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The effect of Osteocalcin on restoring pancreatic islet function

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Aging is the predominant risk factor for chronic diseases, such as type 2 diabetes (T2D), that affect quality of life and longevity. However, organismal chronological age is not synonymous with biological age, and evidence suggests that different organs, within the body, age at different rates, depending on the unique cellular properties and function of the organ. Furthermore, the physiological demands on an organ, such as the demands on the pancreas from metabolic stress induced by obesity and T2D, may also influence the rate of aging. T2D is partly characterized by dysfunctional insulin secretion from pancreas islets and the continual functional decline in the compensatory increased pancreatic β-cell proliferation (i.e., mass) in response to increased blood glucose levels. Aging, itself, leads to increased blood glucose levels, resistance to insulin, decreased metabolism, and limited exercise capacity, which also contributes to weight gain and obesity, but it is the inability of the pancreas to compensate for insulin resistance that determines whether T2D develops. Although lifestyle changes, such as physical exercise and body mass index maintenance, can reverse T2D, at least temporarily, and limit some of the morbidities associated with aging, aging-related physical and mental frailty can hinder an aging individual's ability to exercise. The exercise-induced bone-derived hormone, osteocalcin (OCN) has been shown to promote insulin secretion and to enhance pancreatic β-cell mass and function. However, bone loss and frailty in aging individuals can limit their exercise capacity, and the decreased ability to exercise coincides with the decline of OCN levels with age. This study tested the effect of OCN on pancreatic β-cell senescence expression and pancreatic islet function. Several senescence markers (e.g., CDKN2a, MAPK14, IGF1R, and CD99) have been used to identify aged β cells, but whether the aged β cells and the loss of mature cell identity can be reversed is not known. Both aging and obesity are associated with the loss of β-cell identity, defined as the failure to express pancreatic β-cell transcription factors (e.g., FOXO1, FOXA2, SUR1, PDX1, NKX6.1, PAX6, NEUROD1, PCSK1, MAFA, and UNC3), which directly regulate the transcription of insulin and are associated with β-cell function. In this study our data show that OCN decreases markers of senescence in pancreatic β-cells and restore pancreatic β-cell transcription factors which as result improves human pancreatic islet function. Our data derived from obese, non-obese, diabetic, and compared to non-diabetic function.

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