RUTGERS–NEW BRUNSWICK
School of Environmental and Biological Sciences

The Department of Nutritional Sciences
Fall 2025 Seminar Series

"Hepatic Gluconeogenesis Transcription, Gene Expression and Activity: Living in the Zone "

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Host: Malcolm Watford, D. Phil
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Wednesday, December 3, 2025 @ 2:15 PM

FSNS Building, 65 Dudley Road, New Brunswick, Room 120

Zoom option: https://go.rutgers.edu/pessin-seminar

The liver generates glucose via gluconeogenesis and glycogenolysis during states of nutrient deprivation and conversely in states of nutrient excess repletes its glycogen stores, stimulates de novo lipogenesis for fatty acid synthesis from carbon precursors, and packages both newly synthesized and ingested dietary lipids into very low-density lipoprotein particles for secretion and utilization by peripheral tissues. Dysregulation of these liver glucose and lipid metabolic processes are primary hallmarks of insulin resistance and dyslipidemia that occur in obesity and type 2 diabetes. Within each lobule the hepatocytes are distributed from the portal triad (portal vein, hepatic artery, bile duct) at the six vertices of the hexagon and the central vein located at the centroid. The hepatocytes aligned within a lobule display functionally distinct metabolic properties and are generally divided into three zones across the periportal to pericentral axis. Early studies indicated that gluconeogenesis is 2 to 3-fold greater in the periportal hepatocytes compared to pericentral hepatocytes.

We recently reported that gluconeogenesis across the liver lobule is highly plastic, and the magnitude depends upon the precise experimental conditions analyzed. Moreover, we have now identified a subset of naturally occurring periportal hepatocytes, that we termed as Dual-Modal hepatocytes, that express both de novo lipogenic and gluconeogenic genes in the fully fed state. I will present data based upon several single cell spatial and metabolic technologies including scRNAseq, smFISH, PrimeFlow and Metabolic Spatial Imaging that this subset of periportal hepatocytes is responsible for basal gluconeogenesis and is resistant to the actions of insulin.

For parking permits, please contact Christina Duffy, Department of Nutritional Sciences, cduffy@sebs.rutgers.edu , 848-932-5425