**Regulatory mechanisms of neuromuscular junction homeostasis**

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Skeletal muscles possess a remarkable plasticity in energy metabolism and contractile function in response to various stimuli like exercise, hormones or nutritional states. Declines in muscle mass and function are implicated in numerous metabolic pathologies, including insulin resistance and obesity. In aging, skeletal muscle exhibits a progressive decline in mass and functionality associated with increased risks to develop various chronic diseases. Although sarcopenia is multifactorial, the mitochondrial dysfunction, anabolic/catabolic impairment and neuromuscular impairment are the main aspects underlying muscle wasting related to aging or other pathological conditions. Thus, enhancing mitochondrial function, anabolic pathways and neuromuscular interaction are viewed as key strategies to intervene muscle loss and weakness. Previously we have shown that Prmt1 deficiency in muscle causes dysregulation of Prmt6/FoxO3/autophagy associated with muscle atrophy and weakness. Our published and unpublished studies on the role of Prmt1 in motor neuron and muscle suggest that Prmt1 is critical for the maintenance of neuromuscular integrity and mitochondrial organization. Thus, Prmt1 represents a potential target for the development of the therapeutic strategy to intervene muscle wasting related to aging or other diseases.