

Glucose-dependent insulin secretion in pancreatic β -cell islets from male rats requires Ca^{2+} release via ROS-stimulated ryanodine receptors

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© 2015, Public Library of Science. All rights reserved. This is an open access article, free of all copyright, and may be freely reproduced, distributed, transmitted, modified, built upon, or otherwise used by anyone for any lawful purpose. The work is made available under the Creative Commons CC0 public domain dedication. Glucose-stimulated insulin secretion (GSIS) from pancreatic β -cells requires an increase in intracellular free Ca^{2+} concentration ($[\text{Ca}^{2+}]$). Glucose uptake into β -cells promotes Ca^{2+} influx and reactive oxygen species (ROS) generation. In other cell types, Ca^{2+} and ROS jointly induce Ca^{2+} release mediated by ryanodine receptor (RyR) channels. Therefore, we explored here if RyR-mediated Ca^{2+} release contributes to GSIS in β -cell islets isolated from male rats. Stimulatory glucose increased islet insulin secretion, and promoted ROS generation in islets and dissociated β -cells. Conventional PCR assays and