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EUROPEAN ENDOCRINOLOGY AND DIABETES CONGRESS

August 22-23, 2022 | Webinar

Mechanisms of skeletal muscle myokines in ameliorating Insulin resistance

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Insulin resistance/type 2 diabetes (T2D) has already reached global pandemic levels. Skeletal muscle accounts for ~80% of postprandial glucose clearance and is a major regulator of peripheral insulin sensitivity. It is the largest organ in the body and secretes circulating factors, including myokines, which are involved in various cellular signaling processes. It is vital for metabolism, physiology and insulin-mediated glucose disposal. Moreover, skeletal muscle insulin resistance is the underlying primary defect that is evident decades before progression to clinical diabetes. Myokines have auto/para and endocrine functions, serving as vital regulators of myogenic differentiation, fiber-type switching, maintaining muscle mass, increase insulin sensitivity and glucose disposal. Over 3,000 possible myokines have been identified to date, and the functions of many myokines have been determined. For instance, Irisin, fractalkine, FGF21, myonectin, and IL-15 are determined to improve β-cell mass and/or function, which regulates glucose and lipid metabolism. FGF21 and irisin have been identified to decrease triacylglycerol levels and increase insulin sensitivity via skeletal muscle glucose uptake, white adipose tissue lipolysis/browning, and increased energy expenditure. Human obese/overweight subjects participating in an aerobic exercise program were found to have higher serum myonectin levels, with significantly decreased susceptibility for insulin resistance. As with many therapeutic targets, the myokine levels in healthy and diseased individuals vary significantly, leading to differential outcomes. In addition, the myokine receptor abundance is often not quantified, which could be a contributing factor to the differential outcomes observed thus far. Nevertheless, the list of new myokines is steadily increasing, and comprehensive analyses of myokine networks, local and systemic levels in health and disease, and their synergistic functions, will help to determine druggable targets in the near future. Therefore, this talk will overall summarize the current status of myokine therapies for T2D.

Biography

Rekha Balakrishnan is a researcher at City of Hope, CA. She is an American Heart Association's postdoctoral fellowship awardee interested in determining how skeletal muscle mediated organ crosstalk improves metabolism. After her Ph.D. in the India, she moved to Rutgers, NJ to work on how alteration of calcium regulating proteins in muscular dystrophy impairs mitochondrial metabolism. She is a recipient of awards including best women researcher/research work and research paper from national and international conferences for her Ph.D. and postdoc works. She has the passion and dedication to develop and refine the skills essential to become a successful independent scientist in the field of diabetes and metabolic diseases.