

Rohit Kohli, MD

Assistant Professor

Department of Pediatrics; Division of Gastroenterology, Hepatology, & Nutrition

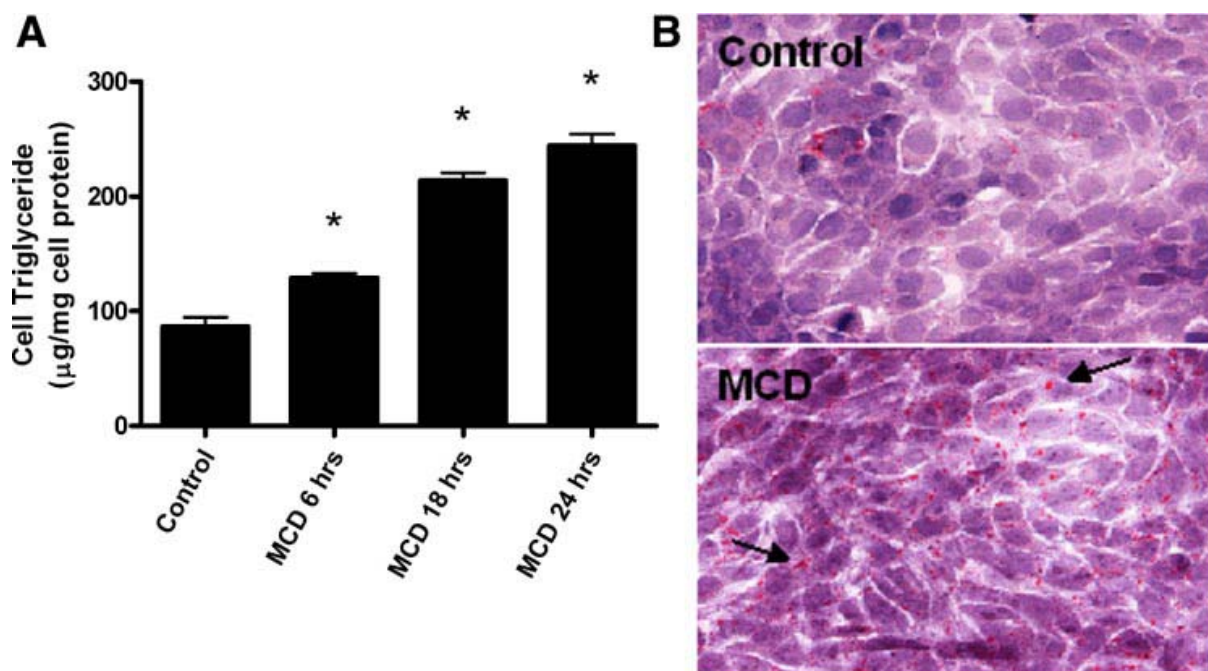
Description of Research:

Dr. Kohli's research focuses on studying the role of mitochondria in the pathogenesis of obesity related to fatty liver disease. Reactive oxygen species (ROS) have been shown to initiate various cell signaling cascades including those mediated via receptor tyrosine kinases, members of the mitogen-activated protein kinase family and PI3-kinase. He has shown that mouse hepatocytes cultured in a methionine and choline deficient (MCD) growth medium develop steatosis that is dependent upon PI3-kinase activation. Furthermore, Dr. Kohli has demonstrated that cultured hepatocytes incubated with MCD media results in increased mROS production. This increase in mROS is the initial signal that results in cellular steatosis mediated through the PI3-K pathway. This outlined pathway and its physiologic end points can be successfully manipulated by either exogenous or complex-III mROS modulation. Dr. Kohli's work highlights a potential role for mROS in hepatocellular steatosis signaling and may lead to novel therapeutic options in the future.

Collaborations:

Dr. Kohli is a new faculty member in the Division of Gastroenterology, Hepatology and Nutrition. He is developing a line of collaboration with Dr. Seeley.

Representative Figure:



Effect of methionine-choline-deficient (MCD) medium on fat accumulation in cultured hepatocytes. Cultures were exposed to control medium or MCD medium for 6–24 h, and cell triglyceride content (A) was assessed. Each value is the mean \pm SE of 5 separate determinations (A). * $P < 0.01$ vs. control. Histological steatosis assessed by Oil-Red O staining at 18 h shows neutral fat (arrows) within the hepatocytes (B). Figure 1 from Am J Physiol Gastrointest Liver Physiol, 2006; 291: G55-G62.