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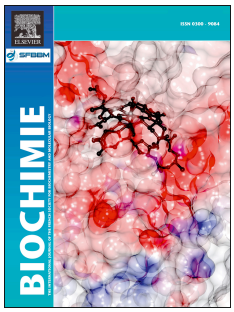


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# Nutrient regulation of pancreatic β-cell proliferation

Valentine S. Moullé[1,2,*], Julien Ghislain[1,2,*] and Vincent Poitout[1,2,3,4]

## Abstract

Excess consumption of energy-dense foods combined with a sedentary lifestyle is driving an obesity epidemic. Although obesity is closely associated with insulin resistance, most individuals meet the insulin demand by increasing their functional β-cell mass. Those who eventually develop type 2 diabetes are distinguished by a failure in this compensatory process. Although a causal role of insulin resistance in compensatory β-cell responses has received considerable experimental support, precisely how the β cell senses changes in the metabolic environment is still unknown. As metabolism of glucose, lipids and amino acids is profoundly altered in obesity, it is not surprising that these nutrients are conspicuous among the factors proposed to contribute. In this review we summarise our understanding of the role of nutrients, in particular glucose, fatty acids and amino acids in β-cell compensation with a particular emphasis on their relation to insulin resistance-induced factors and their underlying mechanism of action. Finally, we describe the concept of epigenetic programming and review recent studies illustrating how the status of the β cell epigenome is a product of its nutrient environment, and how metabolic programming of the β cell contributes to diabetes risk.