

James & Esther King Biomedical Research Program

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Medicine
University of Miami

2009 Program
New Investigator (3-year project)

Project Title: Mechanisms Underlying Nicotine's Proatherogenic Effects in Macrophages

Project Summary: Cigarette smoking is a significant risk factor for development of cardiovascular diseases. Cigarette smoking is known to contain various components that are distributed in both particulate and gaseous phases. One of the major active components of cigarette smoking is nicotine. Nicotine has been shown to contribute to many of the toxicities related to cigarette smoking, and has also been shown to promote atherosclerosis. Atherosclerosis is considered to be a chronic vascular inflammatory disease associated with excessive lipid (cholesterol) accumulation in the vascular wall. Macrophages (one type of white blood cells) play an important role in the progression of atherogenesis through the accumulation of cholesterol and the production of inflammatory cytokines (cellular factors). We have recently identified that human macrophage nicotine increases the expression of CD36, an important intracellular signaling molecule in the regulation of lipid metabolism and inflammatory factors expression. The long-term goal of this study is to clarify the molecular mechanisms by which nicotine contributes, promotes, or accelerates atherosclerotic cardiovascular disease. In this grant, we will investigate how nicotine increases CD36 expression (intracellular signaling pathway) in macrophages and how increased CD36 contributes to atherogenic effect of nicotine. The findings from this grant will provide valuable information to develop new strategies for the prevention and treatment of smoking-related cardiovascular diseases.