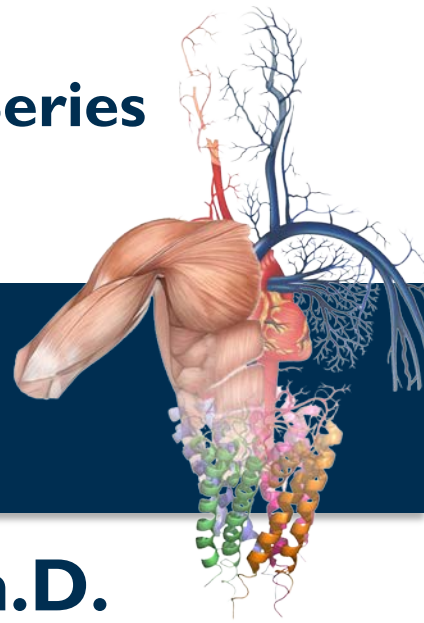


Distinguished Lecture Series in Physiology

Physiology and Membrane Biology

University of California, Davis



Darleen Sandoval, Ph.D.

University of Michigan
Department of Surgery

"Using genetics and pharmacology to understand **GLP-I** physiology"

The proglucagon gene (*Gcg*) is expressed in the intestine, the pancreas, and a small cluster of neurons in the hindbrain and encodes multiple peptides in a tissue-specific manner. One of these peptides, glucagon like peptide-1 (GLP-1) increases following meals, functions to stimulate insulin secretion, and is essential for normal glucose tolerance. Current dogma holds that intestinally-derived GLP-1 acts as a hormone, delivered through the circulation, and binding to pancreatic GLP-1 receptors (GLP-1r) to stimulate insulin secretion. However, in both human and rodent models, there is emerging *in vitro* and pathophysiological evidence demonstrating that GLP-1 is produced in the endocrine pancreas. Our preliminary data are the first to reveal *in vivo* evidence that not only does a pancreatic source of GLP-1 exist, but that this pool of active peptide is necessary for normal glucose tolerance. These data represent a paradigm shift in our understanding of the GLP-1 system. In this model, the major insulinotropic effects of GLP-1 are paracrine rather than endocrine, with secretion from islet α -cells that stimulates β -cells through local, intraislet interactions with the GLP-1r.

Genome Auditorium
Thursday, April 21, 2016
11:00 a.m.

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