Narrative Review of the Relationship between Obesity and Type 2 Diabetes

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ABSTRACT

The global coexistence of obesity and type 2 diabetes mellitus (T2DM), frequently termed "diabesity," represents one of the most pressing metabolic challenges of the 21st century. This narrative review synthesizes epidemiological, mechanistic, clinical, and public health evidence to elucidate the bidirectional and multifactorial relationship between obesity and T2DM. Obesity significantly increases the risk of T2DM through interconnected pathways involving insulin resistance, β-cell dysfunction, adipose-tissue endocrine dysregulation, chronic low-grade inflammation, and lipotoxicity driven by ectopic fat deposition. Additional mechanisms include alterations in gut microbiota composition, genetic and epigenetic variations, and life-course exposures beginning in early childhood. The review highlights distinct obesity phenotypes, particularly metabolically unhealthy obesity, visceral adiposity, and ectopic fat patterns, as more strongly predictive of diabetes than BMI alone. Conversely, metabolically healthy obesity appears to confer comparatively lower but still elevated risk. Clinically, intentional and sustained weight loss improves glycaemic outcomes and can induce diabetes remission, while pharmacologic and surgical interventions further support metabolic recovery. Public health evidence underscores the need for multisectoral strategies targeting food environments, early-life nutrition, and community-level lifestyle interventions. Despite extensive research, critical gaps persist regarding causality, genetic interactions, longitudinal trajectories, and the mechanistic contributions of microbiota and environmental exposures. Advancing precision-based prevention and treatment approaches requires integrated, multi-omics, and life-course studies to deepen understanding of the obesity, T2DM nexus and inform more effective interventions.
Keywords: Obesity, Type 2 diabetes mellitus (T2DM), Insulin resistance, Visceral and ectopic fat, and Metabolic dysregulation.

INTRODUCTION

Obesity is characterized by excessive adiposity, while type 2 diabetes mellitus (T2DM) arises from dysfunctional insulin action, and/or inadequate insulin secretion [1]. Both conditions occur increasingly often in young adults and children in parallel with rising obesity levels and contribute to multiple health complications [2]. Evidence supports a bidirectional link, with obesity increasing T2DM risk and T2DM being more prevalent in people with obesity. However, the association can emerge from several causal pathways, each supported by different lines of evidence. A conceptual framework groups four categories of mechanisms linking obesity to T2DM: insulin resistance and β-cell dysfunction; dysregulation of adipose tissue as an endocrine organ; chronic inflammation or immune modulation; and ectopic fat deposition [2]. These pathways mediate the effects of excess adiposity or obesity on risk, but are not alone sufficient to cause T2DM. Distinct obesity phenotypes, particularly metabolically unhealthy versus healthy, and differences in adipose tissue distribution modify diabetes risk [3].

Epidemiology of Obesity and Type 2 Diabetes

The global prevalence of obesity and type 2 diabetes, a combination termed "diabesity" by the World Health Organization, has attained epidemic proportions [6]. The obesity pandemic is largely responsible for a corresponding increase in type 2 diabetes. Type 2 diabetes remains one of the most serious and widespread complications of obesity, even as other chronic obesity-related comorbidities, such as cardiovascular disease, cancer, and osteoarthritis, also threaten health [12]. One-fifth of diabetic adolescents and one-third of adults with

type 2 diabetes today report a medical history of obesity, highlighting the power of the two conduit conditions to amplify disability burden either independently or in combination [13]. Obesity is responsible for approximately 44% of global diabetes cases and is increasingly regarded as a primary metabolic disease. Worryingly, the total number of diabetes cases linked to obesity is projected to double from around 150 million in 2005 to 300 million by 2025[9]. Approximately 41 million children under five were overweight or obese in 2016, and if current trends persist, by 2030, an estimated 60% of the world's population could be classified as overweight or obese [6]. Increasing body mass index and abdominal fat distribution together exert a linear effect on type 2 diabetes risk that is mediated by alterations in adipose tissue biology and precedes the onset of insulin resistance and beta-cell dysfunction [4]. Around 80% of the population suffering from type 2 diabetes has either central or abdominal obesity, and therefore warrants preventive interventions targeted directly at obesity. Reported obesity prevalence among individuals with type 1 diabetes further underscores the multidimensional nature of the relationship between metabolic disorders and the importance of obesity in prevention [5].

Pathophysiological Mechanisms Linking Obesity to Type 2 Diabetes

The increasing epidemic of obesity, a major risk factor for type 2 diabetes (T2D) and associated complications, continues to rise at an alarming pace [1]. Gaining a deeper understanding of the molecular mechanisms linking obesity and T2D is crucial to addressing this global health issue [7]. Increased body mass index and abdominal fat distribution linearly increase the risk of T2D due to changes in adipose tissue biology that connect obesity with insulin resistance and beta-cell dysfunction [3]. Abdominal obesity is an independent risk factor for hypertension and elevated fasting glucose, even without meeting BMI criteria for obesity [9]. The contribution of obesity to diabetes remains substantial, accounting for 55% of all cases, and the incidence is expected to double by 2025. Consequently, the term "diabesity" has emerged to reflect the need to address diabetes as an early consequence of obesity [5]. Non-esterified fatty acids in obese individuals play a crucial role in developing insulin resistance and beta-cell dysfunction [8]. Modifiable factors and the emphasis on preventing obesity imply that the underlying causative agents remain preventable and that timely intervention efforts will yield substantial long-term benefits. Despite advances, obesity and diabetes remain interconnected public health challenges worldwide [7].

Insulin Resistance and Beta-Cell Dysfunction

Obesity and type 2 diabetes are endemic worldwide, with consequent personal hardship and public health costs. Evidence has accrued since 2015 confirming a bidirectional relationship between the two disorders, under which each exerts mutual positive causation [10]. The current review considers whether the association is causal, and if so, how adiposity induces diabetes. The emphasis is on pathophysiological mechanisms, integrating epidemiological data on the obesity-diabetes nexus with insights into underlying biology [9]. The emphasis on causality reflects evidence that controlling obesity in high-risk individuals can reduce progression to diabetes, whereas glycaemic control alone, even with drug therapies, has little influence on adiposity or related metabolic disorders [8]. Such considerations motivate the specification of these mechanisms for effective preventive strategies to alleviate the severity of the diabetes pandemic. The material draws chiefly from recent reviews and commentaries in Nature, The Lancet, and Diabetes [6]. Obesity exerts its effect through increased insulin resistance and β-cell dysfunction [2]. Adiposity measures (e.g., body mass index, waist circumference) correlate positively with insulin-sensitivity estimates, while baseline measurements of plasma insulin show an inverse association with insulin secretion [3]. Connective-tissue growth factor (CTGF) activates transcription factors and signalling cascades that regulate fibrosis. Pancreatic fat, hepatocyte steatosis, and plasma free fatty acids increase with obesity. An increase in the quantity and saturation of circulating lipids, particularly free fatty acids, associates with reduced insulin sensitivity and elevated β-cell stress, with the former being the more pivotal change [3]. Such saturation produces a glucolipotoxic state that is specified with a model of partial β-cell failure, and underlies the progression of type 2 diabetes [1].

Adipose Tissue as an Active Endocrine Organ

The adipose tissue secretes numerous adipokines, lipokines, and other biologically active molecules into the circulation, thereby exerting important influences on glucose homeostasis, hepatic lipid flux, and many other metabolic and physiological processes [4]. Collectively, they modulate body mass, food intake, and energy expenditure, as well as influencing the development of obesity, type 2 diabetes, and other metabolic disorders. Adipose tissue also regulates the metabolism of orexigenic mediators such as ghrelin and orexins, as well as anorexigenic mediators such as peptide YY (PYY) and glucagon-like peptide-1 (GLP-1). Glucose homeostasis is also affected [7]. Adipokines derived from peripheral tissues such as liver, pancreas, gut, and skeletal muscle, and those produced during inflammation (inflammatory cytokines and lipokines) also exert parallel or opposing influences and modify the overall balance [6]. The product of the adipose tissue itself, non-esterified free fatty acids (NEFAs) secreted by adipocytes, exerts an important influence. An elevation in the plasma NEFA concentration that results from an increase in adiposity influences multiple organs, including the liver, skeletal muscle, pancreas, and β-cells. In the liver, the NEFA elevation increases glucose production, induces lipotoxicity,

and promotes insulin resistance [11]. In the skeletal muscle, it is known to exert the same lipotoxic effect while also inducing insulin resistance [12]. In the pancreas, an elevation in NEFA stimulates the secretion of insulin, while also inflicting damage on the cells and aggravating the stress already borne by them. The overall outcome tends to favour the worsening of hyperglycaemia due to the aggravation of insulin resistance in the liver and skeletal muscle and the existing stress on the β-cells. Consequently, the further elevation in NEFA concentration acts in a feed-forward manner to promote the aetiology of diabetes [13].

Inflammation and Immune Modulation

Expansion of adipose tissue during excessive caloric intake is associated with the recruitment of classical macrophages that promote pro-inflammatory cytokine signaling, such as tumor necrosis factor alpha (TNF-α) and interleukin-1 beta (IL-1β) [4]. Construction of the pro-inflammatory milieu induced by the excessive release of saturated fatty acids hinders the insulin signaling cascade, which ultimately leads to insulin resistance 5. A parallel macrophage polarization shift occurs in adipose tissue along with the development of insulin resistance, wherein B2 lymphocytes and alternatively activated macrophages are suppressed while T helper 1 and T helper 17 cells are activated [9]. While obesity is commonly thought to induce systemic inflammation, adipose tissue appears to remain the primary site generating the pro-inflammatory signaling. Leptin secreted by enlarged adipocytes exerts potentiating effects on pro-inflammatory signals, but downregulation of leptin receptor expression and consequent resistance is reported to occur despite obesity [6]. Although visceral adipose tissue has been emphasized, specialized immune responses in extra-adipose niches are being investigated [3].

Lipotoxicity and Ectopic Fat Deposition

Abdominal fat accumulation accompanies obesity; its presence, in particular a visceral pattern, is regarded as a marker for the development of insulin resistance (IR), type 2 diabetes mellitus (T2DM), and cardiovascular diseases (CVDs) [7]. More recently, fat deposition in non-adipose tissues such as the liver, muscle, and pancreas has emerged as another important factor in the etiology of IR, T2DM, and CVDs[6]. According to the assertion termed the ectopic fat hypothesis, excessive fat within the liver promotes hepatic IR, which drives elevated secretion of glucose, triglycerides, very-low-density lipoprotein, and other lipoproteins from the liver [8]. In addition, lipid accumulation within the pancreas contributes to metabolic dysregulation through multiple pathways, leading also to aggravation of T2DM [8]. The presence of ectopic fat in non-obese individuals has been postulated as a determinant factor in T2DM onset, especially in people of certain race–ethnic backgrounds and varying genetic predispositions. Attention has thereby shifted to the specific anatomical location within the body of fat accumulation, especially for SIP [7]. Detecting peripheral-fat obesity but abdominal-fat ectopy characterizes the phenotypic profile associated with enhanced susceptibility to co-morbidities of metabolic-associated fatty liver disease (MAFLD)[5].

Gut Microbiota and Metabolic Signaling

The gut microbiota plays a pivotal role in the pathophysiology of obesity and type 2 diabetes (T2D) through the modulation of energy harvest, metabolic signaling, and low-grade inflammation [7]. Recent epidemiological and mechanistic research has reported gut microbial alterations in individuals at high risk of these diseases. Patients with T2D exhibit reduced gut microbiota diversity and a lower abundance of major bacterial phyla compared to those without T2D [9]. Increased levels of bile acids, gut-derived hormones, and short-chain fatty acids, as well as hyperaldosteronism, are observed in individuals with T2D whose bacterial composition shifts towards a greater abundance of Bacteroidetes[4]. These changes influence the expression of genes involved in metabolism, inflammation, and the immune response. Metformin and other antidiabetic drugs impact the composition and activity of the gut microbiota, which, along with dietary interventions, is emerging as an important strategy for diabetes prevention and therapy. The microbiota has been recognized as a contributor to the pathophysiology of obesity and T2D[6]. Epidemiological studies indicate alterations in the composition of gut microbiota prior to weight gain, while a Western diet leads to increases in Bacilli, Fusobacteria, and Bacteroidetes, which diminish after the reintroduction of a low-fat diet[8]. Individuals who are overweight or obese exhibit a lower overall richness of gut microbiota compared to their lean counterparts, along with a reduction in the abundance and diversity of Faecalibacterium and Bacteroides species. The gut microbiota of children with obesity shows a higher prevalence of opportunistic pathogens associated with inflammation [9].

Genetic and Epigenetic Contributions

The World Health Organization (WHO) considers obesity a global epidemic that can have serious adverse effects on human health [2]. A body mass index (BMI) greater than 30 kg/m² is the most commonly used definition [10]. The prevalence of obesity has increased worldwide among all ages, especially in low- and middle-income countries. Obesity is associated with a number of health complications, among which type 2 diabetes is one of the most important [6]. Type 2 diabetes occurs when the body becomes resistant to insulin or when the pancreas fails to produce enough insulin [11]. It can lead to a range of health problems, including damage to the eyes, kidneys, nerves, and heart. According to the latest WHO data, the prevalence of diabetes worldwide among people aged 18

years and older was as high as 8.8% in 2014. It is projected that by the year 2040, approximately 642 million individuals will have diabetes [9]. The total health expenditure on diabetes has also been rising substantially, from US$2.30 trillion in 2010 and estimated to increase further to US$4.0 trillion by 2030. Low-income countries are expected to bear 43% of the global expenditure on diabetes [5].

Life Course and Longitudinal Risk

Obesity during early life, at 2 to 5 years, during childhood (6 to 12 years), or in adolescence (13 to 19 years) is associated with type 2 diabetes in adulthood [7]. For instance, participants in the Bogalusa Heart Study with BMI≥95th percentile at 5 to 6 years had a 6-fold higher diabetes risk at 30 to 40 years than peers who were never overweight [12]. A Singapore study showed a 20-fold increase among individuals in the highest 25% of international BMI percentiles for ages 5, 12, and 18 years [5]. Child obesity also raises the risk of diabetes and hyperglycemia by 1.7- to 3.4-fold [11]. Exposures early in life may exert greater long-term effects than those later on (and persistent weight status is a correlate); therefore, duration of exposure is as informative as duration alone. A trajectory-based risk diagram illustrates the interplay between timing and cumulative exposure [10]. Obesity during early life, at 2 to 5 years, during childhood (6 to 12 years), or in adolescence (13 to 19 years) is associated with type 2 diabetes in adulthood [6]. In the Bogalusa Heart Study, participants with BMI≥95th percentile at 5 to 6 years experienced a roughly 6-fold increased diabetes risk by age 30 to 40 compared to peers who were never overweight[13]. Singaporean individuals in the highest 25% of international BMI percentiles at ages 5, 12, and 18 faced a 20-fold increase [8]. Childhood obesity elevated the odds of diabetes and hyperglycemia by 1.7- to 3.4-fold. Early life exposures may exert greater long-term effects than those occurring later (and persistent weight status is a correlate), so duration of exposure is as informative as timing [14]. A trajectory-based risk diagram illustrates the interplay between timing and cumulative exposure [7].

Influence of Obesity Phenotypes on Diabetes Risk

Obesity is influential in the development of type 2 diabetes but does not always lead to this outcome during follow-up [7]. A concept of metabolically healthy obesity describes overweight and obese individuals without hypertension, dyslipidemia, or elevated glucose levels [10]. These individuals appear to be at lower risk for diabetes and cardiometabolic complications than those in an obese but metabolically unhealthy state. Additionally, certain obesity phenotypes are consistently related to diabetes risk [11]. Metabolically healthy obesity has been defined using a variety of criteria. Individuals may have an insulin resistance measure within the normal range, not exceed a specific HOMA-IR or HOMA-BC threshold, have a relatively high disposition index, and/or be free from one or more of the metabolic syndrome components [13]. Although prevention and treatment guidelines still consider the two states equivalent, risk estimates for diabetes are significantly different. Very few studies indicate an increased risk of diabetes [12]. Even among those with metabolically healthy obesity, however, health outcomes are generally worse than in individuals of normal weight [10]. Android obesity, indicated by a high waist circumference or waist-to-hip ratio, has a greater impact on diabetes than other fat distributions. Conversely, gynoid obesity is not associated with increased risk. Other obesity phenotypes also affect the risk of diabetes incidence or remission [9]. Metabolically unhealthy obesity, visceral fat accumulation, and ectopic fat patterns appear to predispose to diabetes, while metabolically healthy overweight, low ectopic fat, and normal-range HOMA-IR protect against it [8].

Clinical Implications for Prevention and Treatment

A growing body of evidence highlights the importance of weight status in treatment and outcomes for T2D [12]. Individuals with high body mass index (BMI) and excess obesity at diagnosis and throughout follow-up experience increased risk of developing diabetes-related complications and premature mortality [12]. Furthermore, weight loss has often been shown to induce remission of T2D. A classification reflecting phenotypic variability in BMI and diabetes status may assist risk stratification and guide selection of lifestyle, pharmacologic, or surgical intervention strategies [13]. During the past two decades, research has uncovered important biological pathways elucidating the relationship between obesity and T2D[2]. The nature of the relationship between the two is complex, and a deeper understanding of the biology linking these modifiable risk factors and disease progression will facilitate the development of novel preventive and therapeutic interventions [1].

Public Health Perspectives and Policy Considerations

The global pandemic of overweight and obesity has accelerated short- and long-term trends in type 2 diabetes (T2D) incidence [11]. Likewise, diabetes continues to rise within countries and regions experiencing rapid increases in the prevalence of obesity, supporting long-standing assumptions of a bidirectional relationship between obesity and T2D. However, compelling although imperfect epidemiologic evidence indicates that the relationship is not exclusively or even primarily one of mutual promotion [13]. Though necessary to establish risk and burden, the location of diabetes within this framework deserves further delineation than that afforded by either initiating event or an assumption of equivalency in causal potency [12]. The balance between event initiation and promotion appears to shift over the life course, with the focus on diabetes risk attributable to obesity

thereafter diminishing as individuals age [10]. An overwhelming majority of adults with prediabetes or undiagnosed diabetes remain obese, as do more than one-third of those receiving a diagnosis of T2D. Recovery from diabetes can occur following substantial and sustained weight loss, despite persistence of significant prediabetes, as it can following metabolic surgery, indicating that, particularly within the middle-aged and older populations, the modulation is likely to be best characterized as secondary promotion of more gradual type 2 metabolic decompensation and hyperglycemia rather than initiation of a strictly self-propagating disease process [11]. This imbalance in temporality and progression applies analogous concepts of primary and secondary prevention in further elucidating the population-level dynamics of obesity and diabetes [13]. During the past several decades, the emergence of new products and policies designed to stimulate changes in food environments and support healthier food choices has ignited intense interest in the role of public policy in addressing health-related aspects of diet, including obesity and diabetes [11]. Alongside population [14] and policy-based changes, affected dietary intake and resultant individual health status comprise critical links extending this interplay to prevention approaches featuring reduced emphasis on food availability. Scholarly examinations of empirical investigations constitute a largely unexplored yet vital component of the comprehensive and timely understanding needed to stimulate policy action against the rising tide of national and global diabetes prevalence [11].

Gaps in Knowledge and Future Research Directions

Several gaps and methodological limitations constrain the available evidence. The majority of studies are cross-sectional rather than longitudinal, leaving unanswered questions about the timing and direction of the relationship [12]. Mechanistic studies are needed to elucidate the causal links [12]. Although obesity and type 2 diabetes share several genetic risk factors, few studies have explored the influence of genetic and polygenic risk on the relationship. The association between obesity and type 2 diabetes, as well as their interplay, may involve epigenetic modifications over the life course [14-16]. Furthermore, natural experiments examining the causal impact of weight gain and loss, particularly obesity phenotypes, and diabetes duration on the incidence of diabetes are scarce. Omics-based human-behavioral-microbiome-lifestyle interaction studies hold great promise for advancing the understanding of type 2 diabetes etiology in general and the role of obesity in particular[13]. Polygenic and epigenetic traits, metabolomic signatures, metabolic imaging, gut-microbiome signatures, and other environmental factors enabling risk differentiation in responses to specific diets or lifestyle patterns can facilitate development of targeted preventive and therapeutic interventions [17-20]. Of special interest are longitudinal mechanistic studies with detailed life-course and over-lifetime trajectory data, especially on early-life and adulthood predictors of metabolic dysregulation. Integrating physiological and biological data across the whole genome, transcriptome, proteome, metabolome, microbiome, and exposome into models that improve predictive accuracy and precision for metabolism and obesity-related diseases remains a priority [12].

CONCLUSION

Obesity and type 2 diabetes are deeply interconnected metabolic disorders whose global rise poses severe health, social, and economic challenges. The evidence reviewed shows that obesity is not merely a contributing factor but a fundamental driver of T2DM through complex physiological pathways that include insulin resistance, β-cell dysfunction, adipokine imbalance, chronic inflammation, and ectopic fat deposition. These biological mechanisms are further shaped by gut microbiome alterations, genetic predisposition, and life-course exposures that begin in early childhood and persist into adulthood. Epidemiological patterns reinforce this link, as rising obesity prevalence mirrors increasing rates of T2DM across regions and age groups. Importantly, not all forms of obesity confer equal risk. Visceral adiposity, metabolically unhealthy obesity, and ectopic fat patterns significantly elevate susceptibility to diabetes, while metabolically healthy obesity, though comparatively safer, still carries increased long-term risk. Clinically, weight loss remains central to improving insulin sensitivity, glycaemic control, and even achieving diabetes remission, whether through lifestyle modification, pharmacotherapy, or metabolic surgery. These findings affirm the necessity of early, targeted interventions that address obesity as a primary prevention strategy against diabetes. At the population level, the obesity–diabetes relationship underscores the urgent need for policies that reshape food environments, reduce socioeconomic barriers to healthy living, and encourage equitable access to preventive and therapeutic care. Despite decades of research, major gaps persist concerning causality, gene-environment interactions, microbiome contributions, and the long-term metabolic consequences of early-life obesity. Future research must therefore adopt integrative, longitudinal, and multi-omics approaches capable of capturing the full complexity of this relationship. Understanding and addressing the intertwined epidemics of obesity and T2DM requires coordinated clinical, scientific, and policy efforts. Strengthening these approaches will be essential for reducing global diabetes burden and improving metabolic health across the life course.

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