
Tumwabudu Mungu Wetu Pdf Download [UPDATED]

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role for glucose-induced protein kinase C activation in islet beta-
cell regulation. Glucose regulates islet function in part through its
effect on insulin biosynthesis. Although the precise signal
transduction mechanisms of this glucose effect are not defined,
glucose has been reported to stimulate protein kinase C (PKC).
Because PKC is thought to influence cellular biosynthetic processes
in part through phosphorylation of lipids and proteins, we
examined the role of glucose-induced PKC in regulating islet beta-
cell replication. Islets were cultured overnight in 17 or 5.6 mM
glucose, then stimulated with 8.3 mM glucose for 30 min. PKC
activity and translocation were measured and insulin secretion was
assessed in cultured islets or beta TC6 cell line. Glucose
stimulation increased PKC activity in islets and beta TC6 cells, and
translocation from cytosol to membrane. Whereas PKC stimulation
was not sufficient to induce insulin biosynthesis, co-stimulation
with glucose did stimulate 3-O-methylglucose transport, reflecting
increased glucose uptake into beta-cells. Thus, glucose-induced
PKC activity is associated with increased glucose transport, and
glucose co-stimulation selectively influences glucose transport but
not biosynthetic processes. These results suggest that activation of
PKC is an obligatory part of the islet cell response to glucose, and
that glucose-induced PKC activity may influence the formation and
translocation of signal transduction proteins.Q: CheckBox in
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