

## Beta Cell Function During Insulin or Chlorpropamide Treatment of Maturity-onset Diabetes Mellitus

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### Summary

Maturity-onset diabetic patients usually have raised overnight-fasting plasma glucose levels associated with 'normal' basal plasma insulin levels. The basal hyperglycemia is proportional to the degree of insulin deficiency. Basal insulin or C-peptide levels become subnormal if normal fasting plasma glucose levels are attained with insulin. Basal hyperglycemia is probably a compensatory response to maintain near-normal basal insulin levels. A logical therapy of maturity-onset diabetes is to produce basal normoglycemia by means of a constant basal insulin supplement, which can be provided by ultralente insulin. The reduced insulin response of diabetics to intravenous glucose is slightly increased when basal normoglycemia is established, suggesting that the high plasma glucose levels compromise beta cell function. The insulin response to meals in a mild diabetic is not affected by mild hyperglycemia but can be depleted if gross hyperglycemia occurs. Maintenance of normoglycemia then allows beta cell "recovery". In mild diabetics (c.<9 mmol per liter basal plasma glucose), chlorpropamide sufficiently stimulates beta cell secretion so that basal normoglycemia can be produced. The C-peptide response to meals is improved, whereas comparable reduction of the plasma glucose with insulin does not alter the meal response. Thus basal normoglycemia can be produced by "resting" beta cells with a basal insulin supplement or by stimulating them with sulfonylurea therapy.