Cadmium effects on human fetal spinal cord

Stefania Pacini, Tiziana Punzi, Gabriele Morucci, Massimo Gulisano, Mirca Marini, Erica Sarchielli, Barbara G. Vannelli

Department of Anatomy, Histology and Forensic Medicine, University of Firenze, Italy

The biological effects of Cadmium (Cd) on the central nervous system is poorly understood; it is thought that during brain development, when the blood-brain barrier is not well established, Cd may enter the brain and exerts its toxic effects. It has also been suggested that several diseases such as Parkinson disease, amyotrophic lateral sclerosis (ALS) as well as malformations such as spina bifida and forelimb ectrodactyly may be related to Cd exposure. In this study we evaluated the role of Cd in affecting motor neurons and glial cells of the ventral horns of human fetal spinal cord. Sections of human fetal spinal cord (9-12 weeks) were exposed for 24 h to 10 and 100 μM $CdCl_2$. Morphology was evaluated by Haematoxilyn-Eosin staining; distribution and number of motor neurons as well as of glial cells were studied by immunohistochemistry and by Western blot; apoptosis were investigated by TUNEL assay and by Western blot. The cell density in the ventral horns, after $CdCl_2$ treatments, appeared dramatically decreased and the number of apoptotic cells became higher in comparison to control specimens as demonstrated by the high expression level of three different apoptotic markers (NGFR p75, caspase 8 and PARP). The number and the distribution of motor neurons, expressing β tubulin III and positive to choline acetyltransferase, appeared significantly decreased after the treatment with different concentrations of $CdCl_2$. Activation and increase of glial cells were confirmed by the high expression level of Glial Fibrillary Acidic Protein (GFAP).

In this study we demonstrate the role of Cd, one of the most diffuse environmental pollutant linked to industrial development, in inducing apoptosis of motor neurons in the ventral horns of human fetal spinal cord. Motor neurons death is accompanied by a surrounding intense glyosis leading to a significant overthrow of the morphological architecture of spinal cord during its development.

Key words
Cadmium, human fetal spinal cord, neurodegenerative disease, morphology