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Perspective

Mitochondrial Integrity is affected by Excess Cellular Glucose Influx in the Absence of Txnip

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PERSPECTIVE

Chronic high blood glucose level results in detrimental effects on tissues systemically. However, the process by which excess glucose influx leads to damage at the cellular level globally is poorly understood.

The mitochondrion is a large intracellular organelle with many functions. Its high cellular abundance is a significant signal of its significance in cellular function. The quantity and shape of these cells varies depending on the cell type. So far, research has established that mitochondria play a variety of vital tasks, including ATP generation, Ca2+ homeostasis, and the creation of reactive oxygen species (ROS).

The mitochondrion has a unique evolutionary history, with proteobacteria presumed to be their ancestors. The existence of mitochondrial DNA is evidence of this. Mitochondrial DNA, unlike genomic DNA, is circular in shape and so resembles bacterial DNA in structure. While mitochondrial DNA codes for several key mitochondrial proteins, the majority of mitochondrial structural proteins are encoded in the cell nucleus, from which they are imported and sorted into the mitochondria. In addition to structure, mitochondrial DNA differs from genomic DNA in terms of copy quantity, with mitochondrial DNA frequently having fewer copies than genomic DNA.

Any biological system's ability to function depends on the integrity of its distinct functional cellular parts. To maintain quality, various pathways have evolved (QC). Individual routes work at various stages of quality control, from single amino acids to proteins, organelles, and complete cells. They form a complicated network that appears to interact in a hierarchical fashion. Imbalances in this network have negative consequences, including as illness and ageing.

Our work aims toward understanding the effects excess glucose has on organelles such as mitochondria assessed within an in vivo setting.

TXNIP negatively regulates glucose uptake by directly facilitating GLUT1-4 endocytosis while integrating metabolic and growth factor signals. Because of their unregulated glucose uptake into peripheral tissues, TXNIP KO mice are hypoglycaemic. Using TXNIP KO mice as a model of excessive cellular glucose uptake, we examined the glucose effect on mitochondrial function in vivo by examining thermogenesis in brown adipose tissue (BAT). Increased glucose uptake by BAT cells caused a shift of BAT lipid composition in cellular and mitochondrial membranes towards shorter, more saturated lipid forms; this shift subsequently affected mitochondrial integrity under stress, helping explain BAT thermogenesis defects. Additionally, this excess intracellular glucose resulted in reduced expression of fatty acid elongation, PUFA transport, and cardiolipin modification genes, further exacerbating lipid composition change. To further confirm our conjecture, these abnormal lipid phenotypes could be rescued using a ketogenic diet that limits the available peripheral tissue glucose uptake, strongly supporting our theory that changes observed in lipid composition and gene expression are due to excess cellular glucose uptake. Overall, our findings highlight that cellular damage caused by excess glucose influx into tissue cells occurs throughout the body far earlier than is currently defined by symptoms found through clinical diagnosis.

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