EP869 Relationship between Insulin and somatostatin secretory response to Glucagon-like peptide-1 (GLP-1) and glucose concentration in perifused rat pancreatic islet cells.

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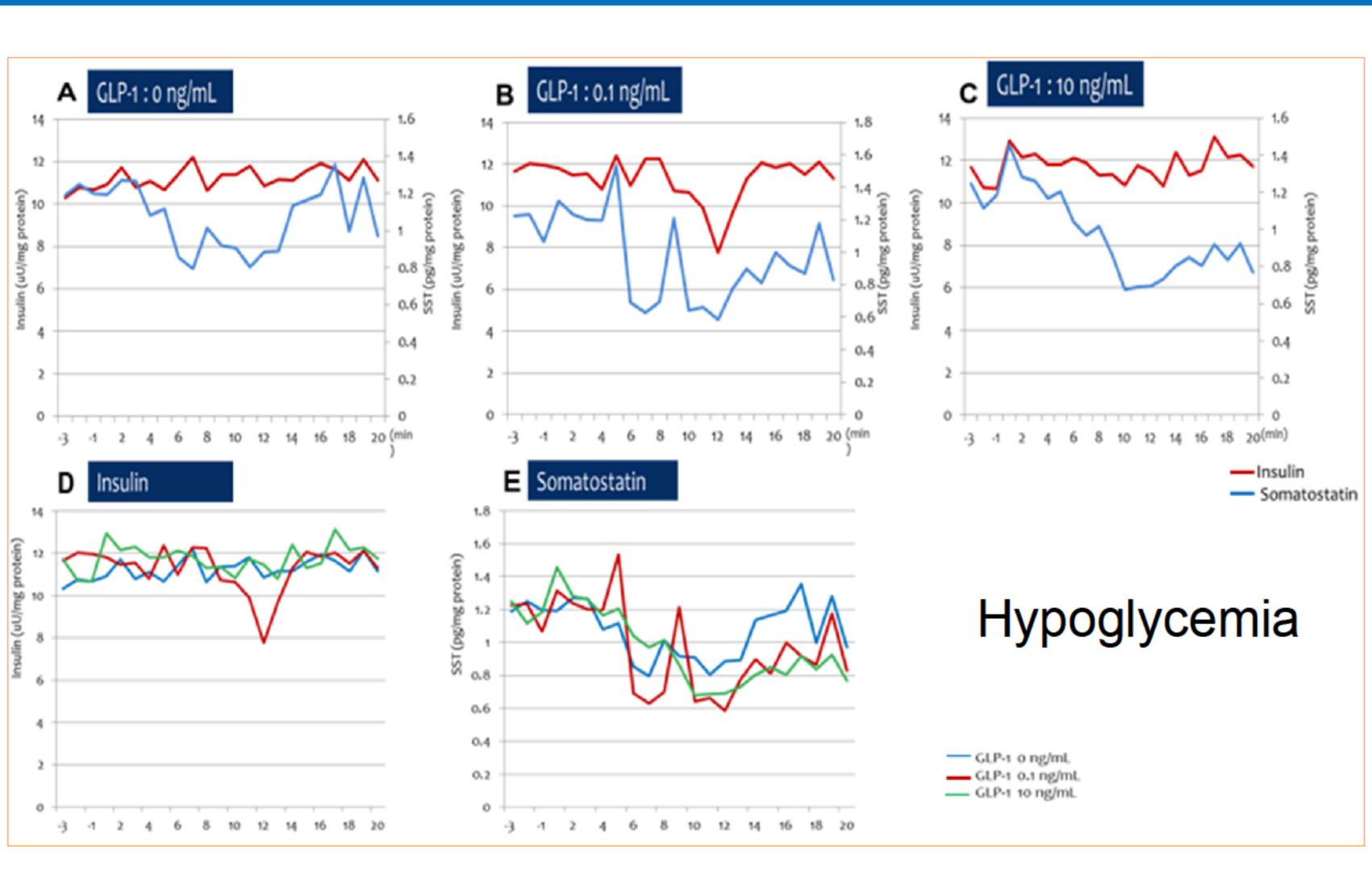
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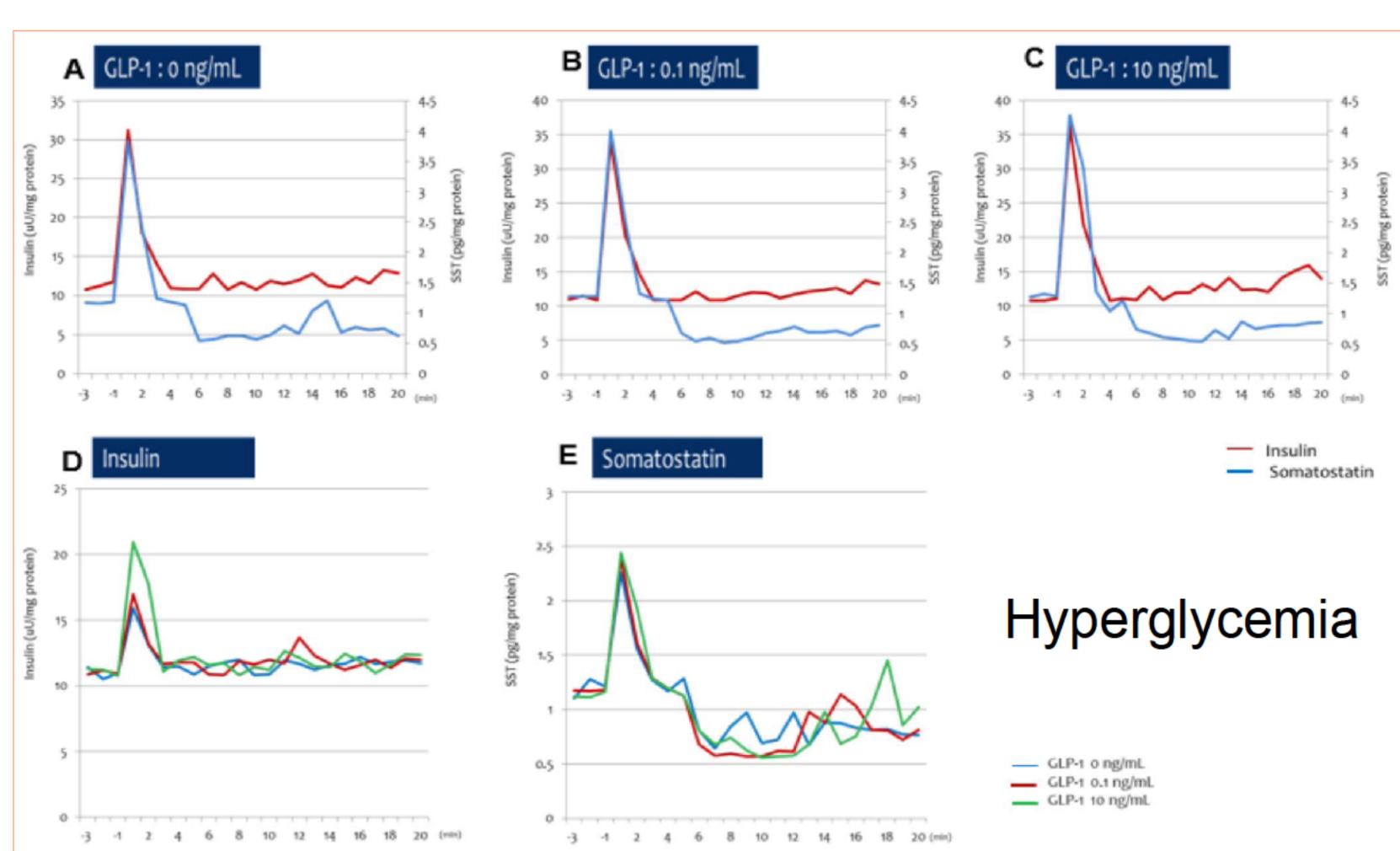
# METHODS

We isolated pancreatic islets from five 8-week-old male Sprague Dawley rats by collagenase digestion. The islets were incubated in RPMI1640 medium before experiments. The insulin and somatostatin were studied depending on glucose (2.7, 5.5 and mmol as hypo-, normo-, and hyperglycemic condition respectively) and GLP-1 concentrations (0, 0.1, and 10 ng/mL) in perifused isolated rat pancreatic islet cells. Because of duplication laboratory settings, statistical analysis did not proceed and analyzed tendencies. The hormonal analysis was conducted using ELISA kit.

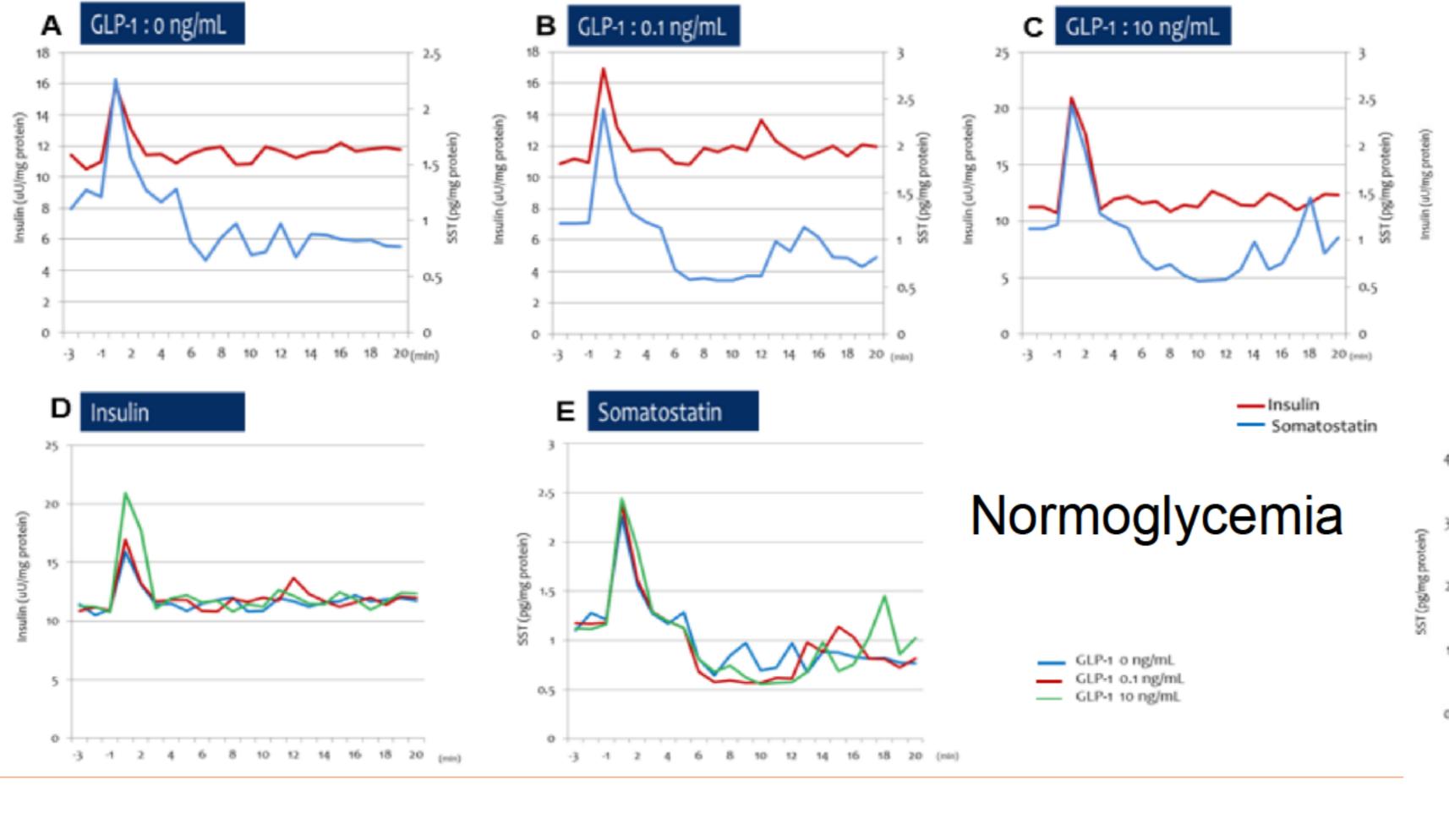
## **OBJECTIVES**

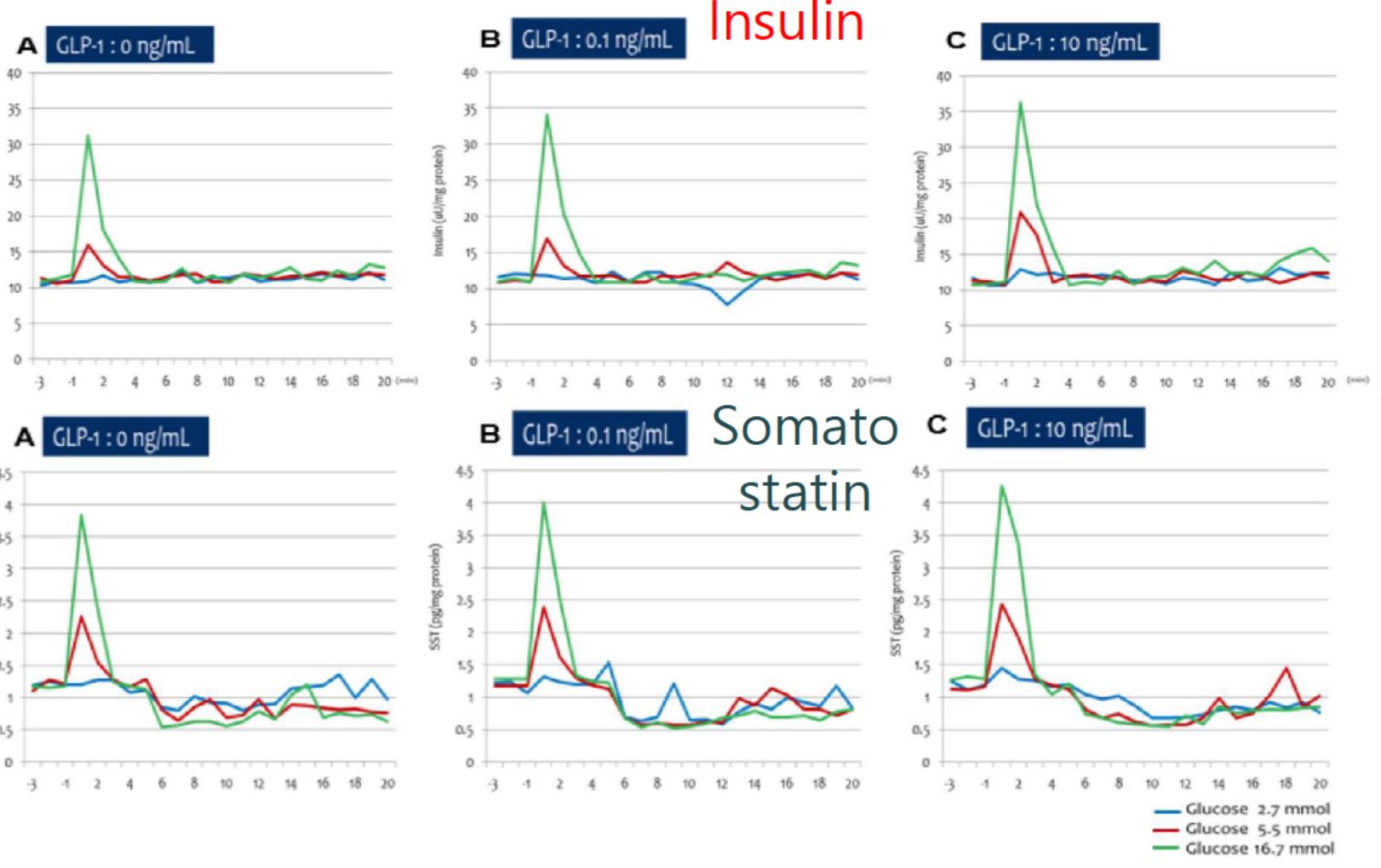
The exogenous somatostatin inhibits insulin secretion in pancreatic cell and could lead to hyperglycemia. But, Glucagon-like peptide-1 (GLP-1) has insulinotropic actions despite stimulating somatostatin secretion in  $\delta$  cell. So, we examined whether there is a time difference of insulin and somatostatin secretion after GLP-1 stimulation or secretion rate difference of insulin and somatostatin depending on GLP-1 concentration and glucose concentration inside the islets.





insulin and somatostatin secretion depending on GLP-1 concentration, GLP-1 0.1 ng/mL(B), GLP-1 10 ng/mL(C), insulin secretion (D), somatostatin secretion(E)





## RESULTS

At 2.7 mmol glucose, insulin and somatostatin did not respond to GLP-1 administration. At 5.5 and 16.7 mmol glucose, insulin and somatostatin secretion increased simultaneously as soon as GLP-1 administration. As GLP-1 concentration increased, so did insulin secretion but, somatostatin secretion was not affected by GLP-1 concentration. After a certain level of somatostatin was stimulated by GLP-1, somatostatin did not secreted any more. Whereas, both insulin and somatostatin secretion increased as increased glucose concentration. And somatostatin secretion decreased significantly compared with baseline after maximal secretion by GLP-1 stimulation.

### CONCLUSIONS

The time difference of insulin and somatostatin secretion after GLP-1 stimulation was not observed. And there was no secretion rate difference of insulin and somatostatin depending on GLP-1 concentration. The somatostatin secretion rate were affected by not GLP-1 concentration but glucose concentration. The significant decline of somatostatin compared with baseline after GLP-1 stimulated somatostatin maximally was observed newly and additional research is needed in the future to prove that the fact might be associated with insulinotrophic action of GLP-1.

