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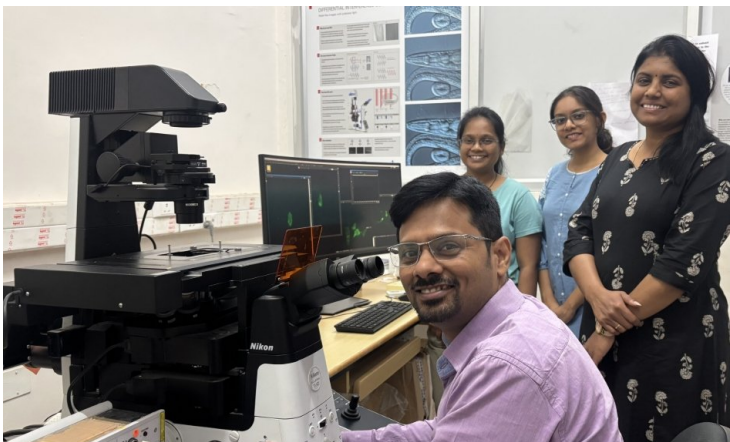
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IISc untangles glucose traffic jams for treatment of Type 2 diabetes

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New work points to I^2 -cell glucose uptake as a promising target



Our body relies on a molecular traffic system to manage the surge in glucose levels after a meal. Pancreatic β -cells play a major role in this system by taking up glucose from the blood and triggering insulin release into the bloodstream. Inside these cells, glucose uptake is managed by glucose transporters (GLUTs) – proteins that move to the β -cell surface when blood glucose levels rise and facilitate the entry of glucose into the cell to kickstart insulin release.

A new study from the Department of Developmental Biology and Genetics (DBG), Indian Institute of Science (IISc), Bengaluru shows how this process falters in Type 2 diabetes (T2D) and how restoring it could open new therapeutic avenues.

Current diabetes treatments largely target insulin action in peripheral tissues like muscle and fat, but this new work points to β -cell glucose uptake as a promising target.

“Most studies have looked at what happens after glucose enters the β -cell,” explains Anuma Pallavi, PhD student in DBG and first author of the study. “We focused on the step before that, the actual entry of glucose, and how this is disrupted in diabetes. By understanding the dynamics of these transporters, we can identify new points to intervene and improve β -cell function. If we can restore proper GLUT trafficking, we may be able to slow down disease progression and personalise therapies based on a patient’s metabolic state,” said the researchers.