

# Classifying a distinct form of diabetes in lean individuals with a history of undernutrition: an international consensus statement

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Since 1955, a form of diabetes in lean, young (BMI <18.5 kg/m<sup>2</sup>, age <30 years) individuals with a history suggestive of undernutrition from before birth and throughout childhood has been described in the literature. In 1985, WHO formally classified it as malnutrition-related diabetes, but subsequently removed the classification in 1999 over a disagreement as to whether undernutrition was a sufficient risk factor to cause this type of diabetes. Emerging evidence now strongly supports the distinct classification of this unique diabetes phenotype, which is characterised by substantial impairment of pancreatic insulin secretion, with normal hepatic and peripheral insulin sensitivity, an absence of ketoacidosis, and no islet cell autoantibodies. In this consensus statement, we synthesise this evidence to produce a set of common features of the disease, proposed pathogenetic mechanisms, and suggested management and prevention strategies. During a consensus meeting in Vellore, India, in January, 2025, type 5 diabetes was proposed as the nomenclature for this distinct form of diabetes, subsequently formalised at the International Diabetes Federation (IDF) World Diabetes Congress in April, 2025. We call upon the international diabetes community to recognise this distinct form of the disease, and to support the IDF's Type 5 Diabetes Working Group and its goals to promote more research into its phenotype, pathophysiology, and treatment, which will benefit millions of patients worldwide, particularly in low-income and middle-income countries.

## Introduction

Diabetes is associated with an increasing public health burden worldwide, and the number of people living with the disease is expected to increase markedly in low-income and middle-income countries (LMICs), which are facing a double burden of undernutrition and overnutrition. Although the growing burdens of obesity and population ageing are key risk factors for the increasing prevalence of type 2 diabetes, undernutrition also appears to contribute to the burden, since atypical forms of non-type-1 diabetes have been reported to occur in young, lean (BMI <18.5 kg/m<sup>2</sup>, age <30 years) individuals with a life course history of undernutrition.<sup>1</sup> This form of diabetes was first documented in *The Lancet* in 1955 by P Hugh-Jones, who named it J-type diabetes, after his observations in Jamaica of young, underweight people with insulin-resistant diabetes who were not prone to ketoacidosis.<sup>2</sup> Although initial clinical observations reported that individuals with J-type diabetes appeared to require large amounts of insulin, more extensive physiological studies have shown that most individuals with this form of diabetes have normal insulin sensitivity, but severe defects in insulin secretion.<sup>1,3</sup> In 1985, WHO recognised J-type diabetes as a distinct category of diabetes under the name malnutrition-related diabetes,<sup>4</sup> but removed it from its official classification in 1999, citing insufficient evidence of its association with malnutrition.<sup>5</sup> However, reports from many LMICs, including India, Pakistan, Bangladesh, Uganda, Ethiopia, Rwanda, Nigeria, and Indonesia,<sup>1,3,6–15</sup> support the existence of an atypical phenotype of diabetes in lean

individuals and its association with low socioeconomic status and a history of prolonged undernutrition. Moreover, the tenth and 11th revisions of the ICD—ie, ICD-10 (code: E12.9) and ICD-11 (code: E12.11)—both continue to provide classification codes for malnutrition-related diabetes.

Based on data (including from the Diabetes Atlas) on the global prevalence and percentage of people with diabetes in LMICs, 25 million people are estimated to have this form of diabetes.<sup>13,16,17</sup> Furthermore, a 2024 study on 20 years of trends in lean individuals with diabetes reports a similar prevalence increase to that of type 2 diabetes in South Korean adults.<sup>18</sup> However, due to disagreement on the naming, a shortage of recognition by WHO, and the fact that it generally occurs in impoverished areas of LMICs, this atypical form of diabetes has received much less attention than type 1 diabetes or type 2 diabetes, thereby leading to inadequate phenotyping and uncertain classification.<sup>19</sup> Additionally, the scarcity of both prospective cohort studies and mechanistic research has led to inadequate understanding of the natural history and pathophysiology of this entity. In addition, insufficient evidence for this form of diabetes makes it challenging to define diagnostic criteria and risk factors that are applicable to diverse populations. This form might be misdiagnosed due to diagnostic overlap with other atypical diabetes forms (eg, fibrocalculous pancreatic diabetes and antibody-negative type 1 diabetes), especially in resource-limited settings, leading to under-reporting of this form of diabetes and a continued diagnostic neglect in global research and

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For more on the Diabetes Atlas see <https://www.diabetesatlas.org>

policy agendas. Finally, treatment response to insulin and oral hypoglycaemic agents in individuals with this form of diabetes has not yet been evaluated in intervention studies, posing management challenges to health-care providers. Therefore, misdiagnosis and underdiagnosis are likely to have negatively impacted the clinical care and lives of millions of individuals worldwide. Incorrectly diagnosing these young, lean individuals with type 1 diabetes could lead to serious iatrogenic hypoglycaemia.

On Jan 8 and 9, 2025, the 39 signatories of the consensus statement met in person in Vellore, India, to present and review research pertinent to this neglected form of diabetes among lean and undernourished individuals. Participants included researchers who had extensively studied lean, undernourished patients with diabetes in LMICs, along with leaders of global diabetes organisations and internationally recognised experts in various areas of diabetes research. Each participant presented their relevant research findings, engaged in structured discussions to critically review the current literature, deliberated a differential diagnosis that would exclude other known forms of diabetes with a low BMI, identified research gaps, and explored opportunities for future research related to recognising a distinct classification and understanding the pathophysiology of this form of diabetes. Following these discussions, the signatories unanimously agreed that a distinct, non-autoimmune type of diabetes in undernourished individuals warranted its own unique classification for the purposes of diagnosis and management (the “Vellore Declaration”). In this Viewpoint, we describe the evidence reviewed leading to the international consensus; list the common features of the disease; and propose pathophysiological mechanisms, management guidelines, and priorities for future research. Recognising that other forms have already been described as type 3 diabetes<sup>20–22</sup> and type 4 diabetes<sup>23,24</sup> in the published literature, type 5 diabetes was proposed as the nomenclature for this form of diabetes. This name was formally announced by the International Diabetes Federation (IDF) at its World Diabetes Congress in April, 2025. At this meeting, IDF President Peter Schwarz officially launched the IDF's Type 5 Diabetes Working Group, which has been tasked with developing specific diagnostic criteria and treatment guidelines for this condition.

### Common features

Over the past seven decades, the phenotype we are calling type 5 diabetes has been increasingly recognised worldwide, most often in underweight individuals in low-resource settings.<sup>1,14</sup> Extensive physiological studies in lean individuals with diabetes, after carefully excluding other known forms of diabetes, have examined the individuals' metabolic characteristics using state-of-the-art technology.<sup>1,3</sup> These studies, along with others using various experimental measures, have collectively documented patients with impaired insulin secretion,

lower C-peptide concentrations, and normal insulin sensitivity, differentiating them from patients with type 2 diabetes.<sup>1,3,6,10–14,25–27</sup> However, insulin concentrations in these patients are not as low as those observed in patients with type 1 diabetes, and ketoacidosis is generally not reported.<sup>1,25,28</sup> Studies have shown that such patients often have a history of undernutrition early in life,<sup>1,6,14,26</sup> which continues into adulthood,<sup>6,26</sup> with the disease usually diagnosed in the third decade of life.<sup>14,26</sup> Lean individuals who have positive autoantibodies indicative of type 1 diabetes, such as antibodies against GAD-65 and IA-2, have been excluded from our definition.<sup>1</sup> On pancreatic imaging, patients with type 5 diabetes should have no evidence of pancreatic calcifications, ductal hypoplasia, or ductal dilatation, as these features are consistent with fibrocalculus pancreatic diabetes.<sup>1,14</sup> On dual-energy absorptiometry or bioimpedance analysis, these patients have low total and truncal fat mass (particularly hepatocellular lipids).<sup>1,3</sup> Additionally, a few sociodemographic features characterise type 5 diabetes. Studies have shown that most individuals with type 5 diabetes belong to low

### Panel: Features of type 5 diabetes

#### Commonly reported features

- BMI less than 18.5 kg/m<sup>2</sup> for adults<sup>\*29,30</sup>
- Moderate to severe hyperglycaemia<sup>1,3</sup>
- Low C-peptide concentrations in the fasting or random state,<sup>1,3</sup> or low serum insulin
- Ketosis resistance<sup>1,14</sup>
- Negative for clinical signs of insulin resistance (eg, acanthosis nigricans)
- Negative for GAD-65 antibody<sup>1,3</sup> (and IA-2 and ZnT8 antibodies, if available)

#### Occasionally reported features

- History of early-life undernutrition, as evidenced by one or more of the following
  - History of low birthweight<sup>1,31</sup>
  - History of undernutrition in infancy<sup>14</sup>
  - History of stunting in childhood<sup>32</sup>
- A history of undernutrition in early life (intrauterine, infancy, childhood, and adolescence), as shown by
  - Weight-for-height less than –3 SD for severe acute malnutrition in children younger than 5 years<sup>\*33</sup>
  - BMI less than –3 SD for age 5–19 years<sup>\*34</sup>
- Normal pancreas on comprehensive pancreatic imaging (ultrasonography or preferably CT)<sup>1</sup>
- Low total body fat percentage on either dual-energy absorptiometry or bioimpedance analysis<sup>1,3</sup>
- Negative monogenic β-cell studies for genetic variants
- Low socioeconomic status<sup>25,32</sup>
- Rural origin<sup>6,14</sup>

\*Although this consensus statement proposes cutoffs for BMIs and the diagnosis of undernutrition, more research is required to establish specific cutoff values for diagnosing undernutrition associated with type 5 diabetes.

socioeconomic conditions and reside in rural areas, with a low dietary intake of proteins and calories.<sup>1,6,14</sup> Features that are commonly, and occasionally, associated with type 5 diabetes are summarised in the panel.

Notably, although not all the aforementioned features are present in every person with type 5 diabetes, their presence would help distinguish this form of diabetes from other forms such as type 1 diabetes; type 2 diabetes with weight loss due to uncontrolled hyperglycaemia and irregular medications; diabetes secondary to pancreatic disorders, including fibrocalculous pancreatic diabetes;<sup>35</sup> maturity-onset diabetes of the young;<sup>36</sup> hereditary lipodystrophic disorders;<sup>37</sup> and ketosis-prone diabetes.<sup>38</sup>

### Pathophysiology

The pathophysiology of type 5 diabetes is incompletely understood. However, intrauterine undernutrition, followed by persistent undernutrition during childhood and adolescence, might increase risk.<sup>39,40</sup> If low-birthweight individuals are exposed to either isoenergetic or hyperenergetic nutrition and diet after birth, they tend to have catch-up growth in childhood and adolescence (figure).<sup>41–47</sup> Studies show that overnutrition in an individual with a history of intrauterine undernutrition frequently results in overgrowth, which in turn is likely to predispose the individual to adiposity, ectopic fat deposition, insulin resistance, and a compensatory overgrowth of pancreatic  $\beta$  cells and the liver, probably leading to classic type 2 diabetes (figure).<sup>41–48</sup>

Although impaired fetal growth has been established as a substantial environmental risk factor for type 2

diabetes, patients with lean diabetes associated with undernutrition—in contrast to individuals with type 2 diabetes—have also been exposed to undernutrition in childhood and adulthood (figure).<sup>1,39</sup> Intrauterine undernutrition, followed by persistent undernutrition during childhood and adulthood, might cause the pancreatic  $\beta$  cells to secrete insufficient amounts of insulin.<sup>6,32</sup> Chronic, persistent undernutrition from fetal life into adulthood can also be associated with a deficiency of antioxidants (such as vitamins A, C, and E; catalase; and glutathione peroxidase), which can lead to dysfunction of two transcription factors, namely, PDX-1 and MafA, resulting in defective insulin gene expression, negatively impacting pancreatic  $\beta$ -cell function and reducing insulin secretion.<sup>49–51</sup> Moreover, excessive amounts of reactive oxygen species can increase the rate of pancreatic  $\beta$ -cell apoptosis, leading to reduced cell mass and insulin secretory defects.<sup>51</sup>

Body composition analysis has shown that individuals with type 5 diabetes have substantially lower fat mass than those with type 2 diabetes.<sup>1,3</sup> Several studies have also shown that insulin sensitivity is normal in type 5 diabetes,<sup>1</sup> as opposed to in type 2 diabetes. However, since most individuals with undernutrition do not develop diabetes despite the pathogenetic mechanisms mentioned previously, genetic factors<sup>52</sup> or epigenetic modifications of pancreatic  $\beta$ -cell function<sup>53</sup> might be involved in the pathogenesis of type 5 diabetes. Furthermore, the pathophysiology of this form of diabetes is likely to be multifactorial, as many of the aforementioned factors could play a role in any given individual.

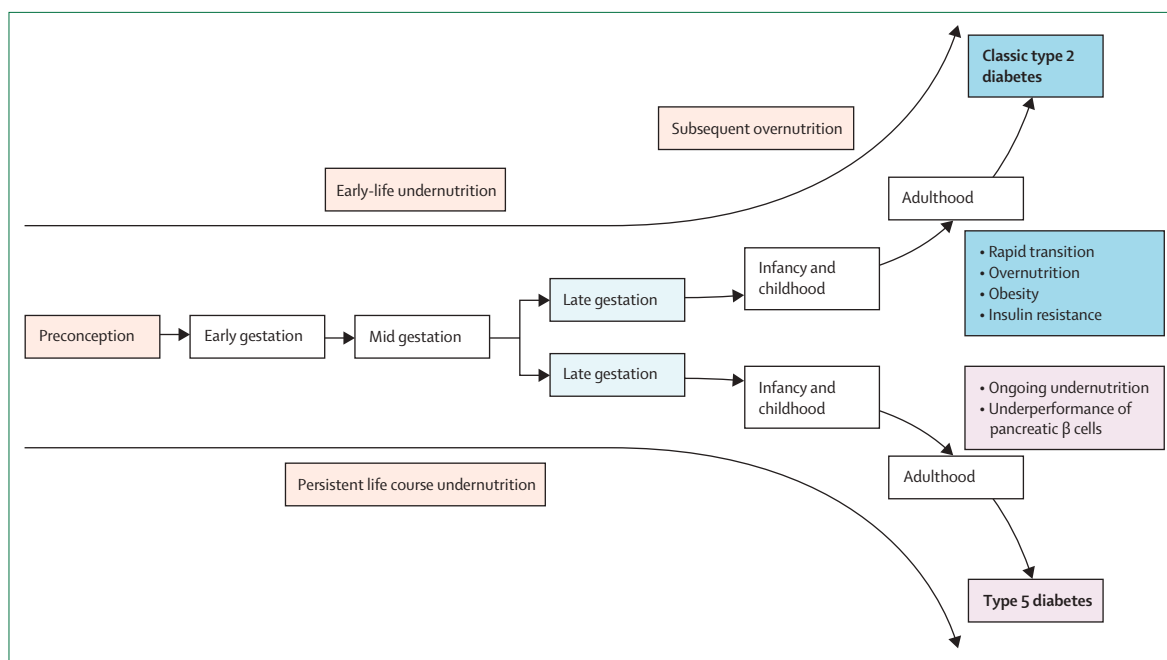


Figure: Life course evolution of adult diabetes phenotypes in different socioeconomic conditions

Prospective studies and randomised controlled trials of undernutrition cannot be done ethically in humans. However, experimental animal models,<sup>50</sup> prospective cohort studies in individuals exposed to famine,<sup>53,54</sup> and human observational studies<sup>46</sup> have provided strong evidence for an association between undernutrition and the clinical features of type 5 diabetes, which might support its classification as a distinct form of diabetes.

### Management

Due to the paucity of data, the ideal management approach for type 5 diabetes is currently not based on evidence-guided interventions. Clearly, the standard recommendation to lose weight, for people with newly diagnosed type 2 diabetes who are overweight, is inappropriate in undernourished individuals.<sup>1</sup> The traditional pharmacological management approaches for type 2 diabetes, such as metformin, can induce weight loss, and the role of insulin therapy in the acute and chronic treatment of this form of diabetes needs further verification.<sup>1</sup> Weight loss could be detrimental in lean individuals with a low muscle mass. Insulin therapy might be challenging to administer in low-resource settings and has substantial financial implications for patients. Inappropriate insulin treatment could induce hypoglycaemia, which can be a particular risk in settings with food insecurity and where glucose monitoring might not be affordable.

Individuals with type 5 diabetes might only need minimal amounts of insulin, or alternative approaches to stimulate insulin secretion, to manage hyperglycaemia and avoid hypoglycaemia. Sulfonylureas could be useful,<sup>25</sup> but also need to be used cautiously to avoid hypoglycaemia. DPP-4 inhibitors can be safe, yet might not be sufficiently effective, considering the degree of insulin deficiency. GLP-1 agonists are unlikely to have a role due to their effect on weight loss and worsening of sarcopenia.<sup>55</sup> Metformin has been used empirically with some success despite not addressing the reduced insulin secretion in these patients. Although some of our authors have empirical experience of using metformin in low BMI individuals (BMI <18.5 kg/m<sup>2</sup>), we are not aware of any literature on its effectiveness in individuals with low BMI. SGLT2 inhibitors might be effective, given that their mechanism of action is unlikely to be affected by insulin deficiency or absence of insulin resistance; however, the associated calorie and weight loss is unlikely to be beneficial in this group. Current treatment for type 5 diabetes is empirical, and data from additional nutritional and therapeutic studies are needed to address this issue.

In addition to pharmacological therapy, clinicians should understand the unmet nutritional needs of patients with type 5 diabetes. Nutritional rehabilitation and health education are essential for promoting a well balanced diet in terms of protein and energy intake, as well as micronutrients, which are crucial for long-term and

feasible treatment.<sup>19,53</sup> For individuals with low socioeconomic conditions, locally available, culturally acceptable, and cost-effective dietary interventions are the most sustainable. These can include low-cost, energy-dense staple foods high in protein and complex carbohydrates (such as lentils, legumes, oil-enriched cereals, and fortified grains), complemented by nutrition education for families and community health workers. When possible, we advocate for integration with existing public health nutrition programmes, such as those that provide midday meals, maternal and child nutrition support, or conditional cash transfers. Additionally, community-based management of undernutrition with ready-to-use therapeutic foods or home-fortified foods might be an effective approach for individuals with severe nutritional deficiencies.

Additionally, management guidelines should address the psychosocial, cultural, and educational challenges faced by patients and their families. Given its atypical presentation, health education needs to be provided to the community regarding the clinical presentation, diagnosis, and nutritional and pharmacological guidelines on type 5 diabetes to promote treatment adherence. Future research should explore methods to integrate education and psychosocial support to improve health outcomes.

### Prevention

To effectively address the global burden of type 5 diabetes, countries need to work towards achieving the targets set by the Sustainable Development Goals (SDGs): specifically, SDG 1 to end poverty in all its forms everywhere, and SDG 2 to end hunger, achieve food security, improve nutrition, and promote sustainable agriculture. To attain these goals, government and non-government agencies should consider: (1) increasing the availability of simple, low-cost, nutritious, protein-rich foods, especially for women of childbearing age, infants, and children (also involving ideal breastfeeding practices); (2) poverty reduction programmes, including job training, employment, food banks, and cash transfers; (3) screening for undernutrition and referring affected individuals to appropriate clinical care that includes nutritional interventions; and (4) improving the awareness and knowledge of the clinical features and management of type 5 diabetes in both health-care providers and policy makers. However, effective prevention strategies will need to be informed by subsequent research.

### The future

Our discussion around type 5 diabetes is based on existing literature pertaining to lean individuals with undernutrition and pertinent animal models. However, this form of diabetes phenotype requires extensive further study to understand its cause, burden, and natural history. Additionally, longitudinal studies are essential for understanding the life course impact of



undernutrition on diabetes risk, and the potentially multifactorial pathophysiology of this form of diabetes. Establishing a global registry could facilitate such research. Global research collaborations to evaluate the diagnostic criteria for this form of diabetes in diverse populations would help refine the definition of this phenotype. Finally, interventional studies are needed to inform evidence-based management guidelines for clinicians treating patients with type 5 diabetes. The Vellore Declaration calls upon the international diabetes community to formally recognise this neglected entity, which likely affects the quality and length of life of millions of people worldwide. We encourage international organisations such as the IDF and WHO to promote more research into the phenotype, pathophysiology, and treatment of type 5 diabetes.

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PW, FJ, NT, CSY, AAV, DK, SB, MB, MSB, CB, SW, NPS, PS, AGJ, and MH contributed more than others. MH conceptualised the consensus

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#### Declaration of interests

We declare no competing interests.

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