Glucose and Glucagon Do Stimulate Somatostatin Release from Isolated Pancreatic Islets

Dear Sir,

We would like to draw your attention to contradictory statements in relation to our work (references 1 and 2 in this letter correspond to references 12 and 11 in the review by K. Hermansen) reported in a recent review published in *Diabetologia* [3]. Indeed, Dr. Hermansen quoted our results correctly by stating that neurotensin "inhibited D cell secretion during stimulation with very high glucose concentration (23 mmol/l)", and by referring to our work [2] among other references when he wrote that "glucagon has consistently been reported to stimulate pancreatic somatostatin secretion." However, quoting the same references [1, 2], Dr. Hermansen contended that "since the D cells (of our preparation) of isolated islets were unresponsive to variations in glucose from 3 to 23 mmol/l, and to the addition of 2 µg/ml glucagon, the results must be viewed cautiously".

This is inconsistent not only with our data [1, 2] but also with what Dr. Hermansen wrote in his review. Therefore, the contention that "the results must be viewed cautiously" is not warranted and is based on misinterpretation of the data (Fig. 1).

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References

1. Dolais-Kitabgi J, Kitabgi P, Brazeau P, Freychet P (1979) Effect of neurotensin on insulin, glucagon, and somatostatin release from isolated pancreatic islets. *Endocrinology* 105: 256–260
2. Dolais-Kitabgi J, Freychet P (1979) Neurotensin inhibits glucose but not glucagon-induced insulin and somatostatin release in isolated islets. *Diabete Metab* 5: 275–277

Fig. 1. Effect of glucagon and glucagon plus neurotensin on somatostatin release. Pancreatic islets were incubated for 60 min in the presence of 12 mmol/l glucose alone (control) or in the presence of 12 mmol/l glucose plus neurotensin (NT) or glucagon at varying concentrations or both. Values are means of 18–26 individual observations obtained from eight separate experiments. * $p < 0.05$ versus control; ** $p < 0.005$ versus control

3. Hermansen K (1980) Secretion of somatostatin from the normal and diabetic pancreas: Studies in vitro, *Diabetologia* 19: 492–504

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Visual Flicker Discrimination in Diabetes

Dear Sir,

One simple means of quantitatively estimating the effect of diabetic complications on visual function is the evaluation of visual acuity. The intent of our efforts has been to develop a sensitive auxiliary method to evaluate another aspect of diabetic vision. We have completed a preliminary study in which measures of visual flicker discrimination were employed. Because diabetics have poorer colour vision than the general population and the deterioration tends to affect the ability to discriminate between blue and other colours [1, 2], we developed an optic stimulator which provides either primarily a blue or a red stimulus. Our objective was to determine whether flicker discrimination using blue light would

be a more sensitive indicator of impairment than would that with red light. The visual test field of our apparatus consisted of a background area (8° by 8°) with a centrally embedded red (660 nm) light-emitting diode which subtended 3/4°. A white light source produced illumination for the background and either a red (approximately 660 nm) or blue (approximately 480 nm) colour could be imparted to it by placement of filters between the eye and the background. Our method for determining the critical flicker fusion frequency (i. e. that frequency at which a flickering light will just appear solid) has been described previously [3]. Here, however, the procedure was done with a blue background as well as with a red background.