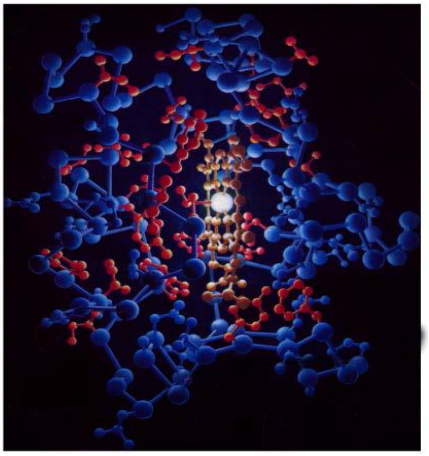

Horse Heart Cytochrome *c* with iron atom illuminated

Seminar Notice

Department of Biochemistry and Molecular Biology

Rajsi Thaker

M.S. Thesis Defense

Biochemistry and Molecular Biology

"Potential drug treatment for Duchenne muscular dystrophy through upregulation of lipin1"

Wednesday, July 28, 2021
10:00 AM

Please contact x3042 if you would like to attend but did not receive an emailed link.

Please Post!

Advisor: Dr. Hongmei Ren, Ph.D.

Boonshoft School of Medicine
WRIGHT STATE UNIVERSITY

WRIGHT STATE UNIVERSITY

http://www.med.wright.edu/bmb

Abstract

Potential drug treatment for Duchenne muscular dystrophy through upregulation of lipin1

Duchenne muscular dystrophy (DMD) is a genetic disorder leading to progressive muscle degeneration and weakness due to mutation in dystrophin gene, which is very important for maintaining muscle membrane integrity. There is currently no cure for DMD. Our lab recently found that lipin1 is important for muscle membrane integrity. Our recent study identified that upregulation of lipin1 expression in mdx mice through AAV-lipin1 gene delivery, strengthened membrane integrity and reduced muscle fiber degeneration. In this study, we identified two drugs, dexamethasone and rosiglitazone, can elevate lipin1 mRNA and protein expression levels. Mdx mice treated with dexamethasone for two weeks and rosiglitazone for one week had elevated lipin1 expression levels and downregulated necroptotic markers including RIPK1, RIPK3, MLKL, and pMLKL. Rosiglitazone treatment in mdx mice also downregulated apoptotic markers including BAX, BAK and cleaved caspase-3. Most importantly, muscle membrane integrity indicated by IgG staining was strengthened after treatment with dexamethasone. Our future study will identify whether the effects of dexamethasone and rosiglitazone on the inhibition of necroptotic markers and the improvement of membrane integrity of dystrophic muscles are through the upregulation of lipin1 expression.