Tata Institute of Fundamental Research
Survey No. 36/P, Gopanpally Village, Serilingampally, Ranga Reddy Dist., Hyderabad – 500 046

Comprehensive Seminar

Investigating skeletal muscle metabolism and trophism as a function of mitochondrial activity in the background of malnutrition

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Skeletal muscle is one of the largest organs of the body, making up approximately 40% of total body mass. Its architecture is unique, in that it is an orderly arrangement of myofibrils that give rise to multiple fibers within a single muscle. These fibers can be of different types based on their energy producing pathways (glycolytic or OXPHOS), contractile profiles, and proteome. Extensive research has shown that muscle fibers are plastic, capable of adapting to environmental stressors and transitioning from one type to another.

Central to understanding the function and phenotype of skeletal muscle fiber types are mitochondria. Mitochondrial content, energetics, and dynamics constitute key pathways that dictate fundamental properties of muscle fibers, such as muscle contraction, calcium signalling and ATP production.

Although mitochondrial dynamics, morphology and biogenesis have been shown to intrinsically modulate fiber type determination, transition, and their growth, these factors rarely act alone in a physiological setting. External factors such as diet or exercise have also been shown to regulate muscle fiber phenotype by impinging on critical mitochondrial functions. Several studies have highlighted the deleterious effects of overnutrition (high-fat diets or western diets) on mitochondrial functions in skeletal muscle such as downregulation of oxidative phosphorylation and increased oxidative stress, leading to insulin resistance and development of metabolic syndrome.

However, similar studies on interaction between undernutrition (protein-energy malnutrition) and mitochondrial functions in skeletal muscle is largely lacking. Furthermore, beneficial interventions such as exercise or amino acid supplements are incompletely characterised, with research largely being restricted to effects on muscle protein synthesis. How these factors modulate mitochondrial activity and further affect muscle fiber phenotype and functions are still unknown and hinder our understanding of skeletal muscle physiology. Uncovering mechanistic details of mitochondrial form and function in the context of malnutrition will aid in tackling this silent pandemic.

Monday, Mar 11th 2024

15:30 Hrs (Tea / Coffee 15:15 Hrs)

Seminar Hall, TIFR-H