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Letters to the Editor

Haloperidol, and Insulin and Glucagon Secretion

Dear Sir,

I became aware of Dr. Samols difficulties in reproducing my results with Haloperidol when we discussed its action in September at the Diabetes Federation meeting in Vienna. I am afraid that I was rather sceptical at his suggestion that it was the 1% ethanol used for dissolving Haloperidol which might be responsible for the observed inhibition of glucagon and insulin secretion. Especially because studies performed concomitantly using the rat soleus showed that Haloperidol strongly inhibited fluxes of sodium and potassium, and this fitted excellently with my observations on the pancreas; and these muscle studies included control experiments with ethanol alone. However, unfortunately for me Dr. Samols is correct, ethanol does depress islet function, and my own current belated control experiments have verified this. I regret also that my original publication must have caused Dr. Samols quite a lot of extra effort in his laboratory.

I wish to utilize this opportunity to stress that there happily is only one further point where my results are at variance with those of Dr. Samols, namely on the action of acetylcholine on somatostatin. Dr. Samols finds an inhibitory effect [1] while I find a stimulatory action with a Merck® product of acetylcholine [2] – and fail to find any with the low concentration of 1 μmol/l of Sigma® [3]. In this case my different results are not due to an 'unspecific effect' of any solvent. A significant point in this context may be that there is large interindividual differences between pancreases in the sensitivity of the D cells to acetylcholine. Thus current experiments with the more stable carbamylcholine chloride (Sigma®) demonstrate that while a definite dose-related stimulation of somatostatin release is obtained in some pancreases, others are refractory. In contrast, insulin and glucagon responses are consistently obtained in all pancreases.

References

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