

New College of Florida Research Uncovers Key Role of β-catenin in Fat Metabolism and Immune System Interactions

A team of researchers has published groundbreaking findings in Molecular Metabolism that shed light on how the body regulates fat storage and metabolic health.

SARASOTA, FL, UNITED STATES, August 20, 2025 /EINPresswire.com/ -- A team of researchers, including New College of Florida's Dr. Romina M. Uranga, has published groundbreaking findings in Molecular Metabolism that shed new light on how the body regulates fat storage and metabolic health. The study, titled "Effects of β -catenin deficiency on adipose tissue physiology," provides compelling evidence that a protein called β -catenin plays a central role in how fat cells (adipocytes) communicate with surrounding immune and support cells to maintain balance in the body.

The paper represents a major career milestone for Dr. Uranga, who worked for three years conducting experiments in transgenic mice in



Co-author Dr. Romina Uranga

Dr. MacDougald's lab at the University of Michigan, followed by an additional year at New College, Division of Natural Sciences, dedicated to analyzing data and writing findings. "It's my pleasure to share what will probably be the best paper of my career," Dr. Uranga said. "This project represents years of effort and a dream come true: working with Dr. Ormond MacDougald, world-renowned in research on the physiology and pathophysiology of adipose tissue, and I'm excited to bring results back to my students at New College, where they will discover the cool experimental strategies we used to answer our research questions." The research demonstrates that when β -catenin is absent in fat cells, male mice—but not females—develop greater fat accumulation and insulin resistance. This imbalance suppresses normal fat metabolism while unexpectedly boosting the efficiency of energy use in immune cells such as macrophages and dendritic cells. The team also discovered that when fat cells lose β -catenin, other cells—endothelial cells—compensate by increasing their own expression of β -

catenin and delivering it back to the deficient adipocytes through extracellular vesicles known as exosomes, thereby helping to preserve overall tissue balance.

"This study further elucidates the intercellular crosstalk that occurs within adipose tissue, and which Wnt signaling plays an integral role in," said Dr. Uranga. "We found that when fat cells lose a key signaling pathway, other cells step in to adapt and preserve metabolic stability. It highlights an elegant, built-in resilience in how our bodies regulate fat and energy."

The findings carry significant implications for understanding obesity

and related metabolic disorders. Wnt/β-catenin signaling, an ancient pathway conserved across species, has long been suspected of influencing fat biology. By mapping out its precise effects in adipose tissue, this research helps explain why disruptions in the pathway can contribute to metabolic disease—and how the body attempts to compensate.

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Ctnnb1^{AdKO} mouse 1 Insulin Resistance 🕈 Fat Mass Immune Cells **β**-catenin ♣ ROS deficiency Mitochondrial -1-7-Small Extracellular Vesicles (sEVs) Adipocytes Hypertrophy Endothelial Cells **↑** Ctnnb1 mRNA Graph showing how B-catenin deficiency impacts Fat

Mass in mice.

"This research underscores New College's growing role in advancing biomedical knowledge," said New College President Richard Corcoran. "It's a powerful example of the kind of faculty excellence that strengthens our reputation and offers students unique opportunities to engage with meaningful, real-world science."

The article will appear in the October issue of Molecular Metabolism.

Read the full study here

About New College of Florida

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