THE MECHANISMS OF NICKEL ON THE CARCINOGENICITY AND TOXICITY OF MAMMALIAN RESPIRATORY EPITHELIAL CELLS (SW1573)

Kellie Brown¹, Kenneth Ndebele¹, Barbara Graham¹ and Paul B. Tchounwou²

¹Cancer Immunology Research Laboratory, ²Environmental Toxicology Laboratory, NIH-RCMI Center for Environmental Health, Environmental Science Ph.D. Program, College of Science, Engineering and Technology, Jackson State University, Jackson, Mississippi 39217. USA

Abstract: Nickel is a persistent environmental pollutant with toxic effects in man and other animals. Nickel has been classified by the International Agency for Research on Cancer and the U. S. Environmental Protection Agency a carcinogen. Nickel is a widespread contaminant in the environment. It is commonly found in soil, water, plants, volcanic emissions, and foods. Exposure to Nickel is a public health concern and is associated with a wide range of adverse systemic health effects. Lung inhalation is the major route of exposure for nickel-induced toxicity. Nickel can also be ingested or absorbed through the skin. The primary target organs are the liver, kidneys and lungs. To our knowledge there are no studies that have addressed the immunological cytokine profile induced by Nickel in lungs. We hypothesize that nickel exposure has the potential of modifying the cytokine milieu within the lungs, which may be important in the nickel-induced cell transformation. In this study we used sw1573 Alveolar carcinoma cells as a model and determined if nickel could alter long-term proliferation and clonogenic survival of alveoli cells. Cells were treated with various concentrations of nickel. Levels of proliferation were assessed using MTS. Here, we demonstrated that in the absence of any further treatment, nickel is sufficient to cause a consistent decrease in cell proliferation over the time-course examined. This decrease in proliferation was associated with an increase in levels of hsp70 as nickel concentrations increased. Nickel-induced suppression of sw1573 Alveolar cell proliferation, cytokine expression and apoptosis may have many ramifications for our understanding of immune and autoimmune responses and for the development of potential therapeutic intervention.

Key words: Nickel, cytokine expression, apoptosis