

MicroRNA-708 overexpression suppresses beta-cell proliferation

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There was an inverse correlation for *Nnat* expression with miR-708 in islets cultured at different [glucose concentrations](#) and in ob/ob mouse islets; this expression was reduced after [overexpression](#) of miR-708. MiR-708 overexpression impaired glucose-stimulated insulin secretion (GSIS), which was recovered by overexpression of *Nnat*, consistent with the role of *Nnat* in the secretory function of β -cells. In islets cultured at low glucose, miR-708 inhibition recovered GSIS. Overexpression of miR-708 suppressed β -cell proliferation and induced β -cell apoptosis.

"To fully understand whether changes in the expression of this microRNA are the cause or the consequence of β -cell dysfunction in these models still requires further investigation," the authors write.

(HealthDay)—Researchers have identified a novel mechanism of glucose regulation of β -cell function and growth by repressing stress-induced microRNA-708 (miR-708), according to a study published online Oct. 2 in *Diabetes*.

Júlia Rodríguez-Comas, from the Institut d'Investigacions Biomèdiques August Pi i Sunyer in Spain, and colleagues examined miRNAs that are modulated by glucose in mouse pancreatic islets.

The researchers found that miR-708 was the most upregulated miRNA in islets cultured at low glucose concentrations, which triggers a strong stress response. MiR-708 was also potently upregulated by triggering endoplasmic reticulum stress with thapsigargin and in islets of ob/ob mice. Treatment with the chemical chaperone 4-phenylbutyrate blocked low-glucose induction of miR-708. Neuronatin (*Nnat*) was identified as a potential glucose-regulated target of miR-708 in integrative analysis.

More information: [Abstract](#)
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