

Insulin rather than glucose homeostasis in the pathophysiology of diabetes

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Summary

Hepatic glucose efflux is very sensitive to changes in plasma-insulin and it is postulated that this provides an important feedback control of insulin secretion. In diabetes this hepatic "insulin sensor" increases the basal plasma-glucose until the impaired β cells are sufficiently stimulated to secrete normal basal insulin concentrations. Glucose regulation thus becomes of secondary importance to the maintenance of basal insulin secretion, which is teleologically needed for the "anabolic" requirements of cell growth. This hypothesis provides an explanation for the "normal" basal plasma-insulin concentrations found in diabetes in spite of impaired β -cell function. This maintenance of basal insulin secretion accounts for the discrepancy between the marked hyperglycaemia and minimal ketosis of maturity onset diabetic patients.