Human Genetics & Embryology

2020

Vol & Issue: S1

Drosophila TRIM32 cooperates with glycolytic enzymes to promote cell growth

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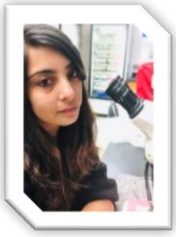
Abstract

Cell growth and/or proliferation may require the reprogramming of metabolic pathways, whereby a switch from oxidative to glycolytic metabolism diverts glycolytic intermediates towards anabolic pathways. Herein, we identify a novel role for TRIM32 in the maintenance of glycolytic flux mediated by biochemical interactions with the glycolytic enzymes Aldolase and Phosphoglycerate mutase. Loss of Drosophila TRIM32, encoded by thin (tn), shows reduced levels of glycolytic intermediates and amino acids. This altered metabolic profile correlates with a reduction in the size of glycolytic larval muscle and brain tissue. Consistent with a role for metabolic intermediates in glycolysis-driven biomass production, dietary amino acid supplementation in tn mutants improves muscle mass. Remarkably, TRIM32 is also required for ectopic growth - loss of TRIM32 in a wing disc-associated tumor model reduces glycolytic metabolism and restricts growth. Overall, our results reveal a novel role for TRIM32 for controlling glycolysis in the context of both normal development and tumor growth.

Biography:



Simranjot Bawa is a final year Ph.D. student at Kansas State University in the deparatment of Biochemistry and Molecular Biophysics. As a senior graduate student, she is accountable for developing, executing, and troubleshooting molecular and biochemical research experiments in the laboratory of my thesis advisor, Dr. Erika Geisbrecht. Our lab broadly seeks to understand cell and molecular level events required for the development and maintenance of healthy muscle tissue. She had published 5 research articles in a peer- reviewed journals

10th International Conference on Human Genetics and Genetic Diseases

September 21-22, 2020.

Abstract Citation:

Simranjot Bawa; Drosophila TRIM32 cooperates with glycolytic enzymes to promote cell growth; Human Genetics 2020; September 21-22, 2020.