"Examine How Lipin1 Ameliorates Dystrophic Phenotype using Mdx:Lipin1 Transgenic Mice"

Tuesday, February 9, 2021
11:00 AM

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

Lab: Hongmei Ren, Ph.D.
Examine How Lipin1 Ameliorates Dystrophic Phenotype using Mdx:Lipin1 Transgenic Mice

Abstract
Duchenne muscular dystrophy (DMD) is a genetic disorder inherited through X-linked manner affecting 1 in 3500 male births. It is characterized by mutations on the dystrophin gene which leads to the loss of functional dystrophin protein. The dystrophin protein is part of a complex of proteins that stabilizes the skeletal muscle membranes due to mechanical stress exerted by movements. Lack of dystrophin leads to membrane tear and damage leading to muscle death through necroptosis. Currently there are no effective treatments for the DMD.

Lipin1 is a phosphatidic acid phosphatase that converts phosphatidic acid (PA) to diacylglycerol (DAG). DAG is an important molecule that participates in phospholipid biosynthesis. Preliminary data from our lab shows that lipin1 expression, both at the protein and mRNA level, is downregulated in Mdx mice, the DMD mice model. Increasing the lipin1 expression through AAV delivery system shows an improvement in muscle health in Mdx mice. This suggests that lipin1 improves skeletal muscle integrity in Mdx mice and possibly in DMD patients. Currently we have generated Mdx muscle specific lipin1 knock-in mice using MCK-Cre system. Preliminary data suggests that skeletal muscle lipin1 overexpression in Mdx mice alleviates necroptosis and improves muscle health in Mdx mice.