German M, et al. J Diabetes Metab Disord 2020, 7: 036
DOI: 10.24966/DMD-201X/100036

HSOA Journal of Diabetes & Metabolic Disorders

Mini Review

Diabetes and Obesity - Inextricable Diseases

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Abstract

Diabetes and obesity are global epidemics and the prevalence of both has increased in parallel. The relationship between obesity and diabetes is clear as nearly 90% of people with diabetes have obesity. Multiple factors contribute to obesity and include dietary, environmental and iatrogenic causes all of which can affect metabolism directly and independently of caloric intake alone. Studies focused on the pathophysiology of the relationship between diabetes and obesity has led to innovations in diabetes care with a focus on obesity. Weight loss is now recommended in all patients with diabetes and adjuvant pharmacological and surgical therapies are available and often recommended, to supplement lifestyle changes for weight loss. Diabetes claims the lives of hundreds of thousands of people every year and the financial burden of diabetes is staggering at over 200 billion dollars annually. Control of obesity is imperative in controlling diabetes.

Keywords: Diabetes; Hormones; Obesity; Weight loss

Introduction

The relationship between type 2 diabetes and obesity has been demonstrated repeatedly in recent decades. Currently the prevalence of obesity in adults in the United States is 39.8% and of these 34 million people, 89% have overweight or obesity. The prevalence of diabetes and obesity is also on the rise worldwide. Globally, there are 422 million people with diabetes and 1.9 billion people with overweight or obesity [1,2]. This represents a quadrupling and tripling of diabetes and overweight or obesity respectively in the last 50 years [1,2]. Although there has been great advancement in pharmacological therapy for treatment of diabetes, only half of those with diabetes have HgbA1c at goal of less than 7.0% [3]. Uncontrolled diabetes is associated with micro vascular (nephropathy, neuropathy and retinopathy) and macro vascular (stroke, myocardial infarction, peripheral arterial disease) complications. Over a third of patients with diabetes have chronic kidney disease and tens of thousands of patients with diabetes undergo lower extremity amputations every year. Diabetic retinopathy is the leading cause of blindness in the United States. The National Diabetes Statistics Report 2020 estimates over 1.5 million people with diabetes were hospitalized for ischemic heart disease or stroke in 2016. These complications are a massive financial burden. The direct cost of diabetes is over 200 billion dollars annually and has increased by almost 50 billion dollars in the last five years [3]. Ultimately, despite current efforts to treat diabetes and prevent complications, it is the seventh leading cause of death in the United States. In 2017 alone, diabetes was listed as the underlying or contributing cause of death of over 250,000 people [3,4].

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Citation: German M, Simonetti J (2020) Diabetes and Obesity - Inextricable Diseases. J Diabetes Metab Disord 7: 036.

Received: November 16, 2020; **Accepted:** November 19, 2020; **Published:** November 26, 2020

Why the weight gain

The reasons for weight gain are multifactorial and caused by a complex interplay between genetics and the environment [5]. It is rare that a person with obesity or overweight has not attempted a diet and exercise plan. Despite these attempts, only about 50% of people are able to maintain weight loss for 1 year [6]. While obesity is ultimately the result of more calories consumed than burned, the reasons for this persistent discrepancy are complex. Genetics play a significant role in Body Mass Index (BMI) variance and in response to the environment, namely food intake and physical activity. Studies in twins have demonstrated that 50-70% of the BMI variance may be explained by genetics [7]. Human genetic studies using the genome-wide association approach have revealed severe monogenic forms of obesity such as congenital leptin deficiency and melanocortin-4 receptor deficiency [8,9]. Genetics alone however cannot explain the increased incidence of obesity in recent decades. There is a natural drive for food consumption and pleasure is derived from eating, an adaptation that has ensured our survival. Animal studies have long suggested that dopamine is involved in mediating feeding behavior as rats in which the dopaminergic neurons were destroyed developed aphagia and died within a week [10]. Palatable foods, especially those high in sugar and fat, elevate dopamine levels in the nucleus accumbens of the ventral striatum, an important part of the reward pathway [11,12]. The pleasurable effect of food is a motivating factor that can override homeostatic signals [13-15]. A predilection for high sugar and high fat foods has not gone unnoticed by the food and entertainment industries. High sugar and calorically dense foods are more readily available, cheaper and often in larger portions than more nutritious and lower calorie foods [16,17]. Accessibility to palatable energy-dense foods is a major risk factor for obesity [18]. The average American today consumes more than 500 calories daily from added sugar alone which is found in suspected sources like soda but also in presumed healthier options like salad dressing, granola and yogurt [19]. Not only are highly palatable foods rewarding, but individuals with obesity experience greater reward from food as compared to their lean counterparts. This was shown in a study using Functional Magnetic Resonance Imaging (FMRI) looking at activation of the gustatory cortex and in

somato sensory regions of the brain in response to anticipated intake of a chocolate milkshake (vs. a tasteless solution) and to actual consumption of milkshake (vs. a tasteless solution) [20]. As more calories are being consumed, fewer calories are being burned. With technological advancements, work and leisure time are comprised of more sedentary activities which leads to decreased energy expenditure. A sedentary lifestyle can also cause physiological changes which lead to decreased resting metabolic rate such as decreased mitochondrial functionality and decreased lean muscle mass [21]. Iatrogenic contributors are often overlooked; beta blockers, antidepressants, glucocorticoids, injectable progestin's, antipsychotics and antiepileptic drugs are all associated with weight gain [22,23]. In addition, anti-diabetes medications themselves, including insulin and sulfonylureas, can cause weight gain [24].

Pathophysiology of obesity and diabetes

The body manages a discrepancy between calories consumed and burned by storing the energy as fat in adipocytes. While functioning as an energy source, these cells comprise an endocrine organ which produces hormones responsible for regulating insulin sensitivity, appetite and energy balance. Increased fat mass increases serum free fatty acids which stimulates insulin secretion from pancreatic beta cells while causing insulin resistance of skeletal muscle. Insulin resistance leads to elevated blood glucose and further insulin secretion. Elevated insulin level elicits insulin resistance in the liver and adipose tissue which then also contributes to the cycle of elevated blood glucose and insulin secretion. Elevated blood glucose causes a state of constant pancreatic beta cell stimulation and diabetes develops when the pancreas is unable to meet insulin demand resulting in hyperglycemia. Hormones produced by adiposities, like leptin and asprosin, are increased in obesity. In normal circumstances, leptin inhibits insulin production, increases skeletal muscle metabolism of glucose and inhibits hepatic glucose production. However, in patients with obesity, resistance to leptin develops, thereby leading to worsening hyperglycemia due to uninhibited insulin production, decreased skeletal glucose metabolism and increased hepatic glucose production. Obesity does not seem to induce resistance to asprosin, a recently identified adipose hormone that stimulates hepatic glucose production and peaks during fasting thereby causing hyperglycemia even in the absence of additional glucose and fatty acid consumption. Ultimately hyperglycemia results in beta cell death and exogenous insulin dependence [25,26].

Weight loss as treatment for diabetes

Effective diabetes treatment is multifaceted and lifestyle modification is essential for diabetes control. The American Diabetes Association (ADA) recommends 5% weight loss in all patients with type 2 diabetes [22]. Weight loss in those with diabetes is uniquely challenging as many of the medications used to treat diabetes, in particular the older classes, also cause weight gain. Some studies suggest that patients can gain as much as 10 kg in a relatively short period (3 to 6 months) after initiating treatment with insulin, sulfonylureas, and other insulin secretagogues like glitinides and thiazolidinediones [24,27]. Medications like sulfonylureas and insulin work by filling the gap between insulin required for adequate cellular glucose transport and insulin actually produced. Glucose that would have otherwise remained in serum is then available for metabolism and fat storage which contributes to insulin resistance. Metformin as well as newer classes of diabetes medications such as Glucagon like Peptide-1 (GLP-1) receptor agonists, dipeptidyl peptidase-4 inhibitors and sodium-glucose co-transporter 2 inhibitors are weight neutral or can lead to weight loss [28]. As such, their use is preferred prior to considering weight promoting anti-glycemic medications like insulin. Trials comparing GLP-1 agonists and other anti hyperglycemic agents have shown weight loss in some subjects ranging between 5.5 and 8 kg. Metformin and SGLT-2 inhibitors produce more modest weight loss, in the 1 to 3kg; these agents have not been studied in the setting of concomitant behavioral therapy, and the full weight loss potential is therefore not yet known [27]. The ADA recommends the use of pharmacological weight loss therapies (e.g. liraglutide, phentermine, phentermine/topiramate, naltrexone/bupropion) for treatment of obesity in diabetics with BMI 27 and greater. In patients with BMI > 40 and diabetes, the ADA recommends bariatric surgery [22]. These recommendations highlight the importance of managing obesity in the treatment of diabetes given the dual benefit of weight loss and glycemic control.

While most trials in the United States have focused on preventing diabetes with weight loss from lifestyle changes [29,30], a trial in Scotland and the United Kingdom has evaluated the effect of significant weight loss from lifestyle changes in people with a diagnosis of diabetes [31]. In this study all diabetes medications were discontinued on initiation of a restricted calorie diet and increase in physical activity. At 12 months, 149 participants had completed the study; 24% reached goal weight loss of 15 kg with mean weight loss of 10 kg and HgbA1c decreased by an average of 0.9%. While standard protocols for blood glucose monitoring and diabetes drug reintroduction were in place, at 12 months 74% of participants were off all diabetes medications. This study demonstrates that diabetes remission is obtainable with weight loss from lifestyle changes alone. However, weight loss must be maintained in order to maintain remission and this is arguably a greater challenge than weight loss itself. Several factors contribute to weight gain following weight loss. In addition to the difficulty of maintaining behavioral changes, physiological changes that occur with weight loss may also contribute to weight gain. Resting energy expenditure decreases with reduced body weight and several studies suggest this decrease in resting energy expenditure is greater than would be expected for decrease in body mass [32-34]. Simultaneously, hormonal changes that occur with weight loss, including increase in ghrelin and gastric inhibitory peptide, stimulate hunger and may promote energy storage [35,36]. Hormones that inhibit appetite and food intake, including pancreatic polypeptide, peptide YY, cholecystokinin, glucagon-like peptide and amylin, decrease with weight loss [37-42] and these changes were found to persist at a year after weight loss [34]. One study performed to quantify whether and to what extent appetite counters weight loss found that weight loss results in an increased caloric consumption of 100 kcal per day per kg of weight lost [42]. The changes in resting energy expenditure and hormonal changes that occur with weight loss contribute to the plateau effect which may discourage people with obesity from continuing their efforts to lose weight [35,43]. Several studies have suggested that hormonal changes that contribute to weight gain are altered with bariatric surgery [44]. Roux-en-Y gastric bypass, one of the most common and effective bariatric surgeries, has been associated with decreased ghrelin and increased cholecystokinin, peptide YY and postprandial GLP-1 levels [45-48]. Weight loss with bariatric surgery is superior to that obtained via lifestyle changes and/or medical therapy and these hormonal changes likely contribute to this success [49].

Conclusion

Successful treatment of diabetes must include concurrent treatment of obesity and overweight. Lifestyle and behavioral changes are necessary to combat a modern society in which addictive energy sources are abundant and readily obtainable with little energy expenditure. However, treatment of obesity is not as simple as calories in, calories out. Weight loss leads to physiological changes that perhaps for the first time in human history are maladaptive. Adjuvant medical and surgical therapies are recommended to overcome these barriers to weight loss and treat diabetes.

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