

In vitro insulin secretion by pancreatic tissue from infants with diazoxide-resistant congenital hyperinsulinism deviates from model predictions

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Supplemental Figure 1

Insulin secretion by individual focal lesions and control adjacent pancreas. The protocol corresponds to that in main Figure 2A that is shown again (mean \pm SEM) in upper left panel. Individual cases shown in the other panels are identified as in Table 1.

Supplemental Figure 2

Insulin secretion by individual focal lesions and control adjacent pancreas. The protocol corresponds to that in main Figure 2C that is shown again (mean \pm SEM) in upper left panel. Individual cases shown in the other panels are identified as in Table 1.

Supplemental Figure 3

Insulin secretion by individual DiCHI pancreas. The protocol corresponds to that in main Figure 2B that is shown again (mean \pm SEM) in upper left panel. Individual cases shown in the other panels are identified as in Table 1.

Supplemental Figure 4

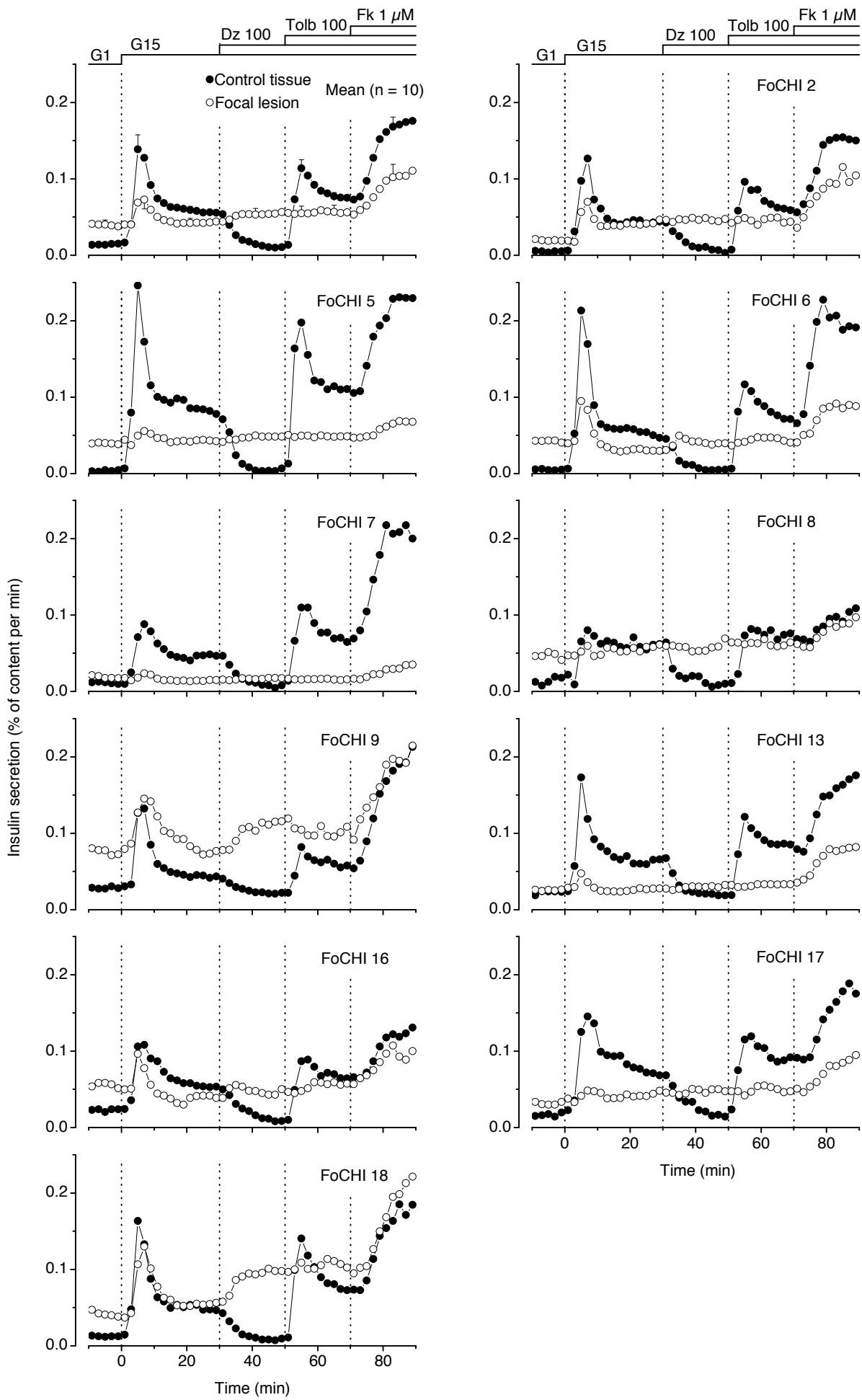
Insulin secretion by individual DiCHI pancreas. The protocol corresponds to that in main Figure 2D that is shown again (mean \pm SEM) in upper left panel. Individual cases shown in the other panels are identified as in Table 1.

Supplemental Figure 5

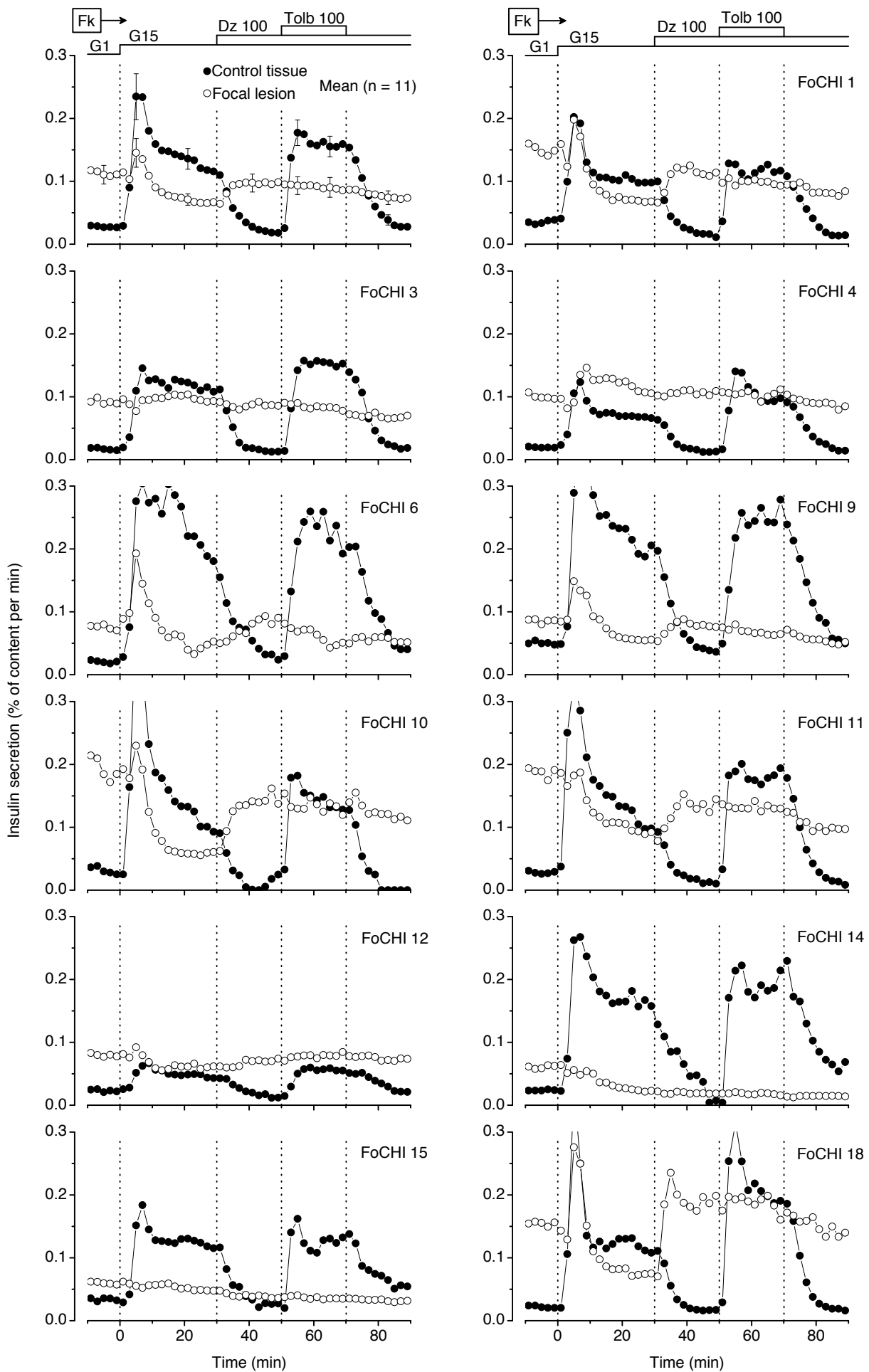
Changes in insulin secretion produced by 250 μ M pinacidil (a K_{ATP} channel opener) in focal lesions and adjacent normal pancreas from the same subjects (left panels), and in DiCHI pancreas (right panels). The upper two panels show mean responses \pm SEM. The other panels show responses by individual cases identified as in Table 1. The concentration of glucose was 3mM (G3) throughout. The drug had no effect in control pancreas and paradoxically increased insulin secretion in focal lesion and DiCHI pancreas.

Supplemental Figure 6

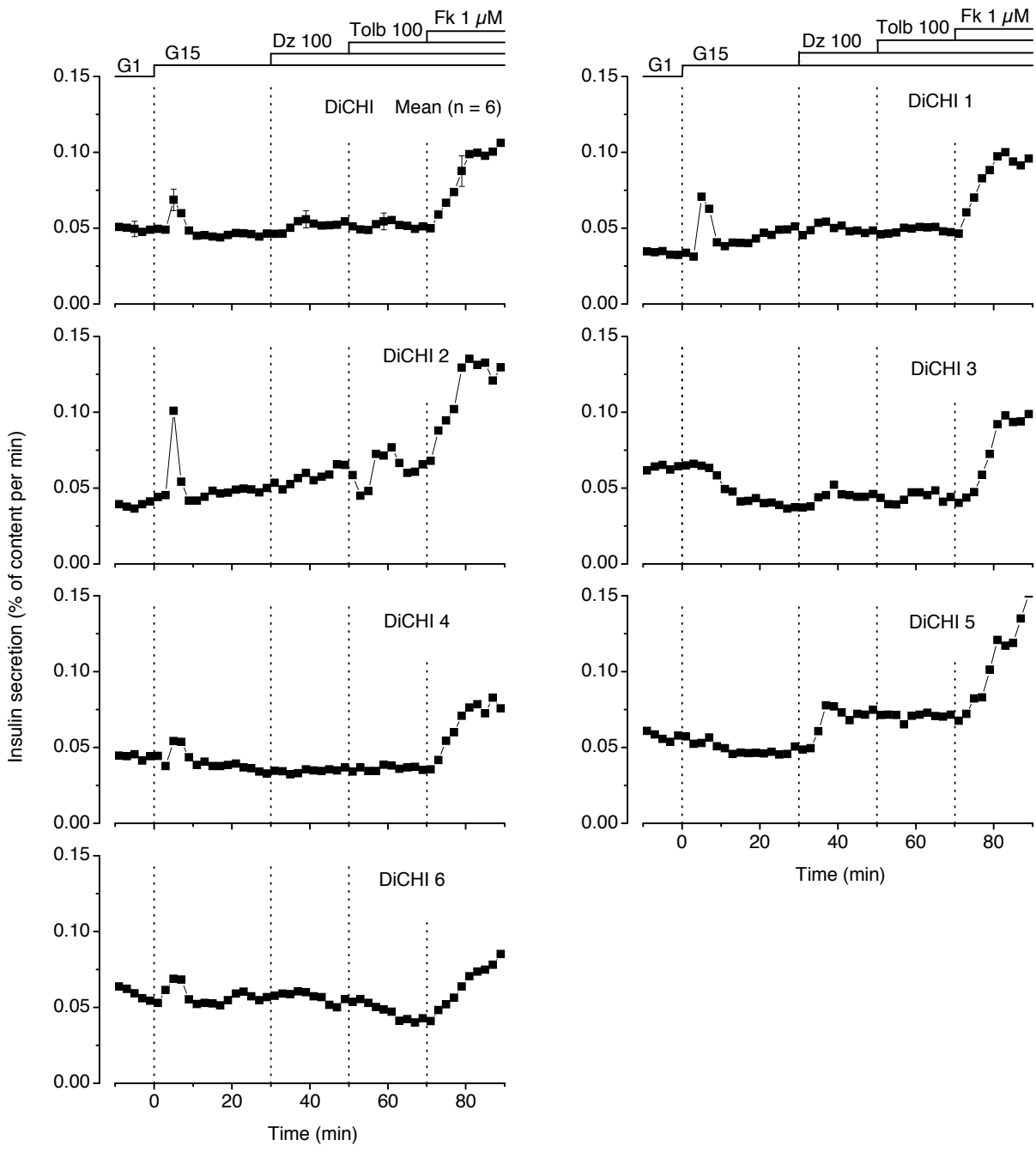
Changes in insulin secretion produced by 2mM Glutamine (Gln 2) and a mixture of four amino acids (1mM Alanine + 1mM Leucine + 1.5mM Glutamine + 1.5mM Lysine) in DiCHI 1 pancreas. The amino acids were added to a medium containing 1mM glucose alone (G1) in the upper panel, or 3mM glucose + 1 μ M forskolin (G3 Fk) in the lower panel. Note the difference in scale. The individual case is identified as in Table 1.



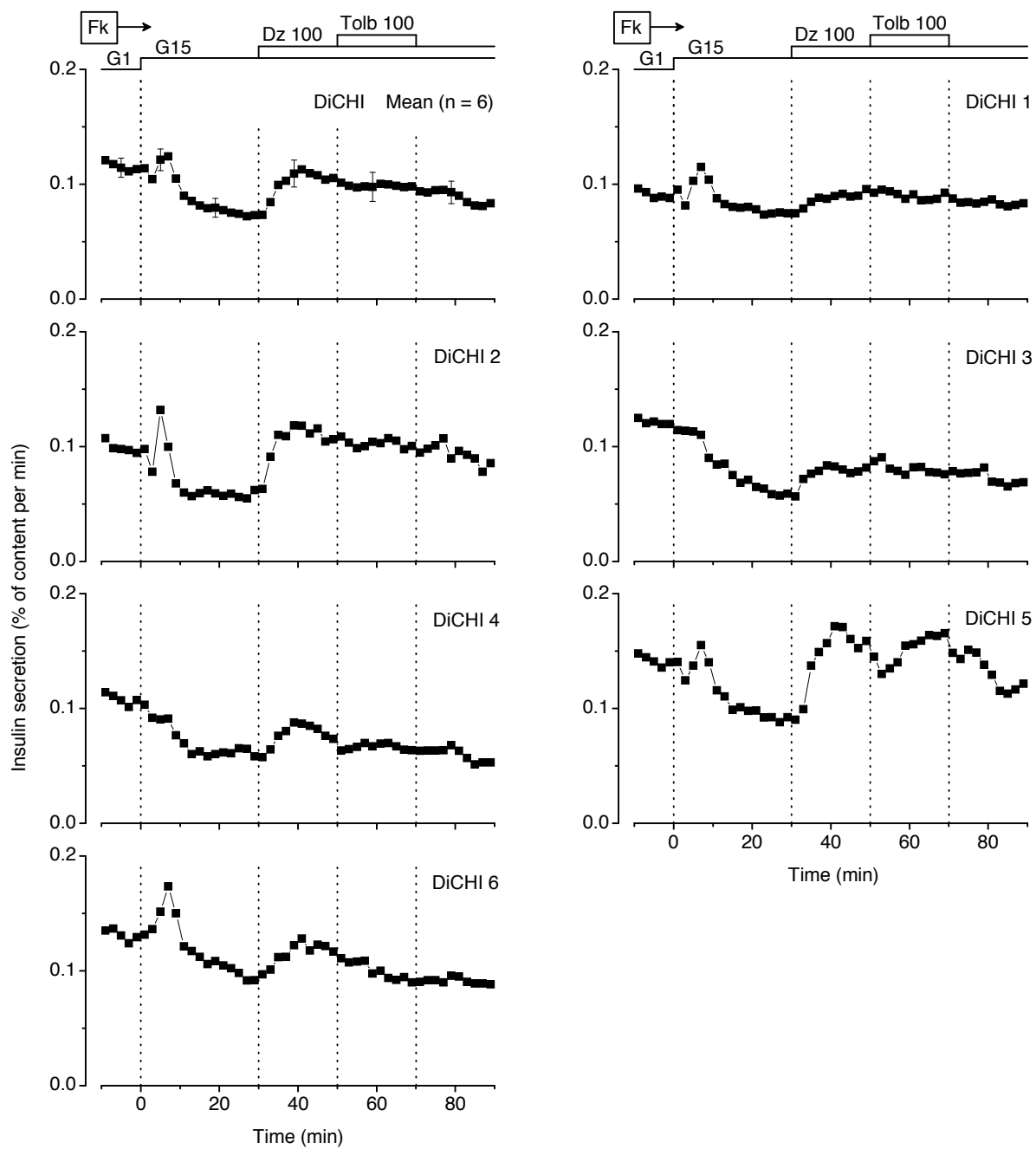
Supplemental Figure 1



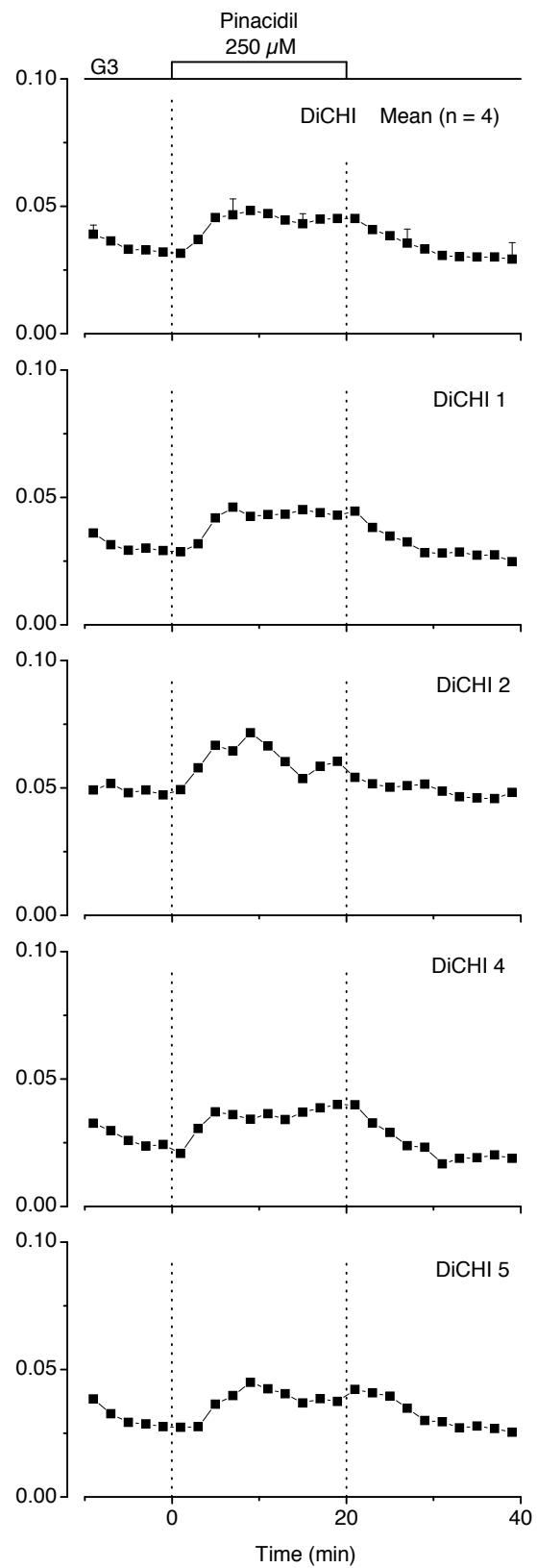
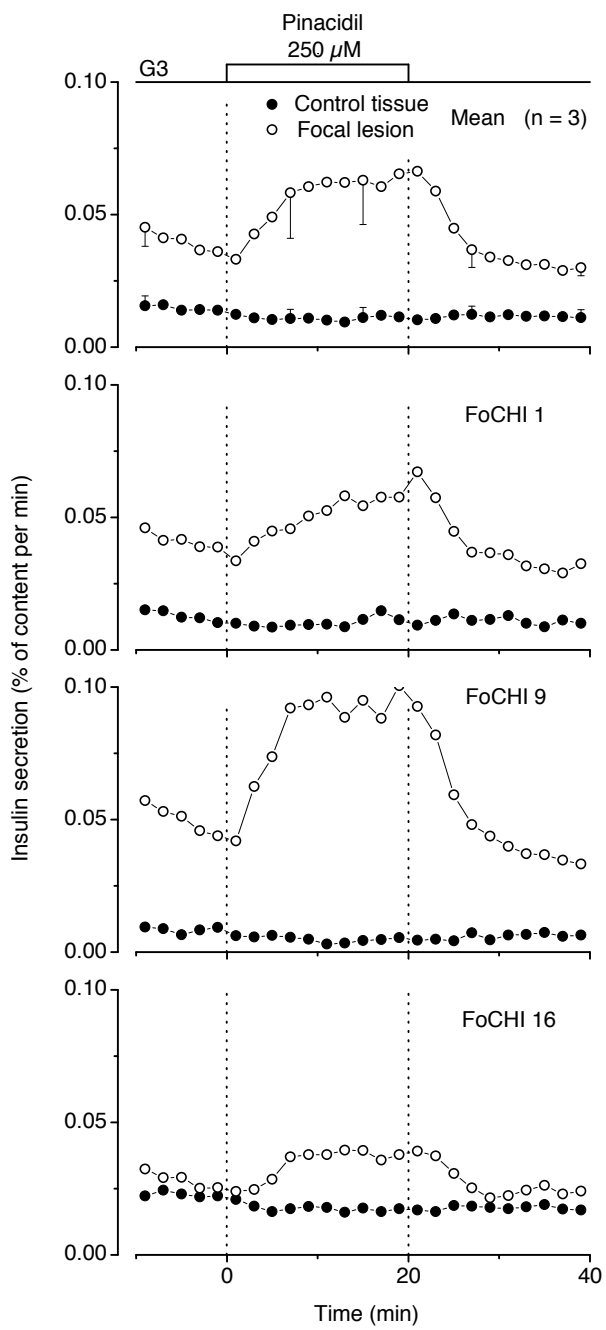
Supplemental Figure 2



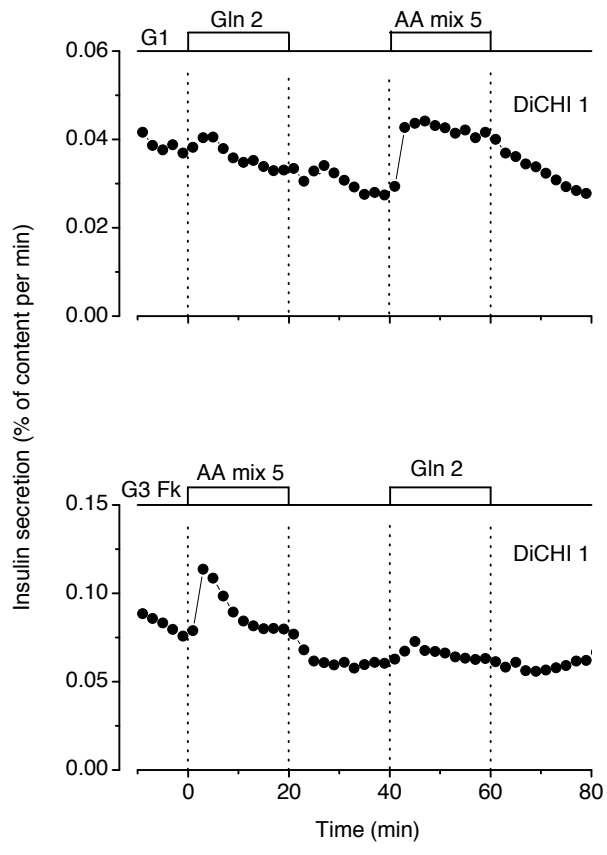
Supplemental Figure 3



Supplemental Figure 4



Supplemental Figure 5



Supplemental Figure 6