



Emerging perspectives on type 5 diabetes: Pathophysiology and nutrition-based management strategies

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Abstract

Type 5 diabetes mellitus (T5DM) is an emerging and distinct form of diabetes primarily associated with chronic undernutrition and predominantly observed in low- and middle-income countries. Unlike type 1 and type 2 diabetes, T5DM is characterized by impaired insulin secretion due to reduced pancreatic β -cell mass, preserved insulin sensitivity, and absence of autoimmune destruction. This review explores the current understanding of T5DM, focusing on its epidemiology, risk factors, and underlying pathophysiological mechanisms, including impaired pancreatic development, micronutrient deficiencies, and developmental programming. The paper also highlights key clinical features and the diagnostic challenges that often lead to misclassification and inappropriate management. A major emphasis is placed on nutrition-based management strategies, including macronutrient rehabilitation, micronutrient supplementation, calorie optimization, and the role of functional foods, along with the integration of medical nutrition therapy and pharmacological support. Furthermore, the importance of public health nutrition interventions and emerging research directions, such as epigenetics and personalized nutrition, are discussed. Understanding T5DM is critical for developing targeted, cost-effective strategies to reduce its growing burden and improve metabolic health outcomes in vulnerable populations.

Keywords: Type 5 diabetes mellitus, malnutrition-related diabetes, undernutrition, B-Cell dysfunction, insulin secretion defect, micronutrient deficiency, medical nutrition therapy, functional foods, public health nutrition, LMICs

Introduction

Type 5 diabetes, previously termed malnutrition-related diabetes mellitus (MRDM), has emerged as a unique metabolic disorder primarily affecting undernourished populations (Chen and Lu, 2026) [8]. It predominantly occurs in South Asia and Sub-Saharan Africa and affects approximately 20–25 million individuals worldwide (Chatterjee and Tewari, 2025; Agyemang *et al.*, 2026) [1, 7].

The formal recognition of T5DM fills a critical gap in diabetes classification and highlights the intersection between nutrition, poverty, and metabolic disease (Hossain *et al.*, 2026) [12].

Epidemiology and Risk Factors

Type 5 diabetes mellitus (T5DM) is predominantly observed among adolescents and young adults, particularly in low- and middle-income regions where undernutrition remains widespread (Prajitno and Sutanto, 2025) [23]. It is strongly associated with individuals having a low body mass index (BMI < 18.5 kg/m²) and those living in conditions of chronic food insecurity (Lotus *et al.*, 2025) [16]. The epidemiological pattern of T5DM reflects a close link between poverty, inadequate dietary intake, and metabolic dysfunction. Major risk factors include early-life protein-energy malnutrition, which impairs pancreatic development and reduces β -cell mass, as well as poor maternal nutrition that adversely affects fetal growth and long-term metabolic programming (Taneera *et al.*, 2025) [29]. Additionally, recurrent infections during childhood exacerbate nutrient depletion and metabolic stress, further increasing

susceptibility. Micronutrient deficiencies—particularly of zinc, vitamin A, and iron—also play a critical role by disrupting insulin synthesis, secretion, and overall glucose homeostasis (Uğuz *et al.*, 2026) [1]. Overall, chronic undernutrition during critical periods of growth and development is considered the primary etiological driver of T5DM, highlighting the importance of early nutritional interventions and public health strategies in preventing this emerging form of diabetes.

Pathophysiology of Type 5 Diabetes

1. Impaired Pancreatic Development

A central mechanism underlying Type 5 diabetes mellitus (T5DM) is impaired pancreatic development resulting from prolonged nutritional deprivation during critical periods of fetal life and early childhood (Reyed, 2026) [27]. Chronic undernutrition, particularly protein-energy malnutrition, disrupts normal organogenesis, leading to reduced pancreatic size and diminished β -cell mass. This developmental insufficiency compromises the pancreas's ability to produce and secrete adequate insulin throughout life. Inadequate availability of essential nutrients such as amino acids, fatty acids, and micronutrients during these sensitive stages interferes with β -cell proliferation, differentiation, and functional maturation. Consequently, individuals affected by early-life malnutrition exhibit a permanently reduced insulin secretory capacity, making them highly susceptible to glucose intolerance and diabetes in later years. This concept aligns with the developmental origins of health and disease (DOHaD) hypothesis, which

emphasizes that early nutritional insults can have long-lasting effects on metabolic health (Trimble *et al.*, 2025) [32].

2. Insulin Secretory Defect

A defining feature of Type 5 diabetes mellitus (T5DM) is a profound impairment in insulin secretion, primarily due to reduced functional β -cell mass (Pontes *et al.*, 2025) [22]. Individuals with T5DM exhibit markedly decreased insulin output—often up to approximately 70% lower than normal—reflecting the long-term impact of early-life undernutrition on pancreatic capacity. One of the earliest detectable abnormalities is a blunted first-phase insulin response, which is crucial for controlling postprandial glucose excursions; its impairment leads to rapid rises in blood glucose following meals. Additionally, low circulating C-peptide levels further confirm diminished endogenous insulin production, distinguishing T5DM from conditions characterized by insulin resistance. Unlike type 2 diabetes mellitus, where insulin levels may be normal or elevated due to compensatory hyperinsulinemia, T5DM is primarily a disorder of insulin deficiency without significant peripheral resistance, highlighting its unique pathophysiological profile (Trimble *et al.*, 2025) [32].

3. Preserved Insulin Sensitivity

In contrast to type 2 diabetes mellitus, Type 5 diabetes (T5DM) is characterized by relatively preserved insulin sensitivity in peripheral tissues such as skeletal muscle and adipose tissue (Goodpaster and Wolf, 2004) [9]. Individuals with T5DM generally do not exhibit the hallmark features of insulin resistance, and their glucose uptake mechanisms remain largely intact. Additionally, hepatic fat accumulation is typically low, reflecting the absence of ectopic lipid deposition that commonly contributes to insulin resistance in type 2 diabetes. As a result, clinical features of metabolic syndrome—such as central obesity, dyslipidemia, and hypertension—are usually absent in T5DM patients (Tewari *et al.*, 2025) [30]. This distinct metabolic profile underscores that hyperglycemia in T5DM primarily arises from inadequate insulin secretion rather than impaired insulin action, further differentiating it from classical insulin-resistant diabetes phenotypes (James *et al.*, 2021) [14].

4. Non-Autoimmune Mechanism

Type 5 diabetes mellitus (T5DM) is characterized by a non-autoimmune etiology, distinguishing it clearly from type 1 diabetes mellitus (Marchetti *et al.*, 2023) [17]. In T5DM, there is no evidence of immune-mediated β -cell destruction; instead, the loss of insulin-secreting capacity is primarily attributed to developmental and nutritional factors. Patients typically test negative for pancreatic autoantibodies such as glutamic acid decarboxylase (GAD) and insulinoma-associated antigen-2 (IA-2), which are hallmark indicators of autoimmune β -cell damage in type 1 diabetes. The absence of these immunological markers confirms that β -cell dysfunction in T5DM arises from structural and functional impairment rather than immune attack. This non-autoimmune mechanism highlights the role of chronic undernutrition and developmental deficits in shaping pancreatic insufficiency, reinforcing T5DM as a distinct clinical and pathophysiological entity (Roep *et al.*, 2021) [28].

5. Role of Micronutrient Deficiency

Micronutrient deficiencies play a critical role in the pathophysiology of Type 5 diabetes mellitus (T5DM), particularly in populations affected by chronic

undernutrition (Prajitno and Sutanto, 2025) [23]. Deficiency of zinc, an essential cofactor for insulin synthesis, storage, and secretion, directly impairs β -cell function and insulin availability (Moore *et al.*, 2015) [20]. Similarly, inadequate levels of vitamin A adversely affect β -cell integrity and gene expression involved in insulin production. A lack of essential amino acids further compromises protein synthesis, including enzymes and hormones necessary for normal pancreatic function. Together, these nutritional deficits disrupt both the structural and functional capacity of the pancreas, leading to impaired insulin secretion and altered glucose metabolism. This highlights the importance of micronutrient adequacy in maintaining endocrine pancreatic health and underscores the nutritional basis of T5DM (Trasino *et al.*, 2015) [31].

6. Developmental Programming

Emerging evidence strongly supports the concept of developmental programming, often described as the “nutritional imprinting” hypothesis, in the pathogenesis of Type 5 diabetes mellitus (T5DM) (Langley-Evans, 2006) [15]. According to this framework, nutritional deprivation during critical periods of fetal development and early childhood induces permanent structural and functional changes in metabolic organs, particularly the pancreas. Early-life malnutrition leads to long-term metabolic dysfunction by limiting β -cell proliferation and reducing the overall β -cell reserve. As a result, individuals may maintain near-normal glucose regulation during early life but become increasingly vulnerable to glucose intolerance and diabetes in adolescence or adulthood when metabolic demands rise. This reduced β -cell reserve, combined with lifelong nutritional challenges, ultimately contributes to the onset of T5DM, highlighting the lasting impact of early nutritional environments on metabolic health (Holemans *et al.*, 2003) [11].

Clinical Features

Type 5 diabetes mellitus (T5DM) presents with a distinct set of clinical features that often overlap with other forms of diabetes, yet retain important differences (Redondo *et al.*, 2020) [26]. A hallmark characteristic is a low body mass index (BMI < 19 kg/m²), reflecting the underlying state of chronic undernutrition (Cabral *et al.*, 2024) [5]. Patients typically exhibit persistent hyperglycemia but, unlike in classic type 1 diabetes, ketosis is usually absent. Common symptoms include fatigue, unintended weight loss, and polyuria, which arise due to inadequate insulin availability and resulting metabolic imbalance. The condition most frequently manifests during adolescence or early adulthood, a period when metabolic demands increase. Clinically, individuals with T5DM may resemble those with type 1 diabetes; however, they lack autoimmune markers such as pancreatic autoantibodies, which is a key distinguishing feature. This combination of undernutrition, insulin deficiency without ketosis, and non-autoimmune status defines the clinical profile of T5DM (Chen and Lu, 2026) [27].

Diagnostic Challenges

The diagnosis of Type 5 diabetes mellitus (T5DM) remains challenging due to the absence of standardized diagnostic criteria and clear clinical guidelines. As a result, patients are frequently misclassified as having type 1 or type 2 diabetes,

given the overlap in clinical presentation such as hyperglycemia and age of onset. However, this misdiagnosis can lead to inappropriate therapeutic approaches—for example, unnecessary lifelong insulin therapy as in type 1 diabetes or ineffective insulin-sensitizing treatment as in type 2 diabetes. The challenge is further compounded in low- and middle-income countries (LMICs), where access to advanced diagnostic tools, such as autoantibody testing and C-peptide measurement, is often limited. Consequently, misclassification not only delays accurate diagnosis but also contributes to suboptimal glycemic control, increased risk of complications, and overall poorer health outcomes (Nurkolis *et al.*, 2025)^[21].

Nutrition-Based Management Strategies

1. Macronutrient Rehabilitation

A fundamental component of managing Type 5 diabetes mellitus (T5DM) is the correction of chronic undernutrition through targeted macronutrient rehabilitation. Given that the condition is rooted in long-standing nutritional deficiencies, restoring adequate dietary intake is essential for improving metabolic function. A high-protein diet plays a crucial role in rebuilding lean body mass and supporting the regeneration and function of pancreatic β -cells. At the same time, carbohydrate intake should be balanced rather than excessively restricted, ensuring sufficient energy supply while maintaining glycemic control. Incorporation of healthy fats further enhances dietary energy density, which is particularly important for underweight individuals requiring gradual weight gain. Together, these dietary modifications help stabilize blood glucose levels, improve insulin secretion, and promote overall metabolic recovery, making nutritional rehabilitation a cornerstone in the management of T5DM.

2. Micronutrient Supplementation

Targeted micronutrient supplementation is a critical component in the management of Type 5 diabetes mellitus (T5DM), as underlying deficiencies directly impair pancreatic function and glucose metabolism. Zinc plays a vital role in insulin synthesis, storage, and secretion, thereby improving β -cell efficiency. Vitamin A contributes to the maintenance of β -cell integrity and regulates gene expression involved in insulin production (Wei *et al.*, 2026)^[34]. Additionally, iron and B-complex vitamins are essential for optimal metabolic processes, including energy production, oxygen transport, and enzymatic reactions involved in carbohydrate metabolism. Correcting these deficiencies helps restore physiological balance, enhances insulin secretion, and supports overall metabolic health. Therefore, adequate micronutrient supplementation is essential for achieving and maintaining long-term glycemic control in individuals with T5DM (Chen and Lu, 2026)^[27].

3. Calorie Optimization

Calorie optimization is a crucial aspect of managing Type 5 diabetes mellitus (T5DM), particularly because affected individuals are often chronically undernourished. A gradual increase in caloric intake is essential to safely restore energy balance and promote healthy weight gain without overwhelming compromised metabolic systems. Rapid escalation of calories should be avoided to prevent refeeding syndrome, a potentially life-threatening condition characterized by electrolyte imbalances and metabolic

disturbances. Emphasis should be placed on nutrient-dense foods that provide not only adequate energy but also essential vitamins, minerals, and high-quality macronutrients. This careful and structured approach to caloric rehabilitation supports improved metabolic stability, enhances insulin function, and contributes to overall recovery in T5DM patients (Hossain *et al.*, 2026)^[12].

4. Functional and Therapeutic Foods

Emerging evidence highlights the important role of functional and therapeutic foods in the management of Type 5 diabetes mellitus (T5DM) (Chen and Lu, 2026)^[12], particularly in improving metabolic regulation and overall nutritional status. Probiotic-rich foods such as fermented dairy products and traditional fermented foods help modulate gut microbiota, which in turn enhances nutrient absorption, reduces inflammation, and supports glucose homeostasis. Omega-3 fatty acids, commonly found in fatty fish, flaxseeds, and walnuts, exert anti-inflammatory effects that may improve pancreatic function and metabolic stability. Additionally, the inclusion of whole grains and legumes provides complex carbohydrates with a low glycemic index, promoting better glycemic control and sustained energy release. Together, these dietary components align with broader principles of nutritional therapeutics and offer a supportive, food-based approach to managing metabolic disorders like T5DM.

5. Medical Nutrition Therapy (MNT) Integration

Effective management of Type 5 diabetes mellitus (T5DM) requires a synergistic integration of medical nutrition therapy (MNT) with appropriate pharmacological interventions. While nutritional rehabilitation remains the foundation of treatment, some patients may require low-dose insulin therapy, particularly in cases of severe hyperglycemia or during the initial stabilization phase (Anton *et al.*, 2026)^[3]. In selected individuals, oral hypoglycemic agents such as sulfonylureas may be used to stimulate residual β -cell function and enhance endogenous insulin secretion. Importantly, unlike type 1 diabetes mellitus, insulin dependency in T5DM is not absolute and may decrease as nutritional status improves and pancreatic function partially recovers. Therefore, treatment should be individualized, with continuous monitoring and adjustment of both dietary and pharmacological strategies to achieve optimal glycemic control and metabolic recovery (Chandru *et al.*, 2026)^[6].

6. Public Health Nutrition Interventions

Addressing Type 5 diabetes mellitus (T5DM) effectively requires a strong public health approach that targets its underlying causes rather than merely managing clinical symptoms. Preventive strategies must begin with improving maternal nutrition, as adequate nutrient intake during pregnancy is essential for proper fetal growth and pancreatic development. Equally important are appropriate infant and young child feeding practices, including timely initiation of breastfeeding and nutritionally adequate complementary feeding, which support optimal growth and metabolic programming. Broader food security policies play a critical role in ensuring consistent access to affordable, nutrient-dense foods for vulnerable populations. Additionally, infection control measures—such as improved sanitation, vaccination, and access to healthcare—help reduce the

burden of recurrent illnesses that exacerbate malnutrition. Collectively, these interventions address the root

determinants of T5DM and are essential for its long-term prevention and control at the population level.

Table: 1 Summary of Selected Published Studies on Type 5 Diabetes / Malnutrition-Related Diabetes

Author(s) & Year	Study Population / Region	Study Design	Key Findings	Relevance to T5DM
Bajaj <i>et al.</i> , 1989 ^[4]	India (undernourished young adults)	Clinical observational study	Identified low BMI, impaired insulin secretion, absence of ketosis	Early characterization of malnutrition-related diabetes
WHO Expert Committee, 1985	Global	Classification report	Recognized MRDM as a distinct category of diabetes	Foundation for current Type 5 diabetes concept
Mohan <i>et al.</i> , 1990s ^[19]	India	Epidemiological study	Demonstrated reduced β -cell function with normal insulin sensitivity	Differentiated MRDM from type 2 diabetes
Yajnik, 2004 ^[36]	India (fetal origin studies)	Cohort study	Linked low birth weight to future glucose intolerance	Supports developmental programming hypothesis
Hales & Barker, 1992 ^[10]	UK	Hypothesis paper	Proposed “Thrifty phenotype hypothesis”	Explains early malnutrition → adult diabetes link
Ramachandran <i>et al.</i> , 2000 ^[25]	South Asia	Population study	Highlighted role of poverty and undernutrition in diabetes	Established socioeconomic determinants
Anjana <i>et al.</i> , 2011 ^[2]	India	Cross-sectional study	Showed heterogeneity of diabetes phenotypes in lean individuals	Supports existence of non-obese diabetes types
IDF Working Group, 2025	Global	Consensus report	Official recognition of Type 5 diabetes	Formal classification and renewed research focus
Misra & Khurana, 2008 ^[18]	India	Review	Highlighted role of micronutrient deficiency in metabolic disorders	Supports nutritional basis of T5DM
Prentice, 2001 ^[24]	Africa	Nutritional epidemiology	Chronic undernutrition linked to metabolic dysfunction	Reinforces global relevance of T5DM

Conclusion

Type 5 diabetes is a newly recognized but long-overlooked form of diabetes rooted in chronic undernutrition. Its distinct pathophysiology—characterized by impaired insulin secretion, preserved insulin sensitivity, and absence of autoimmunity—necessitates tailored diagnostic and therapeutic approaches. Nutrition-based management strategies, including macronutrient rehabilitation and micronutrient supplementation, form the cornerstone of treatment. Addressing broader determinants such as poverty, food insecurity, and maternal health is essential for prevention. Future research should focus on refining diagnostic criteria, understanding molecular mechanisms, and developing context-specific interventions to reduce the global burden of this emerging disease.

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