ultimately lead to decreased protein synthesis and muscle catabolism during the fetal period and in postnatal ontogeny [4,7]. It has been reported that maternal stress can lead to decreased body weight in the offspring [2,9]. Maternal stress affects postnatal development and growth by altering liver and skeletal muscle metabolism. This study reports that the pesticide-exposed groups showed weight deficits as early as 30 days after birth. As possible causes of abnormal fetal and neonatal growth under prenatal stress, some researchers point to disorganization of the hypothalamic-pituitary system [1,5,8], impaired thyroid regulation in the offspring [10].

Delayed eye opening may indicate delayed or altered brain development. The present result shows that stress may cause a delay in eye opening, as the experimental groups took longer to see clearly than the control group; this may be due to a delay in the comparison of synaptic connection formation in brain centers [6,11].

## 5. Conclusions

- The developing fetus is highly sensitive to adverse maternal influences because the developing fetal brain is functionally less mature and unable to adequately respond and adapt to maternal stress [5,13].
- Maternal stress affects the fetus by increasing glucocorticoid secretion and placental glucocorticoid levels, which can lead to stimulation of gluconeogenesis and inhibition of tissue glucose uptake, decreased protein synthesis, and increased muscle atrophy [6,8].

The present study shows that maternal exposure to stress during pregnancy has long-term adverse effects on fetal development. For example, maternal chemical stress can cause stillbirth, neonatal mortality, birth defects, low birth weight, postnatal development, and delayed epiphyseal development.

Further studies are needed to understand the underlying morphofunctional and pathophysiological mechanisms of gestational stress-induced disorders [12]. Thus, it will be important in the future to identify the chemical stress-related factors that cause this dysregulation of fetal and neonatal development. We hypothesize that the observed changes are unlikely to be due to the action of chlorpyrifos alone, but are mediated by multiple causes. We hope that our subsequent work will deepen our understanding of these mechanisms and help identify ways to correct the abnormalities and prevent disease later in life.

## REFERENCES

[1] Джуманиязов Ш.А., Карабаев А.Г. Гипоталамо-гипофизарная нейросекреторная система у плодов и потомства животных, отравленных хлорпирифосом во время беременности. // Среднеазиатский журнал медицинских и естественных наук. - 2022. -3 (6) -с. 274-280.

[2] Джуманиязов Ш. А., Карабаев А.Г. Гипоталамо-гипофизарная нейросекреторная система у потомства крыс, при воздействии ФОС хлорпирифос на организм матери. // Мат. XXXIV Всеросс. научно-практич. конференции с междунар. участием. Казань. 2023. С. 38-41.

[3] Burenkova, O.V., & Grigorenko, E.L. (2024). The role of epigenetic mechanisms in the long-term effects of early-life adversity and mother-infant relationship on physiology and behavior of offspring in laboratory rats and mice. Developmental psychobiology, 66 3, e22479.

[4] Eick S. M. et al. Associations of maternal stress, prenatal exposure to per-and polyfluoroalkyl substances (PFAS), and demographic risk factors with birth outcomes and offspring neurodevelopment: // International jour. of environmental mental research and public health. - 2021. - Vol. 18. - No. 2. - p. 742.

[5] Djumaniyazov Sh.A., Nurimov P.B. Hypothalamic-pituitary neurosecretory system in the offspring of rats, when exposed to organophosphate pesticides // Ethiopian International J. of Multidisciplinary Research. - Ethiopian International Multidisciplinary Research Conferences. Vol. 2 No. 1 (2023) pp. 3-6.

[6] Glover, V., O'Connor, T. G., & O'Donnell, K. J. (2023). Fetal programming and public policy. Journal of the American Academy of Child & Adolescent Psychiatry, 62(6), 618620.

[7] Friedman, H.S., Markey, C.H. Encyclopedia of Mental Health, Third Edition: 2023, Volume 1-3; Vol. 1-3, Pages 1-885.

[8] Hall, J., Mercugliano, C., & Conti, L. (2023). Prenatal Stress and Endocrine Disrupting Chemical Exposure: Hypothalamic -Pituitary-Adrenal Axis Dysregulation as a Mechanism for the Health Consequences of Both. Medical Research Archives, 11(6).

[9] Lamichhane N. et al. Associations between maternal stress during pregnancy and offspring obesity risk later in life—A systematic literature review // Obesity Reviews. - 2020. - Vol. 21. - No. 2. - p. e12951.

[10] Yin, J., Liu, S., Li, Y., Hu, L., Liao, C., & Jiang, G. Exposure to MEHP during Pregnancy and Lactation Impairs Offspring Growth and Development by Disrupting Thyroid Hormone Homeostasis. Environmental Science & Technology. Environmental Science & Technology. 2024 58 (8), 3726-3736.

[11] Ying Sze, Paula J. Brunton; How is prenatal stress transmitted from the mother to the fetus?. // J Exp Biol 7 March 2024; 227 (Suppl_1): jeb246073.

[12] Zakharova, L. (2023). Long-term effects of prenatal stress and approaches to disorder correction during early development. Medicine and Art 1(3): 21-36.

[13] Zheng, B., Zheng, Y., Hu, W., & Chen, Z. (2024). Dissecting the networks underlying diverse brain disorders after prenatal glucocorticoid overexposure. Archives of Toxicology, 1-16.