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REVIEW ARTICLE

The growing concern of Type 5 diabetes mellitus: A narrative review

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Abstract

Background: Type 5 diabetes mellitus (T5DM) is a developing form of diabetes linked to early childhood malnutrition, resulting from pancreatic underdevelopment due to nutritional deprivation during critical growth periods. This neglected health issue poses a challenge to achieving sustainable development goal 2: Zero Hunger, as food insecurity drives childhood stunting and poor maternal nutrition, contributing to T5DM, and ultimately jeopardising the targets of sustainable development goals 3: Good health and well-being. This narrative review explores T5DM in the context of global trends to guide nutritional policy and chronic disease prevention in low- and middle-income countries, where poverty and metabolic dysfunction intersect.

Methods: A review of 47 articles was conducted, including original studies, reviews, case studies, and policy reports. A structured search of PubMed, Scopus, Google Scholar and grey literature identified relevant studies published between 2020 and 2026 that focused on T5DM, malnutrition-related diabetes mellitus, and early-life malnutrition.

Results: Evidence indicates that T5DM is driven by impaired pancreatic β–cell development resulting from fetal and early childhood nutritional deprivation. Mechanisms include epigenetic modifications, reduced β–cell mass, and altered incretin responses. Epidemiological patterns show that T5DM disproportionately affects populations exposed to chronic undernutrition, with low- and middle-income countries representing a high-risk setting due to persistent childhood stunting, wasting, and maternal undernutrition. These nutritional deficits create a biological environment conducive to long-term insulin deficiency and metabolic dysfunction.

Conclusion: T5DM represents an emerging metabolic consequence of early-life nutritional deprivation. Addressing this challenge requires multi-sectoral strategies that strengthen maternal and child nutrition, improve food security, and integrate malnutrition-related diabetes awareness into national public health policies, supporting sustainable growth toward the 2030 development agenda.

Key messages

This review of 47 articles emphasises that malnutrition is not only a humanitarian crisis but also a driver of long-term non-communicable disease. Type 5 diabetes mellitus, primarily caused by early-life malnutrition, is exacerbated by persistent food insecurity in low- and middle-Income countries, highlighting the need for multi-sectoral strategies to improve nutrition, food security, and diabetes awareness.

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Introduction

Diabetes mellitus is a chronic disease characterised by the body's inability to produce enough insulin or to effectively utilise the insulin produced, leading to hyperglycaemia [1]. Type 1, type 2, and gestational diabetes mellitus (developing during pregnancy) are the common forms of diabetes; however, there is a broader spectrum of diabetes, including several lesser -known or emerging forms. Recently, the International Diabetes Federation (IDF) made a historic pronouncement that reshaped the medical community's understanding of diabetes. For the first time, the medical community officially recognized Type 5 diabetes mellitus (T5DM) as a distinct variant of diabetes. This monumental decision brought to light a health crisis that had long been hidden and neglected, who had suffered owing to misdiagnoses and a lack of targeted treatment [2]. Currently identified as a unique condition tied to profound early -life malnutrition, T5DM demands global attention because it affects approximately 20 - 25 million people globally, mostly in developing countries around Asia and Africa. The IDF's recognition of the disease represents a significant step forward in understanding the diverse spectrum of diabetes, particularly among lean teenagers and young adults who often face chronic undernutrition [2, 3].

Unlike types 1 and 2 diabetes mellitus, T5DM is not linked to autoimmune responses or insulin resistance caused by obesity. It emerges from severe protein and nutrient deficiencies during critical periods of childhood development. These early nutritional insults hinder the growth and function of the pancreas and related metabolic systems, increasing the susceptibility of individuals to chronic high blood glucose later in adolescence or young adulthood [4]. Type 5 particularly shows symptoms that mirror those of other types of diabetes, such as increased thirst, frequent urination, exhaustion, and unexplained weight loss. However, affected individuals are strikingly lean and often live in low-income settings, making standard diabetes treatments less effective [5].

Globally, the distribution of T5DM mirrors patterns of chronic undernutrition, with the highest burden observed in low- and middle-Income countries (LMICs) across Asia and sub-Saharan Africa [2, 6, 7]. Cases of its occurrence have been documented in specific countries such as Bangladesh, India, Jamaica, Sri Lanka, Nigeria, Uganda, Ethiopia, Rwanda and Korea [8]. In these settings, widespread food insecurity, maternal undernutrition, and childhood stunting create the biological and environmental conditions necessary for the development of this disease [7, 9]. Despite this, T5DM remains under-recognised, frequently misdiagnosed, and largely absent from standard diabetes classification frameworks until recently [4]. Therefore, this paper aims to explores T5DM as a nutrition-driven metabolic disorder, examining its pathophysiology, epidemiological patterns, and clinical implications.

Methods

Study design

This study was conducted as a narrative review aimed at synthesising the existing evidence on the association between early-life malnutrition and the development of T5DM. The review incorporates the biochemical, epidemiological, and policy approaches to comprehend the role of maternal and childhood undernutrition in the development of pancreatic impairments and risk of diabetes, especially in LMICs.

Sources of data and search strategy

A thorough literature review was conducted in PubMed (n=29), Scopus (n=18), and Google Scholar (n=97) to identify relevant publications published between January 2020 and April 2026. The search strategy combined keywords and controlled vocabulary related to T5DM, malnutrition-related diabetes mellitus, early-life malnutrition, maternal malnutrition, childhood stunting, pancreatic β–cell development, food insecurity, and LMICs. The search was refined by using the Boolean operators (AND/OR). The indexing of each database was modified to the search strings. Other grey literature was sourced through international organisations, such as the World Health Organisation, the International Diabetes Federation, and the United Nations Children's Fund. The IDF diabetes atlas and the joint child malnutrition estimates were the key reports reviewed to obtain global epidemiological statistics on diabetes and child malnutrition. Manual screening of the reference lists of relevant studies was also conducted to identify additional relevant publications.

Inclusion criteria and exclusion criteria

Original research articles, systematic reviews, narrative reviews, case reports, and relevant policy or guideline documents published in the search period on T5DM, early-life and maternal undernutrition, and pancreatic development were included in the review. Conference abstracts that lacked enough methodological information, opinion articles that were not scientific or clinically relevant, and non-English language publications were excluded.

Selection and screening procedure

The initial search revealed 144 records. Following the elimination of 27 duplicates, 117 unique records were then filtered by the title and abstract. Seventy-seven (77) records were eliminated because of irrelevance to diabetes associated with malnutrition, early-life nutritional setting, or inaccessibility to the complete text. Thereafter, 40 full-text articles were assessed for eligibility and met the inclusion criteria. Additionally, 7 grey literatures (policy documents and national nutritional guidelines), were incorporated. These papers provided important background information on diabetes, malnutrition patterns, and national nutrition policies relevant to T5DM risk factors in low-

and middle-income nations. In the final analysis, a total of 47 articles, including 24 reviews, 11 original research articles, 7 grey literature articles, and 5 clinical case reports, were included in the study.

Quality measurement and risk of bias

Since the study was a narrative review, a formal quantitative risk-of-bias assessment was not conducted. However, the methodological rigour, relevance, clarity of objectives, and strength of evidence were critically appraised qualitatively during data interpretation. Preferential attention was given to peer-reviewed articles, world health and national reports, and official policy proposals of reputable international agencies.

Information gathering and integration

Extracted data included study design, geographic location, population, nutritional indicators, biochemical processes that occurred in the development of the pancreas, and the reported association of malnutrition with the risk of diabetes.

The synthesis of findings was conducted descriptively to outline the biochemical pathways between maternal and childhood undernutrition and pancreatic dysfunction, the epidemiological trends of malnutrition and diabetes, and policy interventions to address food insecurity and nutritional health.

Discussion

From nutritional deprivation to metabolic failure: The emergence of T5DM

Although the broader consequences of malnutrition influence the overall health of the system, the most specific and devastating metabolic effect is the recently named T5DM, previously known as malnutrition-related diabetes mellitus (MRDM) [2]. In contrast to type 1 (autoimmune) or type 2 (lifestyle-related) diabetes mellitus, T5DM is a 'diabetes of poverty' resulting from chronic nutritional deficiencies in the most important growth periods, which causes irreversible insulin deficiency in the body [2, 10].

Type 2 diabetes mellitus is the most common form of diabetes and usually starts in adulthood; it is characterised by insulin resistance or insufficient insulin production [11]. Type 1 diabetes mellitus is a chronic condition in which the pancreas secretes little or no insulin [10]. Gestational diabetes mellitus occurs during pregnancy and poses high risks for both the mother and the child [12]. According to the IDF, diabetes encompasses diverse subtypes beyond the common type 1, type 2, and gestational forms. Approximately 1.5–2% of global cases consist of heterogeneous subtypes, including monogenic forms, autoimmune variants like latent autoimmune diabetes in adults, and secondary diabetes resulting from underlying pathologies or medications [13]. Despite their lower prevalence, these forms are clinically significant due to their distinct pathophysiology and specialized management requirements.

First identified in Jamaica in 1955, malnutrition-related diabetes primarily affects young, lean individuals (BMI <19 kg/m^2) in LMICs. These patients are frequently misdiagnosed with type 1 diabetes mellitus; however, they present a unique clinical phenotype: they do not develop ketonuria or ketosis despite profound hyperglycemia and high insulin requirements [2]. In 1985, the World Health Organisation (WHO) formally recognised MRDM, dividing it into two subclasses: Fibrocalculous pancreatic diabetes (FCPD) and protein-deficient pancreatic diabetes [7]. Although the WHO withdrew this classification in 1999, citing a lack of causal evidence [5, 14], the clinical reality in resource-poor regions has led to a modern revitalisation of these categories under the contemporary IDF framework, type 3c (formerly FCPD), categorised as "diabetes of the exocrine pancreas." This subtype is defined by physical, obstructive damage. Chronic undernutrition triggers non-alcoholic pancreatitis and the formation of pancreatic calculi (stones), leading to structural destruction of the organ [7, 14]. T5DM (formerly protein-deficient pancreatic diabetes) represents a functional insulin deficiency in a structurally "clean" pancreas. It is driven by the impact of severe early-life undernutrition on β–cell development and metabolic programming rather than mechanical injury [2, 7].

In a study of South Asian populations, Lontchi-Yimagou *et al.* [15] provided compelling evidence that "low BMI diabetes" (LD) commonly represents a metabolically distinct subtype of diabetes that does not fit the conventional classifications of type 1 or type 2 diabetes mellitus. In their state-of-the-art study utilizing advanced metabolic phenotyping, including C-peptide deconvolution, hyperinsulinemic-euglycemic clamp studies, and ^{1}H-NMR spectroscopy, LD individuals were directly compared with demographically matched cohorts of type 1 diabetes mellitus, type 2 diabetes mellitus, and nondiabetic controls. The LD group displayed a markedly reduced total insulin secretory response, which was significantly lower than that of both lean nondiabetic controls and individuals with T2D. Despite this impairment in insulin secretion, LD individuals exhibited preserved insulin sensitivity, as evidenced by lower endogenous glucose production and higher peripheral glucose uptake than T2D individuals. Additionally, visceral adiposity and hepatic lipid accumulation, hallmarks of insulin resistance in T2D patients, were significantly lower in the LD group. These findings strongly support the hypothesis that LD is not simply a lean variant of T2D but rather a distinct metabolic entity, likely influenced by early-life undernutrition, environmental stressors, and possibly unique genetic or epigenetic factors.

Some scholars argue that the heterogeneity, diagnostic uncertainty, and declining epidemiological relevance of MRDM render its reclassification as a distinct type of diabetes—referred to as "T5DM"—currently unnecessary [16]. They suggest that MRDM

represents one end of the spectrum of type 2 diabetes (T2D), characterised by an earlier onset, significant insulin deficiency, and a heightened risk of microvascular complications [17]. However, given its distinct pathophysiology, low-BMI diabetes aligns closely with the emerging framework of recently named T5DM.

The pathophysiological link: from deprivation to disease

Nutrition plays a critical role in the developmental programming of children. The physical and cognitive development of children is strongly dependent on their nutritional status, which is determined by the availability and levels of essential nutrients [18]. Poor nutrition during the early stages of development (fetal or infant stages) may induce epigenetic modifications in gene expression that increase susceptibility to diseases and adverse long-term health outcomes, some of which may be irreversible [19]. Possible biological mechanisms include alterations in tissue development that affect maternal nutritional and metabolic profiles, as well as impaired placental function, which ultimately affect fetal growth and development [20].

Pregnancy involves many metabolic adaptations and epigenetic regulators that drive changes in beta cell function, ensuring an adequate supply of nutrients to the developing fetus [21].

Impairment of pancreatic development during a critical growth period results in reduced β-cell mass and function, ultimately leading to reduced insulin secretory capacity and increased susceptibility to metabolic disorders, including diabetes [20]. Severe acute malnutrition has been linked to malabsorption and impaired glucose metabolism due to pancreatic dysfunction [22]. Protein deficiency has been reported to impair insulin secretion, resulting in the pancreas's inability to produce sufficient insulin to regulate blood glucose levels [22]. This can result in T5DM, characterised by insulin secretory defects rather than resistance. Early-life malnutrition negatively affects pancreatic β-cell development, critical for insulin production and glucose homeostasis, ultimately leading to a lasting reduction in insulin secretion that persists into adulthood [23]. Similarly, maternal malnutrition, low birth weight, and childhood malnutrition are risk factors for this type of diabetes [24]. Moreover, studies on animals have shown that maternal protein deficiency leads to reduced beta-cell mass and impaired regeneration of beta cells in offspring [25]. Similarly, research in humans indicates that neonates classified as small for gestational age have fewer islet cells and a lower pancreatic blood supply [21].

Emerging evidence suggests that problems with the incretin axis, which is essential for maintaining balanced blood sugar levels, may be a key pathological feature MRDM [26]. In cases of chronic undernutrition, such as starvation or early-life malnutrition, the incretin response of the gut is weakened. This leads to changes in the expression of signaling molecules such as PAK1 and β-catenin. These disruptions can reduce GLP-1 production and secretion from intestinal L cells. As a result, this contributes to the impaired insulin response typical of MRDM [22–26].

Experimental studies in rats have shown that when a fetus does not receive enough nutrition, the liver produces fewer growth hormone receptors, and the IGF-1 signaling pathways become weaker, leading to lasting impairments in somatic growth and metabolic efficiency [27].

This condition is much more common in poor countries, where malnutrition remains widespread and access to diabetes treatments is limited [2]; thus, proper identification and classification of T5DM are essential for creating suitable diagnostic guidelines, management approaches, and public health initiatives [4].

Regional-specific manifestations of T5DM

Malnutrition, a major global health challenge, is closely linked to the emerging burden of T5DM. In 2024, 42.8 million children under five were affected by wasting, including 12.2 million with severe wasting. The burden is greater in Asia (30.0 million) and Africa (11.7 million), with substantially lower prevalence in Latin America, the Caribbean (0.6 million) and Oceania (0.1 million); Australia and New Zealand reported fewer than 0.1 million cases [6]. Stunting affects 150.2 million children globally, concentrated largely in Southern Asia and Africa [6]. Paradoxically, these same high-burden regions for undernutrition are also experiencing a rising burden of T5DM, estimated to affect 20–25 million people worldwide, with the majority of cases concentrated in Southern Asia and Africa [2, 3]. While most common in LMICs, T5DM also affects vulnerable groups in high -income settings, including individuals facing food insecurity and migrants with early-life undernutrition, where it may be under-recognised [2, 9].

The global distribution of T5DM reflects significant structural inequalities in health, as this condition disproportionately affects populations in resource-constrained environments [28]. Below, we present geographic and population-specific perspectives on how this disease manifests.

South Asia

T5DM is a major health issue in South Asia, especially in Bangladesh, India and Pakistan. It has been established that young-onset type 2 diabetes in South Asians has a distinct clinical presentation, such as earlier onset, low BMI, and a rapid β-cell decline [15, 29, 30]. Moreover, genetic predisposition and intrauterine fetal programming, combined with subsequent overconsumption of nutrients and refined carbohydrates, leading to the epidemic, are contributing features of the South Asian phenotype [31, 32]. Among the resource-limited countries, malnutrition-related diabetes is a poorly studied subject in Bangladesh, although prevalence rates are high [8]. Additionally, income-based disparities show that underweight individuals are primarily found among poorer population groups [33]. Considering the

limitations of BMI in assessing obesity among South Asians, more body composition indices are necessary to address the rising burden of metabolic disease in the region [34]. Furthermore, the identification of T5DM in India underscores the urgent need for integrated strategies that address nutritional vulnerabilities from early childhood through adulthood [35].

Sub-saharan Africa

Malnutrition-related diabetes is unevenly distributed in Sub-Saharan Africa, and this situation is predetermined by endemic food insecurity and chronic undernutrition [15, 36]. T5DM in Sub-Saharan Africa is manifested in lean individuals without autoimmune evidence, and has been incorrectly diagnosed as type 1 diabetes [37]. Moreover, the burden of the T5DM is mostly borne in nations such as Nigeria, where food insecurity and stunting are the key factors contributing to the risk of T5DM [38, 39]. Additionally, the burden of the condition in Africa, which has remained historically underestimated, has led to poor management and outcomes [40].

Caribbean and Latin America

The Caribbean is known to be a location of early identification of T5DM, with the first incidence reported in 1955 in Jamaica [2, 41]. Irrespective of this historical background, the disease has been largely ignored; however, chronic undernutrition has continued to be an occurrence in areas with food insecurity and geopolitical tensions [40]. In the Caribbean and Latin America, there is a dual challenge of metabolic diseases and undernutrition. Most of the region is undergoing a nutritional transition, yet chronic undernutrition remains a significant issue [42]. This ongoing problem predisposes individuals to develop T5DM [8]. Furthermore, recognizing T5DM as a distinct clinical entity in nations like Mexico, where socio-political shifts often dictate access to healthcare, is critical. Such identification has far-reaching implications for community health, potentially averting diagnostic errors and substandard therapeutic interventions common among high-risk, vulnerable populations [41].

While the overarching driver of T5DM is rooted in global resource constraints [29], the specific catalysts for the disease vary by regional socioeconomic contexts. In South Asia, inequality is primarily income -based, where the poor face distinct nutritional vulnerabilities [33, 35]. In contrast, the burden in Sub-Saharan Africa is linked to endemic food insecurity and its physical impact on pancreatic development [36]. Meanwhile, in the Caribbean and Latin America, the condition is exacerbated by geopolitical instability and the complexities of a rapid nutritional transition [41].

Diagnosis and management of T5DM

The increased prevalence of T5DM in resource-limited regions demands heightened clinical awareness to ensure early diagnosis and management of this disorder [2, 5, 43]. Diagnosing T5DM involves a thorough evaluation of clinical features, relevant laboratory results, and the careful exclusion of other diabetes types [24]. The IDF working group is in the process of establishing standardised diagnostic criteria [2].

Diagnostic indicators and clinical profile

T5DM typically appears in adolescents and young adults and may present symptoms like type 1 diabetes, such as weight loss, excessive thirst, frequent urination, and fatigue. However, a key diagnostic signature of T5DM is a body mass index typically below 19 kg/m^2. Crucially, unlike T1DM, these patients do not show signs of autoimmune β–cell destruction, and they remain uniquely "ketone-resistant" despite dangerously elevated glucose levels [5]. According to the recent IDF classification, malnutrition-related diabetes is mainly differentiated through pancreatic imaging to distinguish between type 5 and type 3c. Type 5 is the non-calcific variant, where the pancreas is underdeveloped but structurally "clean" **(Table 1)**. Case reports from Ethiopia described children who met the Type 5 criteria: severe wasting, hyperglycemia without ketonuria, but without pancreatic calcification [5, 44, 45]. In Nigeria, Ubani *et al.* [46] and Oputa [47] reported cases of diabetes mellitus with pancreatic calcifications. While these patients share the type 5

Table 1 Clinical summary of Type 5 diabetes mellitus

Category	Clinical details and diagnostic indicators
Primary profile	Demographics: Adolescents and young adults.
	Body mass index: Typically, <19 kg/m² with a history of endemic undernutrition 2, 5, 9, 43.
Symptom triad	Core symptoms: Increased thirst, frequent urination, and profound exhaustion.
	Physical sign: Severe weight loss (wasting) 2, 5, 44.
Metabolic signature	The paradox: Severe hyperglycaemia combined with unique ketone-resistance. Patients rarely present with ketonuria or acidosis 2, 5.
Pathology	Non-autoimmune: No evidence of autoimmune β-cell destruction; pancreatic autoantibody tests are negative 24.
Differential diagnosis	Exclude Type 3c diabetes (formerly Fibrocalculous pancreatic diabetes): Requires imaging (Ultrasound/X-ray) to confirm a non-calcific pancreas. Type 5 lacks the stones/calcifications seen in Type 3c 46, 47.
Imaging findings	Radiographic evidence reveals a hypoplastic or small pancreas with unremarkable parenchyma (no stones or structural markers of pancreatitis) 5, 44, 45.
Treatment	Nutritional rehab: High-calorie, high-protein diet.
	Pharmacotherapy: Low-dose insulin or sulfonylureas with careful titration 4, 24.
Risk of misdiagnosis	Frequently misclassified as type 1 diabetes (Type 1 diabetes mellitus), leading to delayed or incorrect treatment protocols 4, 5, 45.

phenotype of low BMI and malnutrition, the presence of stones confirms FCPD (Type 3c), which often necessitates adjunctive enzyme replacement therapy due to exocrine failure.

The problem of misclassification, combined with the effects of comorbid infections and financial constraints, complicates management processes. [5, 45]. Classifying T5DM as either Type 1 or Type 2 often leads to delays in diagnosis and incorrect treatments, which can worsen patient health [4].

Management of T5DM

While global treatment protocols for T5DM continue to evolve, it is crucial to recognise this "diabetes of poverty" for timely diagnosis and to prevent potentially life-threatening mismanagement [3]. Unlike type 1 or type 2 diabetes, patients with T5DM may develop episodes of severe hypoglycaemia, despite persistent hyperglycaemia, highlighting the need for careful, individualised therapy [2]. Addressing malnutrition in LMICs requires more than just nutrition-specific measures, which can reduce stunting by only about 20%. It is essential to implement broader nutrition-sensitive interventions in areas such as agriculture, education, and social protection to tackle the underlying drivers of malnutrition, including poverty, food insecurity, and poor sanitation [1, 2].

Management of malnutrition-related diabetes follows the C-peptide–guided approach proposed by Garg [24], which classifies patients into three groups. Group A (C-peptide >1.0 ng/mL, mild disease) is managed with sulfonylureas, with metformin if needed. Group B (severe hyperglycaemia or oral therapy failure) requires insulin with careful dose titration. Group C (intermediate reserve) benefits from combined oral agents and low-dose basal insulin. Across all the treatment groups, nutritional support with adequate protein, energy intake, and micronutrient supplementation is essential for long-term metabolic stability [24].

The advantages of addressing the root causes of malnutrition extend beyond just preventing diseases; they also contribute to improved national development, a higher standard of living, and sustainable growth aligned with the 2030 development agenda. However, to realise these benefits and tackle the rising threat of T5DM in LMICs, accurately diagnosing and characterising this condition is an essential first step. This process is crucial for avoiding misdiagnoses, tailoring effective treatments, enhancing health outcomes, and improving food sourcing within the country.

Limitations

This review is limited by its reliance on previously published literature, which may vary in quality, methodology, and regional representation. In addition, evidence from LMICs remains sparse, and underreporting or misdiagnosis may contribute to important gaps in the available epidemiological data. As a narrative review, quantitative synthesis or meta-analysis was not conducted, and publication bias cannot be ruled out completely.

Conclusion

Recommendations include integrating T5DM into national health policies with clear guidelines; developing nutrition training for women, expectant mothers, and young children focused on protein supplementation and breastfeeding; launching campaigns to educate communities on the links between malnutrition and diabetes; investing in sustainable food systems and supporting smallholder farmers to address economic shocks; and establishing long-term studies and national registries to monitor T5DM; and promoting multisectoral collaboration to tackle malnutrition's root causes.

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Author contributions

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Conflict of interest

We do not have any conflict of interest.

Data availability statement

No datasets were generated or analyzed during the current study.

Supplementary file

None

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