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Beta Cell Deficiency in maturity onset diabetes

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Diabetics have normal basal plasma insulin levels, and a raised basal plasma glucose which is characteristic for each person. It has been suggested that only stimulated and not basal insulin secretion is deficient in diabetes. We show that both are impaired and postulate that in diabetes insulin control of hepatic glucose efflux acts as insulin 'sensor'. This causes the basal plasma glucose to rise until the reduced number of beta cells are sufficiently stimulated to secrete normal basal insulin levels. Thus glucose regulation is of secondary importance to maintenance of basal insulin secretion. The increased plasma glucose load further stresses the remaining beta cells which then have to operate nearer their maximal capacity. The degree of basal hyperglycaemia provides a bioassay of the decrease in insulin secretion capacity, enabling one to estimate the number of functioning beta cells. The observed insulin secretion in diabetes is similar to that predicted from this estimate. The height of the basal plasma insulin gives a measure of the degree of insulin resistance associated with obesity, and from the estimated beta cell defect one can indicate the extent to which dieting alone would reduce the fasting plasma glucose.