Abstract

In diabetes mellitus type 2 (DMT2), malfunction and apoptosis of -cell provoke a deficient insulin secretion. Generally, has been sustained that -cell function is severely compromised in type 2 diabetes before the disease appears and then continues to decrease linearly with time. Diversionary bariatric procedures such as gastric bypass, biliopancreatic diversion, one anastomosis gastric by-pass (BAGUA) and others that bypasses the foregut, induce a rapid non-weight-loss-associated improvement in glycemic control, especially if treated early before irreparable -cell damage has occurred. The antidiabetic effect of bariatric operations is likely due to the improvement in the hormonal dysregulation associated with the development of diabetes. Now we know that the bariatric surgery through the reorganization of the gastrointestinal tract can affect to -cells mass homeostasis, stopped apoptosis and stimulate the replication and neogenesis. These effects are caused mainly by three stimuli: caloric restriction, rapid transit of food to the ileum and the exclusion of an intestinal portion including the stomach, duodenum and part of the jejunum. Several mechanisms have been proposed for this exciting effect that may provide key insights into the pathogenesis of type-2 diabetes. All of these mechanisms include from gut hormones such as ghrelin to second messengers such as AKT system or protein kinase B. Although not all the processes involved in the homeostasis of -cells are clear, we can explain some of the effects of bariatric surgery exerted on this important set of endocrine cells, which are essential in diabetes control.

Keywords