Insulin Resistance, Inflammation and a Muscle-Saving Protein

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In the online May 2 issue of the journal *Cell Metabolism*, researchers at the University of California, San Diego School of Medicine publish three distinct articles exploring:

- the complex interactions of lipids and inflammation in insulin resistance
- the roles of omega 3 fatty acids and a particular gene in fighting inflammation
- how elevated levels of a particular protein might delay the muscle-destroying effects of amyotrophic lateral sclerosis.

Type 2 diabetes has reached epidemic proportions around the world, fueled in large part by the equally alarming expansion of obesity as a global health problem. But while it's well-known that obesity is the most common cause of insulin resistance – the primary metabolic abnormality in type 2 diabetes – researchers have only recently begun to effectively parse the underlying, complicated relationships between lipids (fats and related molecules essential to cell structure and function) and chronic tissue inflammation (a key cause of obesity-induced insulin resistance).

In a wide-ranging Perspective article published in *Cell Metabolism*, Christopher K. Glass, MD, PhD, a professor in the departments of Cellular and Molecular Medicine, and Medicine at the UC San Diego, and Jerrold M. Olefsky, MD, associate dean for Scientific Affairs and Distinguished Professor of Medicine at UC San Diego, survey where the science stands, describing, for example, the pro-inflammatory effects of saturated fatty acids and the anti-inflammatory benefits of omega 3 fatty acids. They also discuss how inflammation impacts lipid metabolism at the cellular, tissue, organ and whole-body levels.

In a second, related article, Olefsky and colleague Da Young Oh, an assistant project scientist, discuss the critical role of a gene called GPR120 in inhibiting pro-inflammatory macrophages while simultaneously boosting the anti-inflammatory benefits of omega 3 fatty acids. They argue that new research highlights the importance of GPR120 as an attractive target for new drugs that could increase insulin sensitivity and, perhaps, have anti-obesity effects as well.

Finally, Don W. Cleveland, PhD, professor and chair of the Department of Cellular and Molecular Medicine and head of the Laboratory of Cell Biology at the Ludwig Institute for Cancer Research at UC San Diego and colleagues report the effects of elevated levels of a gene- regulating protein in mouse cells afflicted by a form of amyotrophic lateral sclerosis or ALS.

In humans, ALS is a progressive, adult-onset neurodegenerative disorder characterized by selective motor neuron and muscle loss that ultimately results in fatal paralysis. Among the key players in muscle function is a transcriptional activator protein called PGC-1alpha, which helps enhance various aspects of muscle cell function, including metabolism and mitochondrial biogenesis.

Cleveland and colleagues report that elevated levels of PGC-1alpha in the muscles of a mouse model of inherited ALS helps maintain health and function, though it does not extend survival time. The researchers suggest that increasing PCG-1alpha activity in muscle could be a new and attractive therapeutic target for maintaining, improving and extending physical abilities in ALS patients.

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