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Impact of hypercholesterolemia on endothelial K+ channels

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Plasma hypercholesterolemia is well known to be a major risk factor for the development of atherosclerosis with the earliest effects manifested by endothelial dysfunction. The mechanisms, however, of hypercholesterolemia-induced endothelial dysfunction are still not well understood. Our earlier studies demonstrated one of the dominant types of endothelial ion channels, inwardly-rectifying K+ channels (Kir) are suppressed by enriching the cells with cholesterol or by exposing them to acetylated and very-low density lipoproteins (acLDL and VLDL). We have also shown that endothelial Kir channels are suppressed *in vivo* by diet-induced hypercholesterolemia in a pig model of atherosclerosis. The effect was observed both in mature aortic endothelial cells and in bone-marrow-derived progenitor cells. More recently, we extended these studies to cardiomyocytes and demonstrated that hypercholesterolemia has differential effects on different types of Kir channels with Kir2 channels being suppressed and Kir3 channels being facilitated. In the current study, we analyze the impact of pro- and anti-atherogenic lipoproteins on endothelial Kir channels. Our observations show that endothelial Kir channels are suppressed by the elevated levels of LDL and that this effect can be rescued by HDL. Moreover, endothelial Kir channels are sensitive to LDL/HDL ratio, a strong predictor of the developing cardiovascular disease. We propose that hypercholesterolemia-induced suppression of endothelial K+ channels plays a major role in the development of endothelial dysfunction.

Biography

Irena Levitan was a Research Assistant Professor/Research Associate Professor at the Department of Pathology and Lab Medicine Institute for Medicine and Engineering University of Pennsylvania. She is an Associate Professor of Pharmacology and Bioengineering in Medicine, Department of Medicine University of Illinois at Chicago.

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