Oxidative Stress, Signal Transductions, Cell-Cell Communication

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Abstract: The objective of this research project was to study the mechanisms by which non-genotoxic or epigenetic chemicals induce multiple disease endpoints such as birth defects, tumor promotion, reproductive and neurotoxicities. The purpose is to develop a 'biologically-based' risk assessment model for human exposure to this class of toxic chemicals. The working hypothesis to have been tested was non-genotoxic chemicals disrupted homeostatic control of cell proliferation, differentiation and adaptive responses of differentiated cells. Three specific aims were designed to be tested (e.g., test a series of toxicants of interest to the USAFOSR for their ability to inhibit gap junctional function; to examine if these toxic chemicals alter the redox state of the cells; to determine if these chemicals alter apoptosis frequency via some oxidative damage-induced signal transduction mechanism). Results showed a structure- function relationship between PAH molecules and inhibition of gap junctions; jet fuels JP8 and JP4 were inhibitory to gap junctions; and perfluorinated fatty acids with chain length of 7 to 10 carbons were inhibitory to gap junctions.

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