Cytoskeletal Remodelation in Brain Tissue Exposed to Lead Nanoparticles

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Lead nanoparticles (PbNPs) in industrial areas pose a potential risk to organisms, therefore it is necessary to study the effects of their chronic inhalation. This project aimed to analyze the impact of lead nanoparticles inhalation on the olfactory epithelium and brain, as their potential toxic effect can induce neurodegenerative disorders. However, the effect of PbNPs inhalation on neurons or glial cells has not been described yet. Laboratory mice were exposed to insoluble or soluble PbNPs for eleven weeks in whole-body inhalation chambers. Experimental groups with a five-week-long elimination period were also included, which enabled me to observe the sufficiency of initiated reparative processes. All the work with live animals was done by my supervisor. I analyzed histopathological alterations in the olfactory epithelium as well as brain tissue. In the brain of exposed animals, I found necrotic neurons and spongiform alterations. Next, I focused on the most affected regions (hippocampus, telencephalon, cerebellum), and further analyzed the expression of selected cytoskeletal-associated markers. My study revealed changes in actin protein expression and Tau gene expression in exposed animals. Expression of other members of PI3K/Akt/mTOR signaling pathway, closely related to Tau protein, was after PbNPs inhalation alternated. In conclusion, uncovered morphological findings and changes in gene expression resemble processes observed during induction of neurodegenerative diseases. The selected five-week-long elimination period was determined as insufficient for effective tissue reparations. In the future, it will be necessary to discover tools that would support lead elimination or compensation processes in the olfactory epithelium and brain tissue.

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