FALL 2020

Biochemistry and Molecular Biology Brown Bag Series

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Ph.D. Student

"Lipin1 overexpression ameliorates the dystrophic phenotype in mdx mice by enhancing myofiber membrane integrity"

Tuesday, November 17, 2020

11:00 AM

Please contact x3249 if you would like to attend but did not receive an emailed link.

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https://science-math.wright.edu/biochemistry-and-molecular-biology

Abstract

Lipin1 overexpression ameliorates the dystrophic phenotype in mdx mice by enhancing myofiber membrane integrity

Duchenne muscular dystrophy (DMD) is a severe and progressive muscular dystrophy caused by mutations in the dystrophin gene. The absence of dystrophin triggers instability of the plasma membrane in skeletal muscle, leading to membrane damage during muscle contraction, as well as repeated cycles of death and regeneration of muscle fibers. Currently, there is no cure for this disease. Lipin1 is a phosphatidic acid (PA) phosphatase (PAP) that catalyzes the conversion of PA to diacylglycerol (DAG). Our data show that lipin1 mRNA and protein expression levels are dramatically reduced in skeletal muscles of the mdx mouse model of DMD. Strikingly, we found that increasing lipin1 levels with a viral vector in mdx mice lessened muscle fiber degeneration and improved muscle mass. We further found that lipin1 overexpression effectively improved plasma membrane integrity and reduced muscle necrosis in mdx mice. Since loss of muscle membrane integrity is the proximate cause of dystrophic muscle, our data collectively suggest that lipin1 plays a major role in inhibiting muscle degeneration by improving membrane integrity in mdx mice. Further research is required to investigate how lipin1 contributes to improving the integrity, stability and function of myofibers.