PRENATAL STRESS AS A RISK FACTOR FOR STRUCTURAL ALTERATIONS IN THE CEREBELLUM

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Relevance of the topic: The prenatal period represents a critical stage of central nervous system development, during which the fetal brain demonstrates high sensitivity to adverse environmental influences. Chronic stress during pregnancy is considered one of the significant risk factors capable of disrupting neurodevelopment through neuroendocrine, metabolic, and inflammatory mechanisms. Elevated maternal stress hormones may interfere with normal brain maturation, leading to persistent structural and functional abnormalities. Among various brain regions, the cerebellum plays an essential role not only in motor coordination but also in cognitive and emotional regulation, making it particularly vulnerable to prenatal stress exposure. However, the structural consequences of chronic prenatal stress on cerebellar development remain insufficiently investigated.

The aim of this study was to assess the impact of chronic prenatal stress as a risk factor for structural alterations in the cerebellum of offspring under experimental conditions.

Materials and Methods: The study was conducted using an experimental animal model involving laboratory rats. Chronic stress was induced in pregnant females throughout gestation using standardized stress exposure protocols. Offspring born to stressed mothers were examined during the postnatal period. Cerebellar tissue samples were collected and analyzed using morphological and histological methods. Structural assessment focused on the organization of the cerebellar cortex, including cortical layers, cellular composition, and general tissue architecture. Microscopic evaluation was performed using conventional staining techniques to identify morphological deviations.

Results: The analysis revealed that prenatal exposure to chronic stress was associated with distinct structural alterations in the cerebellum of offspring. These changes were characterized by disturbances in the normal organization of cerebellar cortical layers, a reduction in cellular density, and morphological irregularities of neuronal elements. In several cases, thinning of specific cortical layers and altered cellular distribution were observed, indicating impaired cerebellar maturation. Such structural modifications suggest that prenatal stress negatively affects cerebellar development and may compromise its functional capacity during postnatal life.

The obtained findings are consistent with the concept that prenatal stress acts as a significant biological risk factor influencing neurodevelopment. Structural alterations in the cerebellum may underlie later functional impairments, including motor coordination deficits and cognitive disturbances. These results emphasize the importance of considering maternal stress as a critical factor in early brain development and highlight the need for preventive strategies aimed at reducing stress exposure during pregnancy.

In conclusion, chronic prenatal stress contributes to structural alterations in the developing cerebellum, confirming its role as a risk factor for disrupted neurodevelopment. Understanding the mechanisms through which prenatal stress affects cerebellar morphology may provide a basis for early interventions and improved strategies for protecting fetal brain development under adverse conditions.

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