Relative contributions of insulin deficiency and insulin resistance in maturity-onset diabetes.

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The effects of raised insulin resistance and decreased beta-cell function on basal plasma glucose and insulin concentrations have been estimated by means of a mathematical model which uses the available data on control of glucose and insulin flux. The model shows that near-normal basal plasma insulin levels could be maintained by means of basal hyperglycaemia when beta-cell function is deficient. The hyperglycaemia does not become pronounced until more than 80% of beta-cell function has been lost. However, when only 5-10% of beta-cell remains, plasma insulin levels fall in spite of hyperglycaemic stimulation. Conversely, raised insulin resistance in the absence of decreased beta-cell function induces only a small rise in basal plasma glucose concentrations, which stimulates a compensatory increase in basal plasma insulin concentrations from a normal beta-cell capacity. However, if the beta-cell dysfunction is sufficient to induce a basal plasma glucose concentration of 5-6 mmol/l, a slight rise in insulin resistance causes pronounced basal hyperglycaemia.