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A Review on How Diabetes Cause due to Obesity

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Abstract

Type -2 Diabetes can be caused by having too much fat on your body, and the risk of getting Type 2 diabetes goes up in a straight line with your BMI. As a result, the prevalence of Type 2 diabetes has also increased in tandem with the rise in obesity rates worldwide. Adiposity-induced alterations in cell function, adipose tissue biology, and multi-organ insulin resistance are the complex cellular and physiological mechanisms that are linked to obesity and Type 2 diabetes. These alterations are frequently ameliorated and can even be normalized with adequate weight loss.

Keywords: Obesity; Diabetes; Type 2 diabetes

Introduction

The body's primary fuel reserve is adipose tissue, which also serves as an important source of transportable energy, which is essential for survival when food is scarce [1]. Triglycerides are five times more efficient as a fuel per unit mass than glycogen due to their high energy density and hydrophobic nature; Glycogen is stored intracellularly as a gel that contains approximately 2 g of water for every gram of glycogen and only produces 4.1 kcal per gram when oxidized, whereas triglycerides are compactly stored as an oil within adipocytes and produce 9.3 kcal per gram when oxidized. The size of the mass of adipose tissue determines how long a person can survive during starvation. Men who are lean die when they lose more than 35% of their body weight in 60 days, whereas people who are extremely obese can tolerate long-term fasting; A man with extreme obesity who fasted for 382 days and only ate fluids, vitamins, and minerals had the longest known fast. He lost 60% of his body weight without experiencing any negative effects. In addition, adipokines and exosomes, which are produced and secreted by adipose tissue and play a role in the regulation of important physiological functions like appetite, reproduction, and insulin action, are produced and secreted by the tissue [2].

Literature Review

Insulin resistance, atherogenic dyslipidemia (high plasma triglyceride and low plasma HDL-cholesterol concentrations), nonalcoholic fatty liver disease (NAFLD), cell dysfunction, prediabetes, and Type 2 diabetes are among the metabolic abnormalities and diseases that are brought on by an excessive amount of body fat. A progressive increase in the risk of developing Type 2 diabetes is typically correlated with a rise in BMI, which is a measure of adiposity [3]. However, the distribution of fat and triglycerides alters the likelihood of metabolic dysfunction caused by adiposity. Compared to people with a lower body (gluteofemoral) fat phenoType, obese individuals with a predominant increase in upper body fat (abdominal subcutaneous and intra-abdominal fat), intrahepatic triglyceride content, intramyocelluar lipid content and pancreatic fat are more likely to develop Type 2 diabetes. In fact, in people who are lean, overweight or obese an increase in gluteofemoral body fat mass is linked to lower plasma triglyceride levels and higher HDL-cholesterol levels, lower fasting blood glucose and insulin levels, increased oral glucose tolerance and insulin sensitivity, and a lower risk of Type 2 diabetes [4].

Multi-organ insulin resistance and a decline in cell insulin secretory function are the causes of Type 2 diabetes. Due to the fact that obesity has an effect on both the action of insulin and the function of cells, it is likely that the recent rise in the prevalence of Type 2 diabetes is due to the global increase in obesity. The connection between excess adiposity and Type 2 diabetes as well as the therapeutic metabolic effects of weight loss (fat loss) will be discussed in this article.

Cell physiology and insulin kinetics

The pancreatic cells secrete insulin directly into the portal vein, where it is transported to the liver, the primary location for insulin clearance. The balance between the rate of insulin secretion and insulin removal by the liver and extrahepatic tissues determines plasma insulin concentration. During the first transit, approximately 50% of the insulin delivered to the liver by cells is eliminated, and another 20% is eliminated during subsequent transits; The kidneys (about 20%) and skeletal muscle (about 10%) remove the remaining 30% of insulin that is secreted. In obese individuals, both increased pancreatic insulin secretion and decreased fractional extraction and clearance of portal and peripheral plasma insulin are responsible for the rise in basal and postprandial plasma insulin concentrations [5].

The function of pancreatic cells is a crucial factor in determining whether obese individuals will develop Type 2 diabetes [6]. Obese individuals without Type 2 diabetes typically have higher plasma insulin concentrations and rates of insulin secretion during basal conditions and after consumption of glucose than lean individuals. The resistance to insulin action is frequently overcome by this increase in insulin secretion rate and plasma insulin concentration, allowing normal oral glucose tolerance and fasting blood glucose concentrations. However, prediabetes and eventually Type 2 diabetes result from a progressive decline in glycemic control caused by a decline in cell function.

Discussion

The secretion of insulin may be influenced by the number of pancreatic cells themselves, both at rest and after eating. People who are obese have approximately 50% more pancreatic cell mass than people who are lean, which is often expressed as the relative volume (the ratio of the cell area to the exocrine area measured at autopsy).

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Cell apoptosis, on the other hand, results in a relative cell volume that is approximately 50% smaller than that of lean individuals in those with impaired fasting glucose or Type 2 diabetes [7,8]. Obese people have higher basal and postprandial insulin secretion rates than lean people when both groups are matched on insulin sensitivity, so it is unlikely that insulin resistance alone is the cause of the obesity-related increase in cell mass.

Conclusion

Due to the fact that it causes both insulin resistance and cell dysfunction, obesity is a major risk factor for prediabetes and Type 2 diabetes particularly when it is associated with increased abdominal and intra-abdominal fat distribution. As a result, the prevalence of Type 2 diabetes has also increased as a result of the worldwide rise in obesity. New therapeutic approaches to both preventing and treating this crippling condition could be developed if we gain a deeper comprehension of the mechanisms underlying the negative effects of excess body fat on the factors that contribute to the pathogenesis of Type 2 diabetes. Adipose tissue biology has undergone changes that have been linked to obesity, insulin resistance, and cell dysfunction in a series of human and mouse models. Adipose tissue fibrosis (increased rates of fibrogenesis and expression of genes involved in the formation of the extracellular matrix), inflammation (increased proinflammatory macrophage and T cell content and the production of PAI-1), and the production of exosomes that have the ability to cause insulin resistance are examples of these changes. However, without a mechanism for adipose tissue communication with other organs, none of these factors can influence systemic metabolic function. PAI-1, adiponectin, FFAs, and exosomes, among other adipose tissue secretory products that are released into the bloodstream, may be involved in this signaling process. However, more research is needed to fully assess their clinical significance. Adipose tissue, the liver, muscle, and pancreatic islets may also interact with one another to cause insulin resistance and hepatic steatosis. If there is sufficient restoration of cell function, reducing body fat mass without surgical removal can ameliorate or normalize obesity-induced metabolic dysfunction and even achieve diabetes remission.

Acknowledgement

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Conflict of Interest

None

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