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K **:** Obesity; Diabetes; Type 2 diabetes

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e 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 ve times more e cient 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. e 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 uids, vitamins, and minerals had the longest known fast. He lost 60% of his body weight without experiencing any negative e ects. 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].

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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

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.

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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 e ects 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 brosis (increased rates of brogenesis and expression of genes involved in the formation of the extracellular matrix), in ammation (increased proin ammatory 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 in uence 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 signi cance. Adipose tissue, the liver, muscle, and pancreatic islets may also interact with one another to cause insulin resistance and hepatic steatosis. If there is su cient restoration of cell function, reducing body fat mass without surgical removal can ameliorate or normalize obesity-