LOSS OF GENOMIC STABILITY IN TESTICULAR LEYDIG CELLS BY CADMIUM EXPOSURE

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Abstract: Cadmium is an important industrial element that has well established toxicities and is a known environmental carcinogen. Human exposures to environmental cadmium are primarily the result of the burning of both fossil fuels and made-man wastes. However, there have been notable instances of toxicity as the result of long-term exposure to cadmium in contaminated food and water. While cadmium is a known carcinogen, the mechanism(s) underlying cadmium-induced carcinogenesis are not clear, although evidence suggest that it is cadmium’s ability to perturb the DNA repair process. However, the hallmarks of cancer development are well established and include the induction in the cell proliferation, changes in gene regulation, and the accumulation of mutations. Thus, the purpose of this study was to determine whether long-term exposure of both low and high concentrations of cadmium can perturb cell proliferation, increase DNA damage, reduce DNA repair, and the ultimately induce mutations in testicular Leydig cells. The methods used in this study include, cell proliferation assay, real time PCR profiling, and random amplified polymorphic. The effect of cadmium on cell proliferation was determined by cell count data, and cadmium-induced gene expression changes were measured by quantitative real-time polymerase chain reduction (PCR). The results this study revealed a concentration-dependent induction of cell proliferation by cadmium perturbation of DNA repair genes and genomic mutations by cadmium exposure. Thus, the findings of this study are novel, as they indicate a potential mechanism for cadmium-induced cancers. This is based on the observed increase in cell proliferation and decrease in the capacity of cells to maintain its genomic stability. In addition, our study provides the evidence that cadmium may play a role in the etiology of testicular cancer.

Keywords: Cadmium, carcinogenesis, gene expression, DNA repair, testes

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