OPINION ARTICLE

Diabetes Management

The connection between insulin resistance, obesity and diabetes

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Description

According to epidemiological research, one of the major health implications of obesity is the development of T2D, and obesity has been proven to greatly raise the rates of diabetes mortality. This is further bolstered by the fact that lifestyle adjustments that result in weight loss have been demonstrated to significantly reduce the incidence of diabetes. However, our understanding of the physiological systems underlying obesity and T2D, as well as the ongoing discovery of genes implicated in both diseases, suggest that the link between them is complex, and that it may also be driven by specific patterns of fat deposition, rather than overall obesity.

The capacity of fat to cause IR is the fundamental physiological underpinning for the relationship between obesity and the beginning of T2D. Obesity has been linked to an increase in the quantity of triglycerides stored outside of adipose tissue, in places including skeletal muscle, the heart, the kidneys, and the liver. As a result, the level of circulating free fatty acids rises, and IR may occur in these tissues as a result of the increased lipid exposure. Excess insulin is first produced to compensate for the body's resistance to it, but this compensation is eventually insufficient, and diabetes can ensue. Many genetic research back up the epidemiological and physiological relationship between obesity and T2D.

Five of the loci reported to be strongly related with generalised obesity have also been demonstrated to enhance the risk of T2D and, like FTO, appear to be mediated through BMI. Different patterns of body fat distribution, independent of total adiposity, have a significant impact on IR and T2D risk. An extreme case of this can be found in patients with lipodystrophy, where the precise placement of fat around the liver and pancreas causes IR in the liver and muscle rather than overall fat accumulation. Furthermore, investigations have revealed that greater abdominal adiposity and IR are characteristics of first-degree relatives of T2D patients, implying an overlap in the genes that control fat.

Genetic investigations have revealed WHR-associated loci that control both T2D and IR, which supports this. WHR-increasing polymorphisms at the GRB14 locus were found to be strongly related with IR, with animal models indicating that GRB14 is a tissue-specific regulator of insulin action. Additionally, the signal linked with WHR around ADAMTS9 overlaps a known T2D susceptibility gene. The effect of ADAMTS9 on T2D risk may be mediated to some part by lower insulin sensitivity in peripheral tissues, emphasising the locus' primary effect on fat distribution.

Considering the inextricable relationship between these disorders, T2D is not a foregone

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conclusion. Undoubtedly, aberrant intra-abdominal fat storage is related with T2D, but this accumulation of central adiposity is not simply a result of being overweight or obese. In fact, regardless of total fat mass, patients with IR had much higher levels of abdominal fat. Additionally, people who are prone to gluteal fat accumulation have a lower incidence of T2D. As a result, genetic risk factors implicated in fat distribution may have significant implications for both obesity and T2D.

Obesity and type 2 diabetes both have unknown biological causes. Overlapping genetic variation associated with BMI and T2D is evidence of a genetic relationship between the two diseases, however many obesity-susceptibility mutations are unrelated to T2D risk. Indeed, there are genes that predispose people to T2D that are unrelated to obesity, and not all fat patients acquire T2D, making T2D development in obese people unpredictable.

Body fat distribution has been demonstrated to be more relevant in T2D risk than overall obesity, and many of the genetic variables determining these distribution patterns are independent of overall adiposity. Furthermore, while environmental factors are known to have a major effect on both obesity and T2D risk, the impact of the genetic relationship between these two features remains to be examined.