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REVIEW

Metabolic disorders in young people around the world

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Abstract

Youth-onset metabolic diseases, including obesity and type 1 and type 2 diabetes, and their associated cardiometabolic complications represent a major global health challenge. The incidence and prevalence of these conditions vary across regions, with rising trends and a heavier burden observed in middle- and low-income countries. Diet, physical activity and lifestyle choices are key factors in the development and progression of metabolic diseases during childhood and adolescence, along with additional risk factors such as genetic predisposition, ancestry, ethnicity, lifetime events (i.e. puberty) and other environmental factors. Disparities in access to healthcare, diagnostic and management capabilities and treatment options across the world affect outcomes, contributing to high morbidity and mortality rates, particularly in low-resource settings. Compared with onset during adulthood, an early diagnosis of metabolic diseases is associated with a higher risk and severity of complications, including adverse vascular outcomes and premature mortality. Although clinical signs of cardiovascular complications typically appear in adulthood, they are the result of a long, subclinical disease process that can begin in childhood and adolescence. This underscores the need for early prevention strategies and effective treatments to reduce the short- and long-term health impacts of these conditions. Addressing the rising prevalence of metabolic diseases, especially among vulnerable populations, requires comprehensive, culturally tailored actions that consider the available resources in diverse settings.

Keywords Cardiometabolic · Equity, diversity and inclusion · Obesity · Review · The metabolic syndrome · Type 1 diabetes · Type 2 diabetes · Youth

Abbreviations

DALY	Disability-adjusted life-year
GBD	Global Burden of Disease Study
GLP-1	Glucagon-like peptide-1
HICs	High-income countries
LICs	Low-income countries
LMICs	Low- and middle-income countries
SDI	Sociodemographic index
TODAY	Treatment Options for type 2 Diabetes in Adolescents and Youth

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Introduction

Metabolic diseases such as obesity and diabetes, along with the associated cardiometabolic complications, represent a significant global health burden, particularly among young people [1–3]. Early onset of these conditions contributes to reduced quality of life, increased morbidity and premature death, and has high healthcare costs and intergenerational effects. The prevalence and incidence of metabolic diseases have increased over the past two to three decades, largely driven by rapid economic development and environmental and societal changes [1–3]. There are notable regional and socioeconomic disparities in incidence and prevalence, with rising trends and a higher burden observed in low- and middle-income countries (LMICs). Furthermore, variation in the epidemiology of metabolic diseases is linked to ancestry and ethnicity [4, 5].

Childhood and adolescence are crucial developmental stages marked by significant biological and psychosocial changes [6, 7]. These changes can influence the risk of developing metabolic diseases and related complications, as well as their management (Fig. 1). Puberty, in particular, is

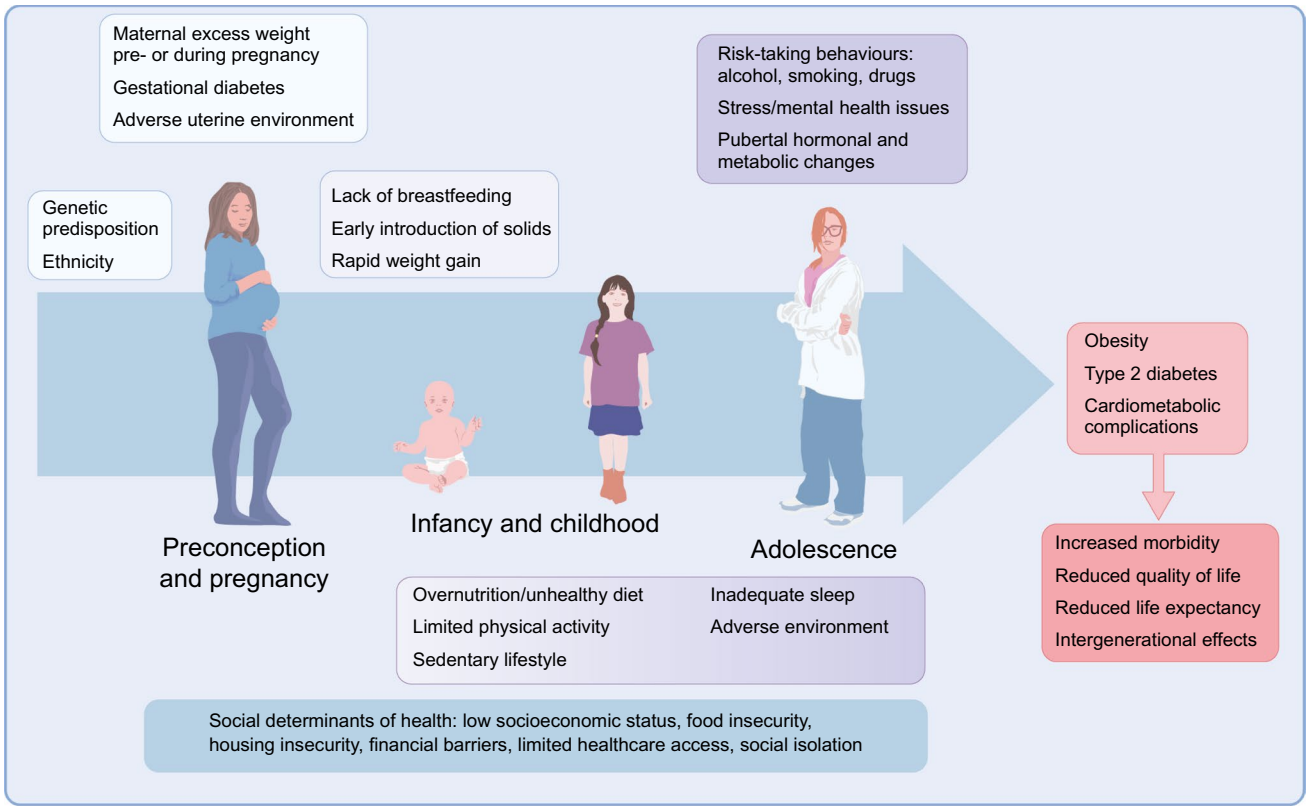

Fig. 1 Life course of metabolic diseases in young people. The figure summarises the main risk factors acting during prenatal life, infancy, childhood and adolescence that contribute to the development of metabolic diseases, such as obesity and diabetes, as well as associated cardiometabolic complications. This figure is available as part of a downloadable slideset

accompanied by several hormonal and metabolic changes, including a physiological decrease in insulin sensitivity and alterations in cardiometabolic risk factors [8]. Evidence suggests that, in children with obesity, puberty can influence the transition from a metabolically healthy status to an unhealthy status [9]. It is also a well-known risk factor for type 2 diabetes and may act as a trigger for type 1 diabetes, whose peak incidence occurs between the ages of 10 and 14 years [10]. Notably, early signs of cardiometabolic complications related to diabetes often emerge during puberty [11]. Adolescence is also a phase of life characterised by a growing desire for independence and autonomy, often accompanied by risk-taking behaviours (drugs, alcohol and smoking). These behaviours can contribute to an increased risk of complications and suboptimal adherence to lifestyle and medical interventions [12]. However, childhood and adolescence also present unique opportunities to improve metabolic health [6, 7]. These developmental stages of life provide a critical window for implementing preventive interventions targeting unhealthy behaviours such as poor diet, excessive energy intake, insufficient physical activity, inadequate sleep, and smoking and alcohol use, while also promoting mental health. These early interventions have the potential to yield long-term health benefits [7, 13].

This review provides an overview of common metabolic diseases, with a focus on youth-onset obesity and type 1 and type 2 diabetes and related cardiometabolic complications. It discusses geographical and socioeconomic variations in the incidence, management and burden of these conditions, alongside the role of ancestry and ethnicity in shaping their epidemiology and outcomes. The review also emphasises the importance of early prevention strategies and effective treatment approaches to mitigate the short- and long-term health impacts of these conditions. With regard to the terminology used in this review to distinguish populations ('ancestry', 'ethnicity' and 'race'), when referring to previously published work we use the terms used by the authors.

Worldwide trends in childhood obesity and associated cardiometabolic risk factors

The prevalence of overweight and obesity in children and adolescents has increased significantly in recent decades, making it a major global public health concern [14]. According to the WHO, in 2022, 37 million children under the age of 5 years and over 390 million children and adolescents

aged 5–19 years were overweight or living with obesity [14]. A recent systematic review and meta-analysis including data from over 45 million children and young people from 154 different countries and regions reported that approximately one in five (22.2%) children were overweight [15]. The overall prevalence of obesity was 8.5%, but there was substantial variation across geographical regions, race and ethnicity and country income, with the highest prevalences in Hispanic and Black non-Hispanic children compared with non-Hispanic White and Asian peers (Table 1).

The development of overweight/obesity is related to several behavioural, environmental and sociocultural influences, which represent potential targets for interventions [16–18]. While the heritability of obesity is between 40% and 70% [16, 17], early exposure to an obesogenic environment plays a key role in its development [18]. A strong link exists between maternal overweight/obesity, excessive weight gain during pregnancy, gestational diabetes, an adverse uterine environment and the risk of childhood obesity and related complications [19, 20] (Fig. 1). Postnatally, lack of breastfeeding and early introduction of complementary foods and beverages, particularly in formula-fed babies, have been associated with an increased risk of excess weight gain [18]. This risk is exacerbated by the widespread availability of ultra-processed and energy-dense foods, and physical inactivity. Of note, the recent increase in childhood obesity has been particularly pronounced among poorer populations and in rural areas in LMICs, where limited access to affordable healthy food is a major contributing factor [21]. Lack of exercise is also common in LMICs, particularly among girls and women living in urban informal settlements, because of restricted space and fewer opportunities for physical activity [22]. Moreover, children from families with lower parental education or income levels are more likely to be overweight or living with obesity [23].

Table 1 Prevalence of obesity in children and adolescents between 2000 and 2023 based on geographical region, race and ethnicity and income

Characteristic	Prevalence (95% CI) (%)
Geographical region	
Polynesia	19.45 (16.06, 23.07)
Caribbean	19.22 (15.1, 23.7)
Northern America	17.17 (16.59, 17.75)
Central America	15.85 (14.23, 17.55)
Western Asia	9.94 (9.03, 10.88)
South America	9.38 (8.24, 10.59)
Northern Africa	9.22 (7.32, 11.3)
South-Eastern Asia	8.71 (8.26, 9.17)
Southern Europe	8.42 (7.84, 9.01)
Eastern Asia	7.78 (7.24, 8.32)
Australia and New Zealand	6.99 (5.74, 8.36)
Micronesia	5.80 (3.95, 7.98)
Southern Asia	5.79 (5.17, 6.45)
Southern Africa	5.76 (4.24, 7.50)
Eastern Europe	4.58 (3.75, 5.50)
Northern Europe	4.55 (3.57, 5.63)
Central Asia	4.28 (2.46, 6.58)
Eastern Africa	4.12 (3.30, 5.04)
Western Africa	3.95 (3.13, 4.87)
Western Europe	3.79 (3.38, 4.22)
Melanesia	3.79 (1.84, 6.40)
Middle Africa	2.36 (1.83, 2.96)
Race and ethnicity	
Hispanic	23.55 (20.66, 26.56)
Black	16.64 (14.06, 19.39)
White	12.28 (11.19, 13.42)
Asian	9.97 (8.73, 11.29)
Country or region income	
High income	9.29 (8.95, 9.64)
Upper-middle income	8.50 (8.02, 8.99)
Lower-middle income	6.35 (6.09, 6.62)
Low income	3.60 (2.54, 4.83)

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The rising rates of youth-onset overweight and obesity are concerning, given their strong association with both short- and long-term complications affecting many organs and tissues. In particular, youth-onset obesity is linked to multiple cardiometabolic risk factors, including insulin resistance, type 2 diabetes, dyslipidaemia, hypertension and metabolic dysfunction-associated steatotic liver disease, from an early age [24]. Early-onset obesity is also associated with subclinical signs of cardiovascular damage, including atherosclerosis and left ventricular hypertrophy, and cardiovascular events as early as 40 years of age [25].

Cardiometabolic risk factors associated with obesity often cluster together and the concept of the metabolic syndrome is often used to define the constellation of visceral obesity, hypertension, dyslipidaemia and abnormal glucose tolerance [26].

According to a recent systematic review, the prevalence of the metabolic syndrome in youth with obesity was 29.4%, varying from 2.1% to 74.4% across studies [27]. The metabolic syndrome is an emerging issue in LMICs, where its prevalence is estimated to be between 24.1% and 56.3% among individuals with overweight and obesity compared with 3.98–8.91% in the general population, although comprehensive data from certain regions, particularly sub-Saharan Africa, are lacking [27, 28]. Racial and ethnic differences in the prevalence of individual cardiometabolic risk factors and the metabolic syndrome exist, even after accounting for environmental factors [4]. For instance, non-Hispanic Black adolescents show lower prevalences of the metabolic

syndrome and its individual components than their Mexican American peers [4].

There is still a lack of consensus around the definition of the metabolic syndrome for the paediatric population and different criteria have been proposed over time, leading to variations in prevalence and incidence across different studies [26, 29]. The developmental changes occurring during childhood and adolescence also impact the long-term reliability of a diagnosis of the metabolic syndrome, with a significant proportion of individuals diagnosed in childhood no longer meeting the criteria during follow-up and when transitioning to adulthood. Considering individual cardiometabolic risk factors as continuous rather than dichotomous variables is more useful for screening, prevention and management strategies during childhood and adolescence [30].

Youth-onset type 2 diabetes across the globe

The incidence and prevalence of type 2 diabetes in youth have increased over recent decades in many countries, although with variable rates based on region, ethnic group and socioeconomic status [31–34] (Fig. 2). The SEARCH for Diabetes in Youth study in the USA has reported a doubling in the incidence of type 2 diabetes between 2002 and 2018 [31, 32]. Increasing trends have also been observed in other parts of the world. Data from the Global Burden of Disease Study (GBD) 2019 [33] showed that, from 1990 to 2019, there were significant increases in the age-standardised incidence rate (per 100,000 population), from 117.22 to 183.36, and the age-standardised disability-adjusted life-years (DALYs) rate, from 106.34 to 149.61, for type 2 diabetes in adolescents and young adults (aged 15–39 years). Countries with a low-middle and middle sociodemographic index (SDI) had the highest age-standardised incidence rates and age-standardised DALY rates in 2019, whereas countries with a low SDI had the lowest age-standardised incidence rates but the highest age-standardised mortality rates [33]. More recent data from the multinational SWEET registry, covering the period from 2012 to 2021, confirmed a worldwide increase in type 2 diabetes in young people diagnosed before the age of 21 years [34]. The proportion of new cases rose from 3.2% in 2012/2013 to 6.0% in 2020/2021. Significant variability was observed across regions, with the lowest rates in Europe and the highest in North America. From 2012 to 2021, a notable increase was observed in Europe, Australia/New Zealand and North America, while changes in Asia/the Middle East and Africa did not reach statistical significance.

Youth-onset type 2 diabetes results from genetic, environmental and lifestyle factors that differ among individuals and populations [35] (Fig. 1). The genetic background is stronger than in type 1 diabetes, supported by the observation that 74–100% of youth presenting with type 2 diabetes have a first- or second-degree relative with the same condition [35]. While obesity is a major risk factor for type 2 diabetes, not all young people with this condition are living with obesity [35]. Of note, the prevalence of obesity is lower in Asian youth with type 2 diabetes than in other ethnicities [36], suggesting that genetic predisposition and other environmental exposures also play a role.

Early life determinants affecting the intrauterine environment, such as maternal obesity and gestational diabetes, as well as small size at birth, have been associated with an increased risk of developing type 2 diabetes [35] (Fig. 1). Youth-onset type 2 diabetes also shows significant disparities across racial and ethnic groups [37, 38], being particularly prevalent among Indigenous Australian, American Indian and Canada's First Nation peoples [35, 39] (Fig. 2). In the Treatment Options for type 2 Diabetes in Adolescents and Youth (TODAY) study, 30% of youth were African American, 40% were Hispanic/Latino and 6% were American Indian [40]. In addition, type 2 diabetes disproportionately affects marginalised and socioeconomically vulnerable youth, with 41–59% of participants across a range of cohort studies on type 2 diabetes in youth, including TODAY and SEARCH for Diabetes in Youth, living in poverty or socially disadvantages households [41].

Youth-onset type 1 diabetes across the globe

Type 1 diabetes, a chronic disorder in which autoimmune destruction of pancreatic beta cells leads to absolute lifelong insulin deficiency, is the most common form of diabetes in children and adolescents [42]. According to the most recent epidemiological data from the IDF, in 2024, 9.15 million individuals worldwide were living with type 1 diabetes; of these, 1.81 million (19.8%) were aged under 20 years, with an estimated 219,000 new diagnoses [43]. A systematic review including data from 55 countries found an overall incidence rate of 14.07 per 100,000 person-years between 2000 and 2022 for those aged under 20 years, with substantial variability between countries and geographical regions. Finland and high-income North America had the highest incidence rates, at 56.81 and 28.78 per 100,000 person-years, respectively, whereas Western sub-Saharan Africa had the lowest rate, at 0.66 per 100,000 person-years [44] (Fig. 3). Alarmingly, the global burden of type 1 diabetes in youth is expected to rise substantially in the coming decades, with an expected increase in prevalence of 65% by 2060, particularly in LMICs [45, 46].

The reported epidemiological variation across countries and regions is multifactorial, with interactions between

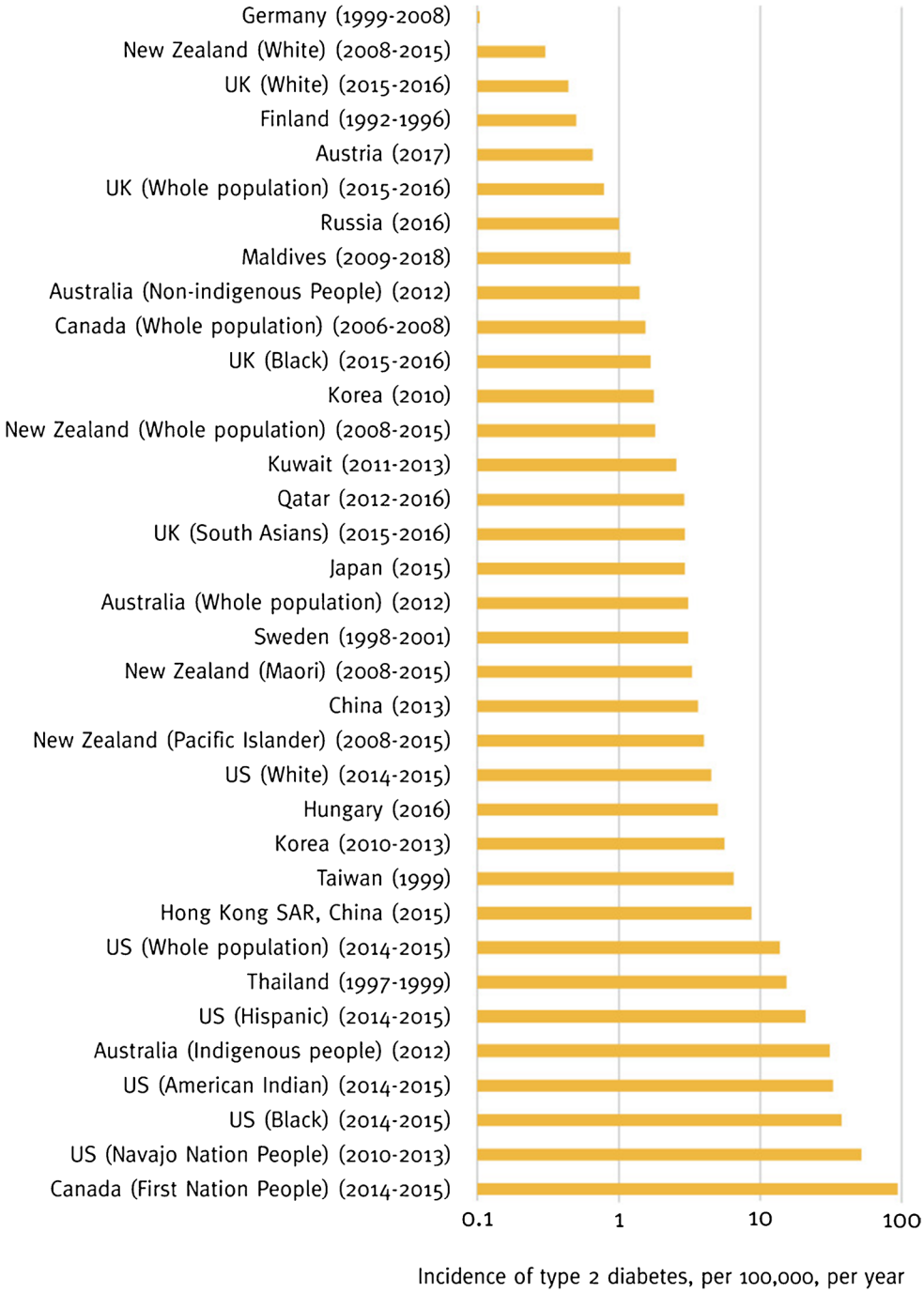

Fig. 2 Most recent data available on the incidence of type 2 diabetes in youth ranked by region and ethnicity. Reproduced without modifications from the IDF diabetes atlas, 10th edition [39], under the terms of the CC BY-NC-ND 4.0 licence (https://creativecommons.org/licenses/by-nc-nd/4.0/). This figure is available as part of a downloadable slideset

genetic predisposition and environmental factors [47]. The role of genetics in the autoimmune process leading to type 1 diabetes is supported by the concordance observed in monozygotic twins, familial clustering of this condition and findings from studies on migrant populations [47]. A comprehensive multi-ancestry genome-wide association study identified 78 chromosomal regions influencing type 1 diabetes risk, underscoring the complex genetic architecture of this condition across diverse populations [5]. Although several loci have been linked to type 1 diabetes, 30–60% of familial clustering is attributable to the HLA region [47].

Genetic factors play a role in explaining some of the differences in the prevalence of type 1 diabetes among various ethnic groups. Studies suggest that certain genetic risk factors (i.e. *HLA-DR3* and *HLA-DR4* alleles) are more prevalent in White populations, potentially contributing to higher incidence rates in these groups [48]. In contrast, the *HLA-DR7* allele may increase risk among African American

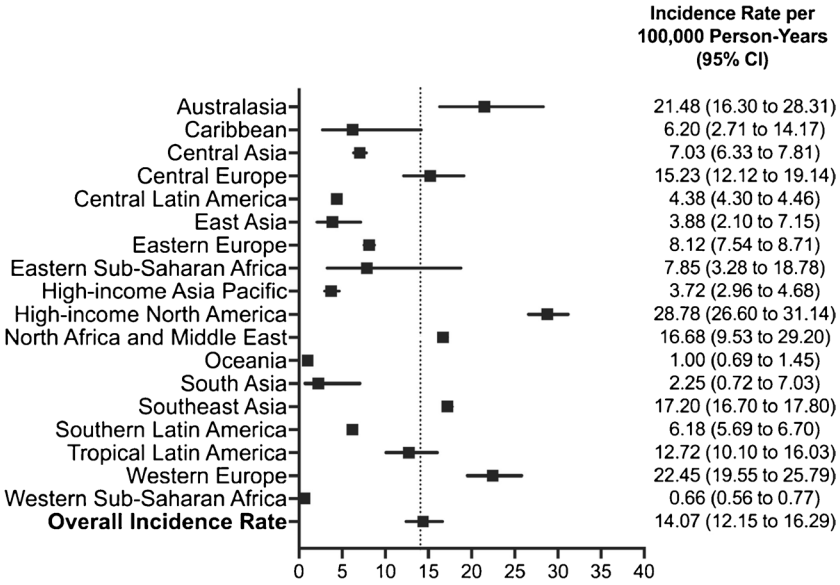

Fig. 3 Incidence of type 1 diabetes in youth between 2000 and 2022 ranked by region. Reproduced without modifications from Hormazábal-Aguayo et al [44] under the terms of the CC BY-NC-ND 4.0 licence (https://creativecommons.org/licenses/by-nc-nd/4.0/). This figure is available as part of a downloadable slideset

populations, while *HLA-DR9* is associated with increased susceptibility in Japanese individuals [49]. In Asian populations, the susceptible *DR* alleles often occur together with neutral or even protective *DQ* alleles, probably contributing to the lower incidence rates in these regions [50].

The rapid increases in type 1 diabetes incidence and prevalence during recent decades cannot be explained by genetic alterations, which typically take much longer to manifest [47]. In addition, the increased prevalence in lower risk genotypes, especially in younger age groups, strongly favours an impact of environmental determinants [51]. Regional variations in type 1 diabetes incidence between countries, and even within some small countries with genetically homogeneous populations, also support the role of environmental factors [52]. Given the consistently high incidence of type 1 diabetes in high-income countries (HICs), socioeconomic status and nutrition and lifestyle factors might be implicated in changes in its epidemiology [53]. The rapid rise in obesity over recent decades may plausibly have an impact on the incidence of type 1 diabetes [54]. The 'accelerator hypothesis' implicates insulin resistance from excess weight gain and physical inactivity in hastening beta cell apoptosis in susceptible individuals [55].

In addition, lower socioeconomic status is closely linked to the achievement of less favourable glycaemic targets and reduced access to advanced diabetes care tools, including continuous glucose monitoring and hybrid closed-loop systems [56, 57]. There is also a substantially higher burden of type 1 diabetes in racial and ethnic minority young people [58]. Non-Hispanic Black and Hispanic individuals experience higher HbA_{1c} levels and lower use of diabetes technologies than their non-Hispanic White counterparts [58].

The burden of early-onset diabetes: risks of complications and early mortality

Risk of complications in youth with diabetes Children and adolescents with type 1 and type 2 diabetes experience higher morbidity and mortality rates compared with the general population due to the acute and chronic complications associated with these conditions [59–61]. The long-term health burden of youth-onset diabetes is primarily driven by the associated cardiovascular complications [62], with individuals diagnosed in childhood facing a greater risk of these complications than those diagnosed in adulthood [11, 63]. This disparity is attributed to the longer diabetes duration and consequent prolonged exposure to the diabetes milieu. In addition, youth with diabetes exhibit a more aggressive disease process than adults, as reflected by more severe insulin resistance in youth with type 2 diabetes, and more rapid deterioration of pancreatic beta cell function in youth with both type 1 and type 2 diabetes [63, 64]. Furthermore, the risk of complications in this young population is affected by lifetime events such as puberty and the related hormonal changes, a decline in insulin sensitivity, psychological issues and risk-taking behaviours [65].

Although overt clinical manifestations of vascular complications, such as cardiovascular events, end-stage renal disease, proliferative retinopathy and neuropathy, typically occur in adulthood, their incidence represents the result of a long subclinical disease process starting earlier in life [66, 67]. Subclinical manifestations of vascular complications, which are often found in youth with diabetes, include increases in urinary albumin excretion (moderately increased albuminuria), background retinopathy, changes in

vascular function and structure (increased carotid intima–media thickness, arterial stiffness, endothelial dysfunction) and cardiac autonomic dysfunction [11, 66, 67]. Data from the SEARCH study highlight that up to one in three youth with type 1 diabetes and nearly three in four with type 2 diabetes show evidence of early manifestations of at least one vascular complication after a mean diabetes duration of 8 years [68]. Youth with type 2 diabetes show an earlier onset of complications than those with type 1 diabetes, as well as a higher prevalence of diabetic kidney disease (20% vs 6%), retinopathy (9% vs 5.6%) and peripheral neuropathy (18% vs 8.4%), even after accounting for differences in HbA_{1c}, BMI and blood pressure [68]. Cardiometabolic risk factors such as obesity, hypertension, dyslipidaemia and insulin resistance are common among adolescents with diabetes and contribute to the risk of complications [63, 68, 69]. In the SEARCH study, 15% of youth with type 1 diabetes had high triglyceride levels and 10% had low HDL-cholesterol levels. Among those with type 2 diabetes, 65% had elevated triglyceride levels, 60% had low HDL-cholesterol levels and 35% had elevated apolipoprotein levels [68]. Alarmingly, in the TODAY study, the 15 year cumulative incidence of dyslipidaemia and hypertension was 51.6% and 67.5%, respectively [70].

Of note, a sexual dimorphism exists for diabetes-associated cardiometabolic risk factors and complications, which tend to be more common in girls than in boys [63, 69]. During adolescence, girls experience more severe insulin resistance, higher HbA_{1c} levels and BMI and a greater prevalence of dyslipidaemia than boys [71, 72], which can contribute to their higher risk of complications [66, 73]. Adolescent girls with albuminuria also show higher testosterone levels, a higher free androgen index and reduced sex hormone-binding globulin levels than those without this complication, suggesting that increased androgen production may contribute to the early signs of vascular complications in girls [73]. Lower levels of IGF-1 in girls with type 1 diabetes have also been associated with the development of albuminuria [73].

The prevalence of vascular complications is higher in youth with type 1 diabetes from LMICs than those from HICs. In a cross-sectional study from Tanzania, 41.9% of youth aged 5–19 years had at least one microvascular complication. Of those presenting with complications, 26% had two complications and 4.6% had three [74]. In another Tanzanian cohort, early signs of diabetic retinopathy and kidney disease were seen even in children aged 5–10 years after a short duration of diabetes [75]. Similar data have been reported from Sudan, where a cross-sectional study found that 58% of youth aged 10–18 years had diabetic kidney disease, retinopathy or both after a median diabetes duration of 7 years [76]. A cohort study in Ethiopia found that 25% of children had at least one microvascular complication within 5 years of type 1 diabetes diagnosis, and 75% had at least one complication by 8 years [77]. Finally, according to 2014 data from India, regardless of the type of diabetes, half of the study population aged 10–25 years developed diabetic retinopathy within 10–12 years of diabetes diagnosis [78].

Ethnic disparities play a significant role in influencing the risk of complications, with non-Hispanic Black individuals with type 1 diabetes having a 4.5 fold increased risk of above-target HbA_{1c} and a twofold increase in systolic blood pressure compared with their non-Hispanic White counterparts [79]. These ethnic differences persist even after adjusting for age and socioeconomic status, suggesting that other underlying mechanisms, such as genetic predisposition, cultural lifestyle differences, healthcare access, diet, physical activity levels and psychosocial stressors, might play a role [80].

Social determinants of health affect diabetes and its related outcomes [81, 82]. Factors such as low socioeconomic status, food insecurity, unstable housing, transport challenges, limited access to healthcare and social isolation have all been found to negatively impact overall health and diabetes-related outcomes [82]. Higher rates of hyperglycaemia, albuminuria, dyslipidaemia and hypertension have been associated with deprivation in youth, irrespective of diabetes type [83, 84].

Risk of mortality in youth with diabetes An earlier age at diagnosis of type 2 diabetes is associated with higher all-cause mortality [63]. A meta-analysis of around 1.3 million youth diagnosed with type 2 diabetes reported a 4% decreased risk of all-cause mortality for each 1 year increase in age at diagnosis after controlling for current age [61]. In addition, the SEARCH study reported 1.5 times higher standardised mortality ratios for youth-onset type 2 diabetes compared with type 1 diabetes (2.3 vs 1.5) [85]. The same study also highlighted that excess mortality was highest among racial and ethnic minority groups and those younger than 25 years at the time of death. Of note, only 9.1% of deaths in youth with type 2 diabetes had diabetes-related factors as the underlying cause, with external factors (injury, assault, motor vehicle accident) being the most common cause of death.

According to GBD 2019 data, type 1 diabetes-related mortality rates increased between 1990 and 1999, after which a steady decline was noticed. Countries with the highest SDI have the lowest mortality rates (0.05 per 100,000), whereas those with the lowest SDI show the highest mortality rates (0.5 per 100,000) [42, 86]. Globally, there is a huge gap in remaining life expectancy (40 vs 64 years) between newly diagnosed 10-year-old children with type 1 diabetes and their peers without type 1 diabetes. This gap ranges from 46 years in low-income countries (LICs) to 36 years in LMICs and 11 years in HICs [45]. The marked reduction in life expectancy in LICs and LMICs is related to higher

mortality rates resulting from acute and chronic complications such as diabetic ketoacidosis, hypoglycaemia, infections and end-stage renal failure. These outcomes reflect differences in healthcare provision, health infrastructure and accessibility to affordable diabetes supplies such as insulin, glucose monitoring systems and diabetes education between LICs/LMICs and HICs. When the governments of LICs and LMICs are unable to provide comprehensive care, studies have shown that scaling up 'minimal' diabetes care to even 'intermediate'-level care (basal–bolus regimen, two to three blood glucose tests per day, regular HbA_{1c} testing, complications screening, age-appropriate diabetes education, peer and school support and 24 h emergency call support) can lead to significant reductions in complications and mortality rates and is very cost-effective [87, 88].

How to decrease the burden of early-onset metabolic diseases

The increasing incidence and severity of youth-onset obesity and type 1 and type 2 diabetes raises concerns about the growing number of individuals at risk of developing cardiometabolic complications throughout their lifespan and experiencing disability and reduced life expectancy [3]. Addressing this issue requires a multifaceted approach that includes early interventions, improved healthcare policies, enhanced treatment strategies and addressing disparities in healthcare access (Fig. 4).

To control the spread of metabolic diseases in youth worldwide, interventions should begin early, starting from pre-conception and continuing through infancy, childhood and adolescence [89]. Preventive measures should focus on modifiable risk factors, including diet, physical activity levels, sleep and stress management [90]. A multidisciplinary approach involving lifestyle and behavioural interventions is essential to instil healthy habits early, supporting long-term health and reducing the risk of chronic diseases later in life [91].

Health policies are essential for preventing early-onset metabolic disorders, fostering a healthier environment through measures such as regulating food marketing, enhancing nutritional labelling and promoting healthy eating. A notable example is the increased taxation on sugar-sweetened beverages, which has proven effective in reducing obesity rates [92]. Policies should also encourage active school environments and support community-based initiatives that aim to make physical activity accessible and enjoyable for all children [93]. Raising professional and public awareness of metabolic diseases and their health implications is another critical step in preventing the escalation of this problem [94].

Managing youth-onset metabolic disorders and mitigating the associated complications also requires the development of more effective treatments as well as strategies to

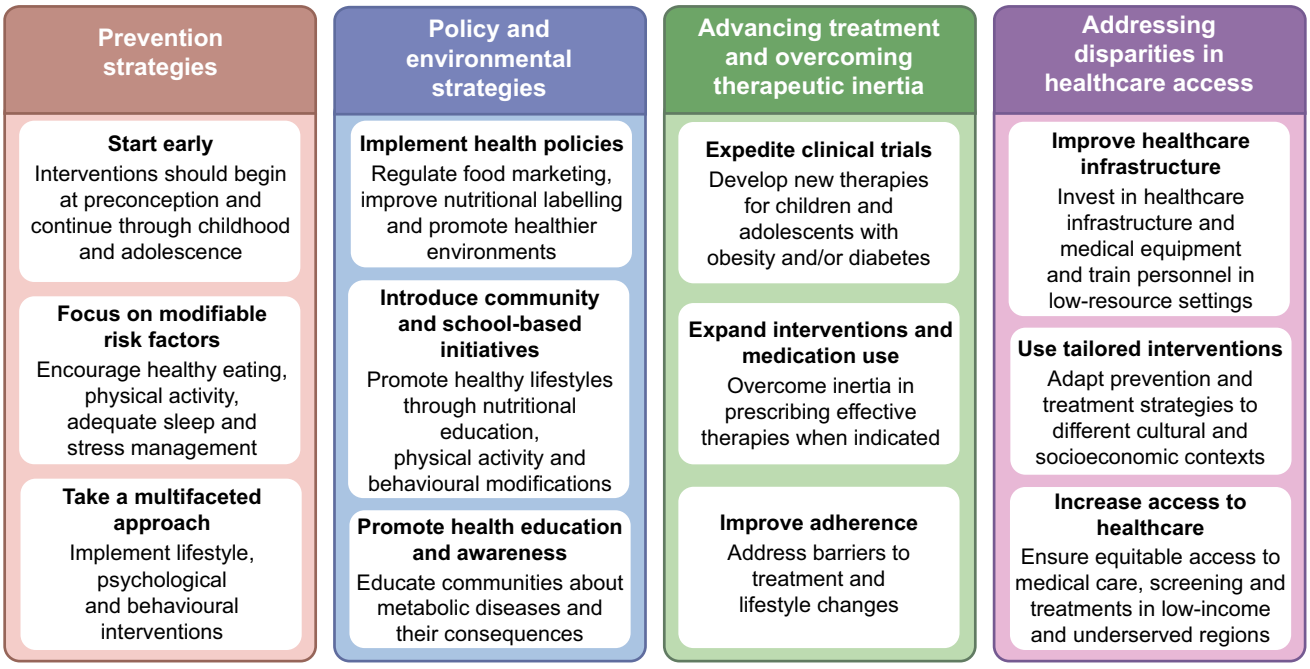

Fig. 4 Strategies to reduce the burden of youth-onset metabolic diseases. This figure is available as part of a downloadable slideset

overcome barriers to adherence, which is suboptimal among youth [95]. Therapeutic inertia—the reluctance to initiate or escalate necessary treatments—should also be addressed. Many healthcare professionals hesitate to prescribe medications such as metformin, glucagon-like peptide-1 (GLP-1) receptor agonists, antihypertensives and lipid-lowering drugs due to concerns over limited long-term safety data, lack of clear guidelines, inadequate training and resource constraints [96]. Expediting clinical trials of new interventions in youth with obesity and/or diabetes is crucial, particularly given the success of therapies such as sodium–glucose cotransporter 2 inhibitors and GLP-1 receptor agonists in adults. Expanding treatment options for children and adolescents is critical, as current strategies are often inadequate.

Further understanding of ethnic and socioeconomic disparities is also crucial to develop targeted interventions that address the unique needs of diverse populations. Because of the differences in attributable risk factors for early-onset metabolic diseases across regions and countries [33], specific policies should be established to manage this epidemic more effectively.

Addressing inequalities in access to healthcare is vital, as the burden of metabolic diseases remains disproportionately high in LMICs [97]. This disparity is partly caused by delayed diagnosis of metabolic diseases and related complications, due to limited and inadequate infrastructure and diagnostic capabilities [97]. In addition, treatment strategies for obesity and diabetes available in HICs are often unaffordable in LMICs. Alarmingly, access to insulin remains limited in some LMICs, and diabetes technologies are largely inaccessible in low-resource settings due to their cost [97]. Therefore, clear strategies and increased investment are needed to improve diagnostics and access to insulin and other essential treatments for young people living in LMICs.

Conclusions

The increasing rates of metabolic diseases among young people, along with regional, ethnic and socioeconomic disparities, raise significant concerns about their short- and long-term health impacts. Early targeted interventions are crucial to prevent the onset and progression of these diseases.

Tackling youth-onset metabolic diseases requires a global, multidisciplinary and policy-driven approach. From early prevention to equitable healthcare access, comprehensive strategies must be implemented to reduce the burden of these conditions and improve long-term health outcomes. Additionally, tailored preventive and treatment strategies that consider cultural contexts, socioeconomic backgrounds and available resources are essential to effectively manage these diseases across diverse settings.

Supplementary Information The online version contains a slide-set of figures for download available at https://doi.org/10.1007/s00125-025-06450-2.

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References

1. Chong B, Kong G, Shankar K et al (2023) The global syndemic of metabolic diseases in the young adult population: a consortium of trends and projections from the Global Burden of Disease 2000–2019. Metabolism 141:155402. https://doi.org/10.1016/j.metabol.2023.155402
2. Drozdz D, Alvarez-Pitti J, Wójcik M et al (2021) Obesity and cardiometabolic risk factors: from childhood to adulthood. Nutrients 13(11):4176. https://doi.org/10.3390/nu13114176
3. Zhou XD, Chen QF, Targher G et al (2024) Global burden of disease attributable to metabolic risk factors in adolescents and young adults aged 15–39, 1990–2021. Clin Nutr 43(12):391–404. https://doi.org/10.1016/j.clnu.2024.11.016
4. Walker SE, Gurka MJ, Oliver MN, Johns DW, DeBoer MD (2012) Racial/ethnic discrepancies in the metabolic syndrome begin in childhood and persist after adjustment for environmental factors. Nutr Metab Cardiovasc Dis 22(2):141–148. https://doi.org/10.1016/j.numecd.2010.05.006
5. Robertson CC, Inshaw JRJ, Onengut-Gumuscu S et al (2021) Fine-mapping, trans-ancestral and genomic analyses identify causal variants, cells, genes and drug targets for type 1 diabetes. Nat Genet 53(7):962–971. https://doi.org/10.1038/s41588-021-00880-5
6. Sawyer SM, Azzopardi PS, Wickremarathne D, Patton GC (2018) The age of adolescence. Lancet Child Adolesc Health 2(3):223–228. https://doi.org/10.1016/s2352-4642(18)30022-1
7. Clark H, Coll-Seck AM, Banerjee A et al (2020) A future for the world's children? A WHO-UNICEF-Lancet Commission. Lancet

395(10224):605–658. https://doi.org/10.1016/s0140-6736(19)32540-1
8. Hannon T, Janosky J, Arslanian S (2007) Longitudinal study of physiologic insulin resistance and metabolic changes of puberty. Pediatr Res 60:759–763. https://doi.org/10.1203/01.pdr.0000246097.73031.27
9. Reinehr T, Wolters B, Knop C, Lass N, Holl RW (2015) Strong effect of pubertal status on metabolic health in obese children: a longitudinal study. J Clin Endocrinol Metab 100(1):301–308. https://doi.org/10.1210/jc.2014-2674
10. Quattrin T, Mastrandrea LD, Walker LSK (2023) Type 1 diabetes. Lancet 401(10394):2149–2162. https://doi.org/10.1016/s0140-6736(23)00223-4
11. Marcovecchio ML, Dalton RN, Daneman D et al (2019) A new strategy for vascular complications in young people with type 1 diabetes mellitus. Nat Rev Endocrinol 15(7):429–435. https://doi.org/10.1038/s41574-019-0198-2
12. Gregory JW, Cameron FJ, Joshi K et al (2022) ISPAD clinical practice consensus guidelines 2022: diabetes in adolescence. Pediatr Diabetes 23(7):857–871. https://doi.org/10.1111/pedi.13408
13. Zarei I, Eloranta AM, Klåvus A et al (2024) Eight-year diet and physical activity intervention affects serum metabolites during childhood and adolescence: a nonrandomized controlled trial. iScience 27(7):110295. https://doi.org/10.1016/j.isci.2024.110295
14. WHO (2022) Obesity and overweight. Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. Accessed 21 Apr 2025
15. Zhang X, Liu J, Ni Y et al (2024) Global prevalence of overweight and obesity in children and adolescents: a systematic review and meta-analysis. JAMA Pediatrics 178(8):800–813. https://doi.org/10.1001/jamapediatrics.2024.1576
16. Pillon NJ, Loos RJF, Marshall SM, Zierath JR (2021) Metabolic consequences of obesity and type 2 diabetes: balancing genes and environment for personalized care. Cell 184(6):1530–1544. https://doi.org/10.1016/j.cell.2021.02.012
17. Littleton SH, Berkowitz RI, Grant SFA (2020) Genetic determinants of childhood obesity. Mol Diagn Ther 24(6):653–663. https://doi.org/10.1007/s40291-020-00496-1
18. Quarta A, Quarta MT, Mastromauro C, Chiarelli F, Giannini C (2024) Influence of nutrition on growth and development of metabolic syndrome in children. Nutrients 16(22):3801. https://doi.org/10.3390/nu16223801
19. Voerman E, Santos S, PatroGolab B et al (2019) Maternal body mass index, gestational weight gain, and the risk of overweight and obesity across childhood: an individual participant data meta-analysis. PLoS Med 16(2):e1002744. https://doi.org/10.1371/journal.pmed.1002744
20. Seneviratne SN, Rajindrajith S (2022) Fetal programming of obesity and type 2 diabetes. World J Diabetes 13(7):482–497. https://doi.org/10.4239/wjd.v13.i7.482
21. NCD Risk Factor Collaboration (NCD-RisC) (2019) Rising rural body-mass index is the main driver of the global obesity epidemic in adults. Nature 569(7755):260–264. https://doi.org/10.1038/s41586-019-1171-x
22. Islam MR, Trenholm J, Rahman A, Pervin J, Ekström EC, Rahman SM (2019) Sociocultural influences on dietary practices and physical activity behaviors of rural adolescents-a qualitative exploration. Nutrients 11(12):2916. https://doi.org/10.3390/nu11122916
23. Kim H, Rajbhandari A, Krile R, Lang IM, Antonakos CL, Colabianchi N (2024) Body mass index trajectories among the healthy communities study children: racial/ethnic and socioeconomic disparities in childhood obesity. J Racial Ethn Health Disparities 11(1):203–215. https://doi.org/10.1007/s40615-023-01511-x
24. Polidori N, Mainieri F, Chiarelli F, Mohn A, Giannini C (2022) Early insulin resistance, type 2 diabetes, and treatment options in childhood. Horm Res Paediatr 95(2):149–166. https://doi.org/10.1159/000521515
25. Chinali M, de Simone G, Roman MJ et al (2006) Impact of obesity on cardiac geometry and function in a population of adolescents: the Strong Heart Study. J Am Coll Cardiol 47(11):2267–2273. https://doi.org/10.1016/j.jacc.2006.03.004
26. Zong X, Kelishadi R, Kim HS et al (2024) A proposed simplified definition of metabolic syndrome in children and adolescents: a global perspective. BMC Med 22(1):190. https://doi.org/10.1186/s12916-024-03406-y
27. Wentzel A, Mabhida SE, Ndlovu M et al (2025) Prevalence of metabolic syndrome in children and adolescents with obesity: a systematic review and meta-analysis. Obesity (Silver Spring) 33(1):12–32. https://doi.org/10.1002/oby.24159
28. Bitew ZW, Alemu A, Ayele EG, Tenaw Z, Alebel A, Worku T (2020) Metabolic syndrome among children and adolescents in low and middle income countries: a systematic review and meta-analysis. Diabetol Metab Syndr 12:93. https://doi.org/10.1186/s13098-020-00601-8
29. Alberti KG, Zimmet P, Shaw J (2005) The metabolic syndrome–a new worldwide definition. Lancet 366(9491):1059–1062. https://doi.org/10.1016/s0140-6736(05)67402-8
30. Magge SN, Goodman E, Armstrong SC (2017) The metabolic syndrome in children and adolescents: shifting the focus to cardiometabolic risk factor clustering. Pediatrics 140(2):e20171603. https://doi.org/10.1542/peds.2017-1603
31. Mayer-Davis EJ, Lawrence JM, Dabelea D et al (2017) Incidence trends of type 1 and type 2 diabetes among youths, 2002–2012. N Engl J Med 376(15):1419–1429. https://doi.org/10.1056/NEJMoa1610187
32. Wagenknecht LE, Lawrence JM, Isom S et al (2023) Trends in incidence of youth-onset type 1 and type 2 diabetes in the USA, 2002–18: results from the population-based SEARCH for Diabetes in Youth study. Lancet Diabetes Endocrinol 11(4):242–250. https://doi.org/10.1016/s2213-8587(23)00025-6
33. Xie J, Wang M, Long Z et al (2022) Global burden of type 2 diabetes in adolescents and young adults, 1990–2019: systematic analysis of the Global Burden of Disease Study 2019. BMJ 379:e072385. https://doi.org/10.1136/bmj-2022-072385
34. Gesuita R, Eckert AJ, Besançon S et al (2025) Frequency and clinical characteristics of children and young people with type 2 diabetes at diagnosis from five world regions between 2012 and 2021: data from the SWEET Registry. Diabetologia 68(1):82–93. https://doi.org/10.1007/s00125-024-06283-5
35. Shah AS, Barrientos-Pérez M, Chang N et al (2024) ISPAD clinical practice consensus guidelines 2024: type 2 diabetes in children and adolescents. Horm Res Paediatr 97(6):542–570. https://doi.org/10.1159/000543033
36. Cioana M, Deng J, Nadarajah A et al (2022) The prevalence of obesity among children with type 2 diabetes: a systematic review and meta-analysis. JAMA Network Open 5(12):e2247186–e2247186. https://doi.org/10.1001/jamanetworkopen.2022.47186
37. Bacha F, Cheng P, Gal RL et al (2021) Racial and ethnic disparities in comorbidities in youth with type 2 diabetes in the Pediatric Diabetes Consortium (PDC). Diabetes Care 44(10):2245–2251. https://doi.org/10.2337/dc21-0143
38. Butler AM (2017) Social determinants of health and racial/ethnic disparities in type 2 diabetes in youth. Curr Diab Rep 17(8):60. https://doi.org/10.1007/s11892-017-0885-0
39. IDF (2021) IDF diabetes atlas, 10th edn. IDF, Brussels, Belgium. Available from: http://www.diabetesatlas.org. Accessed 2 Apr 2025
40. Zeitler P, Hirst K, Pyle L et al (2012) A clinical trial to maintain glycemic control in youth with type 2 diabetes. N Engl J Med 366(24):2247–2256. https://doi.org/10.1056/NEJMoa1109333

41. McGavock J, Wicklow B, Dart AB (2017) Type 2 diabetes in youth is a disease of poverty. Lancet 390(10105):1829. https://doi.org/10.1016/s0140-6736(17)32461-3
42. Gong B, Yang W, Xing Y, Lai Y, Shan Z (2024) Global, regional, and national burden of type 1 diabetes in adolescents and young adults. Pediatr Res. https://doi.org/10.1038/s41390-024-03107-5
43. IDF (2025) IDF diabetes atlas, 11th edn. IDF, Brussels, Belgium. Available from: http://www.diabetesatlas.org. Accessed 21 Apr 2025
44. Hormazábal-Aguayo I, Ezzatvar Y, Huerta-Uribe N, Ramírez-Vélez R, Izquierdo M, García-Hermoso A (2024) Incidence of type 1 diabetes mellitus in children and adolescents under 20 years of age across 55 countries from 2000 to 2022: a systematic review with meta-analysis. Diabetes Metab Res Rev 40(3):e3749. https://doi.org/10.1002/dmrr.3749
45. Gregory GA, Robinson TIG, Linklater SE et al (2022) Global incidence, prevalence, and mortality of type 1 diabetes in 2021 with projection to 2040: a modelling study. Lancet Diabetes Endocrinol 10(10):741–760. https://doi.org/10.1016/s2213-8587(22)00218-2
46. Tönnies T, Brinks R, Isom S et al (2023) Projections of type 1 and type 2 diabetes burden in the U.S. population aged <20 years through 2060: the SEARCH for diabetes in youth study. Diabetes Care 46(2):313–320. https://doi.org/10.2337/dc22-0945
47. Mittal R, Camick N, Lemos JRN, Hirani K (2024) Gene-environment interaction in the pathophysiology of type 1 diabetes. Front Endocrinol 15:1335435. https://doi.org/10.3389/fendo.2024.1335435
48. Redondo MJ, Gignoux CR, Dabelea D et al (2022) Type 1 diabetes in diverse ancestries and the use of genetic risk scores. Lancet Diabetes Endocrinol 10(8):597–608. https://doi.org/10.1016/s2213-8587(22)00159-0
49. Imagawa A, Hanafusa T, Uchigata Y et al (2005) Different contribution of class II HLA in fulminant and typical autoimmune type 1 diabetes mellitus. Diabetologia 48(2):294–300. https://doi.org/10.1007/s00125-004-1626-x
50. Park Y, Eisenbarth GS (2001) Genetic susceptibility factors of type 1 diabetes in Asians. Diabetes Metab Res Rev 17(1):2–11. https://doi.org/10.1002/1520-7560(2000)9999:9999<::aid-dmrr164>3.0.co;2-m
51. Borchers AT, Uibo R, Gershwin ME (2010) The geoepidemiology of type 1 diabetes. Autoimmun Rev 9(5):A355-365. https://doi.org/10.1016/j.autrev.2009.12.003
52. Rytkönen M, Ranta J, Tuomilehto J, Karvonen M (2001) Bayesian analysis of geographical variation in the incidence of Type I diabetes in Finland. Diabetologia 44(Suppl 3):B37-44. https://doi.org/10.1007/pl00002952
53. March CA, Becker DJ, Libman IM (2021) Nutrition and obesity in the pathogenesis of youth-onset type 1 diabetes and its complications. Front Endocrinol (Lausanne) 12:622901. https://doi.org/10.3389/fendo.2021.622901
54. Nitecki M, Gerstein HC, Balmakov Y et al (2023) High BMI and the risk for incident type 1 Diabetes Mellitus: a systematic review and meta-analysis of aggregated cohort studies. Cardiovasc Diabetol 22(1):300. https://doi.org/10.1186/s12933-023-02007-y
55. Buzzetti R, Zampetti S, Pozzilli P (2020) Impact of obesity on the increasing incidence of type 1 diabetes. Diabetes Obes Metab 22(7):1009–1013. https://doi.org/10.1111/dom.14022
56. Agarwal S, Kanapka LG, Raymond JK et al (2020) Racial-ethnic inequity in young adults with type 1 diabetes. J Clin Endocrinol Metab 105(8):e2960-2969. https://doi.org/10.1210/clinem/dgaa236
57. Burckhardt MA, Addala A, de Bock M (2024) Editorial: equity in type 1 diabetes technology and beyond: where are we in 2022? Front Endocrinol (Lausanne) 15:1400240. https://doi.org/10.3389/fendo.2024.1400240
58. Fang M, Wang D, Selvin E (2024) Prevalence of type 1 diabetes among US children and adults by age, sex, race, and ethnicity. JAMA 331(16):1411–1413. https://doi.org/10.1001/jama.2024.2103
59. Rawshani A, Sattar N, Franzén S et al (2018) Excess mortality and cardiovascular disease in young adults with type 1 diabetes in relation to age at onset: a nationwide, register-based cohort study. Lancet 392(10146):477–486. https://doi.org/10.1016/s0140-6736(18)31506-x
60. Huo L, Magliano DJ, Rancière F et al (2018) Impact of age at diagnosis and duration of type 2 diabetes on mortality in Australia 1997–2011. Diabetologia 61(5):1055–1063. https://doi.org/10.1007/s00125-018-4544-z
61. Nanayakkara N, Curtis AJ, Heritier S et al (2021) Impact of age at type 2 diabetes mellitus diagnosis on mortality and vascular complications: systematic review and meta-analyses. Diabetologia 64(2):275–287. https://doi.org/10.1007/s00125-020-05319-w
62. Forbes JM, Fotheringham AK (2017) Vascular complications in diabetes: old messages, new thoughts. Diabetologia 60(11):2129–2138. https://doi.org/10.1007/s00125-017-4360-x
63. Bjornstad P, Chao LC, Cree-Green M et al (2023) Youth-onset type 2 diabetes mellitus: an urgent challenge. Nat Rev Nephrol 19(3):168–184. https://doi.org/10.1038/s41581-022-00645-1
64. Marcovecchio ML, Hendriks AEJ, Delfin C et al (2024) The INNODIA Type 1 Diabetes Natural History Study: a European cohort of newly diagnosed children, adolescents and adults. Diabetologia 67(6):995–1008. https://doi.org/10.1007/s00125-024-06124-5
65. Cho YH, Craig ME, Donaghue KC (2014) Puberty as an accelerator for diabetes complications. Pediatr Diabetes 15(1):18–26. https://doi.org/10.1111/pedi.12112
66. Tommerdahl KL, Shapiro ALB, Nehus EJ, Bjornstad P (2022) Early microvascular complications in type 1 and type 2 diabetes: recent developments and updates. Pediatr Nephrol 37(1):79–93. https://doi.org/10.1007/s00467-021-05050-7
67. Marcovecchio ML, Chiesa ST, Armitage J et al (2018) Renal and cardiovascular risk according to tertiles of urinary albumin-to-creatinine ratio: the Adolescent Type 1 Diabetes Cardio-Renal Intervention Trial (AdDIT). Diabetes Care 41(9):1963–1969. https://doi.org/10.2337/dc18-1125
68. Dabelea D, Stafford JM, Mayer-Davis EJ et al (2017) Association of type 1 diabetes vs type 2 diabetes diagnosed during childhood and adolescence with complications during teenage years and young adulthood. JAMA 317(8):825–835. https://doi.org/10.1001/jama.2017.0686
69. Bjornstad P, Donaghue KC, Maahs DM (2018) Macrovascular disease and risk factors in youth with type 1 diabetes: time to be more attentive to treatment? Lancet Diabetes Endocrinol 6(10):809–820. https://doi.org/10.1016/s2213-8587(18)30035-4
70. Bjornstad P, Drews KL, Caprio S et al (2021) Long-term complications in youth-onset type 2 diabetes. N Engl J Med 385(5):416–426. https://doi.org/10.1056/NEJMoa2100165
71. Schwab KO, Doerfer J, Hecker W et al (2006) Spectrum and prevalence of atherogenic risk factors in 27,358 children, adolescents, and young adults with type 1 diabetes: cross-sectional data from the German diabetes documentation and quality management system (DPV). Diabetes Care 29(2):218–225. https://doi.org/10.2337/diacare.29.02.06.dc05-0724
72. Arnqvist HJ, Ludvigsson J, Nordwall M (2024) Early increase in HbA1c trajectory predicts development of severe microangiopathy in patients with type 1 diabetes: the VISS study. BMJ Open Diabetes Res Care 12(3):e003917. https://doi.org/10.1136/bmjdrc-2023-003917
73. Amin R, Schultz C, Ong K et al (2003) Low IGF-I and elevated testosterone during puberty in subjects with type 1 diabetes developing microalbuminuria in comparison to normoalbuminuric

control subjects: the Oxford Regional Prospective Study. Diabetes Care 26(5):1456–1461. https://doi.org/10.2337/diacare.26.5.1456
74. Msanga D, Reis K, Kayange N et al (2020) Diabetic microvascular complications among children and adolescents in northwestern Tanzania: a cross-sectional study. Ann Glob Health 86(1):43. https://doi.org/10.5334/aogh.2669
75. Najem S, Majaliwa ES, Ramaiya K, Swai ABM, Jasem D, Ludvigsson J (2021) Glycemic control and complications of type 1 diabetes among children in Tanzania. J Clin Transl Endocrinol 23:100245. https://doi.org/10.1016/j.jcte.2020.100245
76. Ahmed H, Elshaikh T, Abdullah M (2020) Early diabetic nephropathy and retinopathy in patients with type 1 diabetes mellitus attending Sudan childhood diabetes centre. J Diabetes Res 2020:7181383. https://doi.org/10.1155/2020/7181383
77. Eshetu K, Regassa LD, Dehresa M, Genete D (2024) Chronic microvascular complication of type 1 diabetes mellitus and its predictors among children with type 1 diabetes mellitus in Ethiopia; a single center experience: ambi directional cohort study. Pediatric Health Med Ther 15:201–212. https://doi.org/10.2147/phmt.S456541
78. Rajalakshmi R, Amutha A, Ranjani H et al (2014) Prevalence and risk factors for diabetic retinopathy in Asian Indians with young onset type 1 and type 2 diabetes. J Diabetes Complications 28(3):291–297. https://doi.org/10.1016/j.jdiacomp.2013.12.008
79. Griggs S, Blanchette JE, Hickman RL Jr et al (2022) Racial and ethnic cardiometabolic risk disparities in the type 1 diabetes exchange clinic registry cohort. Endocr Pract 28(12):1237–1243. https://doi.org/10.1016/j.eprac.2022.10.003
80. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC (1999) Ethnic variation in cardiovascular disease risk factors among children and young adults: findings from the Third National Health and Nutrition Examination Survey, 1988–1994. JAMA 281(11):1006–1013. https://doi.org/10.1001/jama.281.11.1006
81. Golden SH, Brown A, Cauley JA et al (2012) Health disparities in endocrine disorders: biological, clinical, and nonclinical factors–an Endocrine Society scientific statement. J Clin Endocrinol Metab 97(9):E1579-1639. https://doi.org/10.1210/jc.2012-2043
82. Hill-Briggs F, Adler NE, Berkowitz SA et al (2020) Social determinants of health and diabetes: a scientific review. Diabetes Care 44(1):258–279. https://doi.org/10.2337/dci20-0053
83. Wijayaratna S, Lee A, Park HY et al (2021) Socioeconomic status and risk factors for complications in young people with type 1 or type 2 diabetes: a cross-sectional study. BMJ Open Diabetes Res Care 9(2):e002485. https://doi.org/10.1136/bmjdrc-2021-002485
84. Griggs S, Al-Kindi S, Hardin H et al (2023) Socioeconomic deprivation and cardiometabolic risk factors in individuals with type 1 diabetes: T1D exchange clinic registry. Diabetes Res Clin Pract 195:110198. https://doi.org/10.1016/j.diabres.2022.110198
85. Lawrence JM, Reynolds K, Saydah SH et al (2021) Demographic correlates of short-term mortality among youth and young adults with youth-onset diabetes diagnosed from 2002 to 2015: the SEARCH for Diabetes in Youth Study. Diabetes Care 44(12):2691–2698. https://doi.org/10.2337/dc21-0728
86. Zhang K, Kan C, Han F et al (2023) Global, regional, and national epidemiology of diabetes in children from 1990 to 2019. JAMA Pediatr 177(8):837–846. https://doi.org/10.1001/jamapediatrics.2023.2029
87. Ogle GD, von Oettingen JE, Middlehurst AC, Hanas R, Orchard TJ (2019) Levels of type 1 diabetes care in children and adolescents for countries at varying resource levels. Pediatr Diabetes 20(1):93–98. https://doi.org/10.1111/pedi.12801
88. Gregory GA, Guo J, Klatman EL et al (2020) Costs and outcomes of "intermediate" vs "minimal" care for youth-onset type 1 diabetes in six countries. Pediatr Diabetes 21(4):628–636. https://doi.org/10.1111/pedi.12988
89. Jacob CM, Newell ML, Hanson M (2019) Narrative review of reviews of preconception interventions to prevent an increased risk of obesity and non-communicable diseases in children. Obes Rev 20(Suppl 1):5–17. https://doi.org/10.1111/obr.12769
90. American Diabetes Association Professional Practice Committee (2024) 5. Facilitating positive health behaviors and well-being to improve health outcomes: standards of care in diabetes—2025. Diabetes Care 48(Suppl 1):S86–S127. https://doi.org/10.2337/dc25-S005
91. Schnermann ME, Schulz CA, Herder C, Alexy U, Nöthlings U (2021) A lifestyle pattern during adolescence is associated with cardiovascular risk markers in young adults: results from the DONALD cohort study. J Nutr Sci 10:e92. https://doi.org/10.1017/jns.2021.84
92. Itria A, Borges SS, Rinaldi AEM, Nucci LB, Enes CC (2021) Taxing sugar-sweetened beverages as a policy to reduce overweight and obesity in countries of different income classifications: a systematic review. Public Health Nutr 24(16):5550–5560. https://doi.org/10.1017/s1368980021002901
93. Wyszyńska J, Ring-Dimitriou S, Thivel D et al (2020) Physical activity in the prevention of childhood obesity: the position of the European childhood obesity group and the European academy of pediatrics. Front Pediatr 8:535705. https://doi.org/10.3389/fped.2020.535705
94. GBD 2021 Risk Factors Collaborators (2024) Global burden and strength of evidence for 88 risk factors in 204 countries and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet 403(10440):2162–2203. https://doi.org/10.1016/s0140-6736(24)00933-4
95. Taddeo D, Egedy M, Frappier JY (2008) Adherence to treatment in adolescents. Paediatr Child Health 13(1):19–24. https://doi.org/10.1093/pch/13.1.19
96. Nambam B, DuBose SN, Nathan BM et al (2016) Therapeutic inertia: underdiagnosed and undertreated hypertension in children participating in the T1D Exchange Clinic Registry. Pediatr Diabetes 17(1):15–20. https://doi.org/10.1111/pedi.12231
97. Bhutta ZA, Salam RA, Gomber A et al (2021) A century past the discovery of insulin: global progress and challenges for type 1 diabetes among children and adolescents in low-income and middle-income countries. Lancet 398(10313):1837–1850. https://doi.org/10.1016/s0140-6736(21)02247-9