Dear Faculty, IGERT Fellows, IGERT Associates and Students,

You are cordially invited to attend a Seminar presented by Sabrina Lin. Please plan to attend.

Sabrina C. Lin, Ph.D.

Postdoctoral Fellow UCR Stem Cell Center Cell Biology and Neuroscience Department University of California, Riverside Date: Monday, October 8, 2012 Location: Bourns A265 Time: 11:10am

Harm Reduction Cigarette Smoke Impairs Dynamic Cell Process in Stem Cells and Pulmonary Fibroblasts Through Depolymerization of the Cytoskeleton

Abstract:

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Cigarette smoking adversely affects prenatal development and causes postnatal cognitive and respiratory deficits. Harm reduction (HR) cigarettes, which are advertised to contain fewer toxins and be safer than conventional brands, have been introduced to reduce adverse health effects. In this study, the effects of conventional and HR cigarette smoke were examined on various endpoints: (1) attachment, (2) survival, (3) proliferation, and (4) migration. Human embryonic (hESC) and mouse neural stem cells (mNSC) were models for epiblast and developing brain cells, respectively. Human pulmonary fibroblasts (hPF) were chosen for comparison as they come from the respiratory system, a direct target of inhaled smoke. Attachment, survival, and proliferation of the three cell types were evaluated using dose response MTT experiments. Doses ranged from 0.001 to 1.0 puff equivalent (PE), where 1 PE equals the amount of smoke in 1 puff that dissolves in 1 ml of medium. Sidestream (SS) smoke inhibited each endpoint in all cell types with hPF being the most sensitive followed by mNSC and hESC. hPF proliferation was inhibited at 0.03PE but attachment was not affected significantly. mNSC attachment was inhibited at 0.3PE and proliferation at 0.03PE. hESC attachment was inhibited at 1PE and proliferation at 0.3PE. HR SS smoke was generally more potent than smoke from the conventional brand. hESC colony growth and mNSC migration were evaluated at non-cytotoxic doses using live-cell imaging in conjunction with video bioinformatics software analysis. SS HR cigarette smoke inhibited hESC growth and mNSC gap closure, suggesting an effect on the cytoskeleton of the cells. Immunohistochemistry experiments and Nikon NIS-Elements software analysis showed that SS smoke depolymerized hESC and mNSC actin. Doses used in this study were comparable to levels in tissues of human smokers, suggesting that exposure to HR could affect the health of embryos and fetuses and cause adverse effects in adult lungs.

