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2026 CONSENSUS GUIDELINES

Screening, assessment and management of type 2 diabetes in children and adolescents

Full Guidelines

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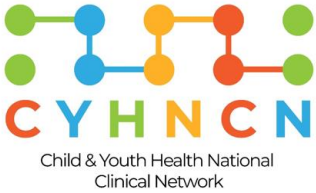
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Conflicts of interest

None declared

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Summary of what is new / different:

Updates since the 2020 Australian and New Zealand Society for Paediatric Endocrinology and Diabetes (ANZSPED) guidelines on this topic include:

- Guidance regarding intergenerational risk of type 2 diabetes (T2D)
- Guidance regarding impact of social determinants of health on T2D risk
- Guidance regarding differing phenotypes of youth-onset type 2 diabetes (YOT2D).
- Updated screening algorithm for youth at risk of T2D.
- Guidance regarding psychological health and wellbeing of youth living with T2D.
- Guidance regarding use of continuous glucose monitoring systems.
- Expanded guidance regarding engagement considerations for youth living with T2D.
- Updated algorithms and tables for treatment, glycaemic monitoring, management and assessment of comorbidities and complications.
- Recommendations regarding management strategies and recently approved pharmacological therapies for treatment of YOT2D.
- Stronger emphasis and guidance regarding management within primary health care.
- These updated guidelines represent for the first time consensus from a collaboration of paediatric, diabetes, primary health care, consumer and research organisations across Australia and Aotearoa New Zealand.

Introduction

The prevalence of type 2 diabetes (T2D) among children and adolescents (thereafter referred to as youth) is increasing worldwide[1, 2], including in Australia and Aotearoa New Zealand. The condition has been described internationally as a 'disease of poverty', disproportionately affecting youth from high risk ethnic groups and those who are socioeconomically marginalised[3, 4]. The prevalence of youth-onset T2D (YOT2D; defined as type 2 diabetes diagnosed before the age of 25 years) among Aboriginal and Torres Strait Islander youth is the highest reported globally in recent years[5], at 14.4 per 1000 (95% CI 12.2-17.0) in the Central Australia region. In Western Australia, the incidence of T2D in youth under the age of 16 increased by 5.2% from 2012 to 2019, and was 18.3 fold higher in Aboriginal and Torres Strait Islander youth than non-Indigenous youth[6]. A similar trend has been observed in Aotearoa New Zealand, where between 1995 and 2023, the incidence of YOT2D increased, particularly among Māori and Pacific Peoples populations[7]. In the Auckland region, the incidence of T2D in youth under 15 was 22.9 times higher in Pacific Peoples, and 21.9 times higher in Māori youth, compared to children of European descent[7]. Other high risk ethnicities include South-East Asian, South Asian (Indian subcontinent) and Middle Eastern populations across Australia and Aotearoa New Zealand[8].

YOT2D progresses more rapidly and has a worse prognosis than adult-onset T2D[4, 9], with distinct pathophysiological features, including accelerated β cell dysfunction[10, 11]. This 'severe aggressive phenotype'[4] is characterised by chronically sub-optimal glycaemia compared to adult cohorts, with only 14% of youth meeting targets[5] and 34% having glycated haemoglobin (HbA1c) >10% (86 mmol/mol) 15 years post-diagnosis[12]. Complications occur earlier and are more frequent in YOT2D compared to paediatric type 1 diabetes (T1D)[13]. The majority of affected youth with T2D develop diabetes-related complications by 15 years post-diagnosis[12], and Canadian data indicate that 50% will experience end-stage renal failure within 20 years of diagnosis[14]. YOT2D also exhibits a higher treatment failure rate compared to adult cohorts[4, 10] and is associated with poorer mental health outcomes[15]. Fifty eight percent of a diverse cohort of Australian youth with YOT2D had depressive symptoms[16] and 47% of First Nations Canadian youth with YOT2D have had a mental health-related hospital admission[17]. Canadian youth with YOT2D were 2.4 times more likely to have pre-existing mood or anxiety disorders than matched peers and 3.2 times more likely to attempt/complete suicide than matched peers[18].

The first Australian and Aotearoa New Zealand guidelines for the screening, assessment and management of T2D in children and adolescents were published by the Australian and New Zealand Society for Paediatric Endocrinology and Diabetes (ANZSPED) in 2020[19]. These new guidelines provide updated recommendations for clinical practice, reflecting expanding treatment options and the growing international evidence base[20, 21], and for the first time represents consensus from a collaboration of thirteen paediatric, diabetes, primary health care, consumer and research organisations across Australia and Aotearoa New Zealand.

While YOT2D refers to T2D diagnosed before the age of 25 years, these guidelines apply only to the paediatric population - youth aged less than 18 years and living with T2D in Australia and Aotearoa New Zealand, or who are at risk of developing T2D. We acknowledge that Australian guidelines already exist for youth aged 18-30 years living with T2D[22]. The guidelines exclude the management of acute complications of T2D such as diabetic ketoacidosis (DKA) and hyperglycaemic hyperosmolar state (HHS). The guidelines aim to support all health professionals caring for youth living with T2D, including primary health care services. The term 'First Nations' is used to respectfully refer to young people of Aboriginal, Torres Strait Islander, Māori and/or Pacific Peoples ethnicity. Please see Appendix Table 1 for suggested terminology.

Methods

In recognition of the increased incidence of T2D in youth and the risks that the condition poses, the ANZSPED Type 2 Diabetes Working Group sought to update guidelines for screening, assessment and management of T2D in youth and achieve consensus across professional societies and consumer organisations. The expert group was multidisciplinary and included individuals across Australia and Aotearoa New Zealand (Appendix Table 6). Representatives from the Australian Diabetes Society (ADS), Royal Australian College of General Practitioners (RACGP), Australian Diabetes Educators Association (ADEA), Child & Youth Health National Clinical Network for Children and Young People with Diabetes (CYHNCN), Royal New Zealand College of General Practitioners (RNZCGP), New Zealand Society for the Study of Diabetes (NZSSD), National Aboriginal Community Controlled Health Organisation (NACCHO), Diabetes Australia, Diabetes New Zealand, Menzies School of Health Research (Diabetes across the Lifecourse: Northern Australia Partnership), Murdoch Children's Research Institute, The Kids Research Institute Australia and ANZSPED were included in the working group and all organisations endorsed the final guidelines (Appendix). The working group included expert Aboriginal, Torres Strait Islander and Māori members, and wider feedback was sought regarding the draft guidelines from other First Nations community representatives and health professionals.

The members met and agreed upon the scope and structure of the guideline. Any conflicts of interest were declared. Members were allocated to subgroups and sections were drafted and then redistributed amongst the group for discussion. Recommendations are presented with National Health and Medical Research Council and Grading of Recommendations Assessment, Development and Evaluation (GRADE) levels of evidence (Appendix Table 2). Recommendations were derived through the best available research evidence, international guidelines and expert opinion where appropriate. For each section, searches were undertaken using multiple databases (see Appendix), with the final search completed on 31st March 2026.

Summary of recommendations

Table 1: Australian and Aotearoa New Zealand consensus guidelines for screening, assessment and management of youth aged <18 years living with T2D

Guideline subsections	Recommendations	GRADE category
Intergenerational risk	Note and counsel women with hyperglycaemia in pregnancy, especially women with T2D in pregnancy, of higher risk of youth-onset T2D in their offspring.	B
	Acknowledging that YOT2D often occurs in family contexts, with shared risk factors and lived experiences, consider the role of pre-conception culturally appropriate family education programs.	B
	Youth with T2D should receive pre-conception counselling regarding contraception and increased risk of congenital malformations linked to hyperglycaemia in early pregnancy.	A
Social determinants of health	Clinicians should follow person-centred care processes that address social context and cultural safety.	B
	Clinicians should explore financial stability, food and housing insecurity, parental employment and education status of the household, acknowledging these social determinants as possible markers of health disparity. Clinicians should consider these issues when formulating treatment plans.	B
Screening	Targeted screening should occur in youth aged ≥ 10 years (≥8 years in First Nations youth).	B
	Screening should occur in First Nations youth who have any additional risk factor. Screening should occur in youth of other ethnicities who have overweight/ obesity (BMI z score ≥ 1 and/or waist circumference to height ratio >0.5) <u>and</u> at least one other risk factor.	B
	Risk factors: • in-utero exposure to maternal diabetes, including gestational diabetes mellitus; • first degree relative with type 2 diabetes; • signs of insulin resistance (acanthosis nigricans); • other conditions associated with obesity and metabolic syndrome (ie, hypertension, dyslipidaemia, fatty liver disease, polyendocrine metabolic ovarian syndrome (PMOS), small for gestational age); • use of anti-psychotic medications; • other high risk ethnicity: South East Asian, Subcontinent, Middle Eastern	B
	HbA1c is recommended for screening, with use of validated point of care measure if possible to allow for immediate action.	B
Diagnosis	Diagnosis of T2D can be made using HbA1c, fasting glucose or 2-hour glucose level from oral glucose tolerance test.	A
	Pancreatic autoantibodies testing (glutamic acid decarboxylase, islet tyrosine phosphatase 2, zinc transporter 8, insulin autoantibodies) should be considered in all youth with clinical phenotype of T2D due to higher prevalence of T1D (cf T2D) in this age group.	B
	Genetic testing for monogenic diabetes should be considered in consultation with paediatric diabetes services.	B

Guideline subsections	Recommendations	GRADE category
	All youth with a new diagnosis of T2D should be discussed with paediatric/ youth diabetes services.	B
Phenotypes	Youth with atypical phenotypes should be discussed with a paediatric/ youth diabetes service to obtain diagnostic certainty. Clinicians should note in-utero exposures and family history of diabetes as part of phenotyping.	B
Diabetes education	All youth living with T2D need comprehensive and specific diabetes self-management education that is family (whānau/ aiga) centred, individualised, developmentally appropriate, culturally safe and acknowledges the social determinants of health.	B
	Education should be delivered by a specialised multidisciplinary team with expertise in managing youth-onset T2D.	C
Glycaemic monitoring	Individualise frequency and method of blood glucose level monitoring, and suggest use of continuous glucose monitoring systems, especially at diagnosis, when changing treatment, if on insulin, or if HbA1c is above target.	C
	Aim for blood glucose levels 4-6 mmol/L (fasting) and 4-8 mmol/L (2 hours post-prandial).	C
	Target HbA1c should be ≤6.5% (48mmol/mol) given the aggressive trajectory and high prevalence of cardiometabolic complications.	B
	Reassess HbA1c every 3 months.	D
Psychological health	Clinicians should explicitly address diabetes distress and actively screen for psychological comorbidities.	B
	Diabetes services should explore eating behaviours, also considering food security and social determinants of health.	C
	Diabetes services should engage mental health and culturally embedded supports early in management.	B
Engagement	Diabetes management should consider the complex interplay of factors influencing engagement and health-related behaviours (see Figure 3).	B
	Clinicians should avoid stigmatising language and refer to Diabetes Australia guidelines regarding recommended terminology.	C
	Identify the priorities and needs of the youth and individualise management, ensuring cultural and peer supports.	C
	Address stigma, shame and isolation experienced by youth living with T2D, using a strengths-based and community led approach.	C
	Ensure strong linkages and collaboration between primary care and paediatric/ youth diabetes services.	C
Nutrition and healthy lifestyle	Weight management and multidisciplinary, holistic approach to lifestyle modification is required at diagnosis and ongoing.	B
	Aim for healthy eating: eliminate sugar-sweetened beverages, reduce caloric dense and nutrient-poor foods; provide education around carbohydrates; address food insecurity; ensure adequate intake of nutrient-rich and low glycaemic index foods.	B
	Reduce total energy intake to achieve ≥7% decrease in total weight if weight is above target.	B
	Aim for at least 60 min/day of moderate to vigorous physical activity to improve body composition, glycaemia and insulin sensitivity.	B

Guideline subsections	Recommendations	GRADE category
	Exercise programs should include resistance activities to increase muscle mass, contributing to improved glycaemia.	B
	Recreational screen time should be limited to ≤ 2 hours a day.	B
	Encourage quality sleep of 8–11 hours duration according to age, with consistent bed and wake-up times and reduction of electronic media use in the evening.	C
Pharmacotherapy	Metformin XR up to 2g daily should be used as the first line medication if HbA1c ≤8.5% at diagnosis (metformin IR if XR unavailable).	A
	Insulin should be first line treatment for youth who present with one or more of the following: HbA1c >8.5% (69mmol/L), diabetic ketoacidosis, hyperglycaemic hyperosmolar state or ketosis, in combination with metformin.	B
	If HbA1c ≤6.5% is not achieved by 3 months post-diagnosis with metformin (with or without insulin), other medications such as GLP-1RA or SGLT2-I should be considered, given nature of emerging evidence for their use in children and adolescents, and also in consideration of need to target medication to presenting phenotype of T2D. These should be prescribed only under the guidance of a paediatric endocrinologist (or paediatrician with relevant expertise in diabetes).	B
Complications and comorbidities	Screen for complications and comorbidities at time of diagnosis of T2D.	A
Retinopathy	Assess retina using dilated pupil exam or retinal photography (preferred) by an optometrist or ophthalmologist annually. If abnormal, more frequent review as dictated by optometrist/ ophthalmologist.	A
Nephropathy	Assess early morning urine albumin to creatinine ratio annually, 6 monthly if abnormal.	A
	Consider angiotension converting enzyme inhibitors or angiotension receptor blockers if persistent albuminuria.	A
Peripheral neuropathy	Foot examination annually unless abnormal.	C
Overweight/ obesity	Optimise weight management as well as glycaemia to reduce risk of comorbidities and complications.	B
	Consider GLP-1RA for youth with T2D and severe obesity, and bariatric surgery for selected post-pubertal adolescents with T2D with severe obesity, taking into account special considerations in relation to consent, procedure, family support and availability and cost of bariatric services.	B
Psychosocial	Screening tools for psychosocial comorbidities and diabetes distress should be used regularly after diagnosis, at least annually or more frequently if clinical concern.	B
	Consider formalised screening for disordered eating behaviours.	D
Reproductive health	For female youth, a review of need for contraception should be done at every visit, especially if the HbA1c is above target or the patient is using teratogenic medications.	B
	For female youth, review risk for PMOS (menstrual cycle regularity, symptoms and signs of hyperandrogenism) at every visit.	B
Liver disease	Assess liver function tests (aspartate aminotransferase, alanine aminotransferase, gamma-glutamyl transferase) annually, 6 monthly if abnormal.	B

Guideline subsections	Recommendations	GRADE category
Obstructive sleep apnoea	Evaluate for symptoms of obstructive sleep apnoea in youth with obesity.	C
Hypertension	Assess blood pressure using appropriate cuff at every visit.	A
Lipids	Optimise lifestyle and consider angiotension converting enzyme inhibitors or angiotension receptor blockers if hypertension.	A
	Assess lipid profile at diagnosis and annually thereafter, 6 monthly if abnormal. Repeat lipids 3 months after diagnosis if initial dyslipidaemia.	B
	Optimise nutrition and consider statins if persistent high LDL, fish oil or fibrate if high triglycerides	C
Transition	Transition to adult endocrinologist and multidisciplinary team given the aggressive nature of YOT2D, acknowledging continuing need for critical collaboration with primary health care services and culturally safe care.	D
Standards of care	All services should audit the clinical care and outcomes of youth with T2D, preferably benchmarking to other services.	A

BMI= body mass index; GRADE = Grading of Recommendations Assessment; HbA1c = glycated haemoglobin; GLP-1RA = glucagon like peptide 1 receptor agonist; SGLT2-I = sodium glucose cotransporter 2 inhibitor

Intergenerational transmission of risk for youth-onset type 2 diabetes

YOT2D is associated with in-utero exposure to elevated glucose levels during pregnancy[23], which may compound genetic susceptibility to diabetes[24]. The combined exposure to maternal obesity and hyperglycaemia in-utero[25, 26] further elevates the risk, contributing to a transgenerational increase in cardiometabolic conditions at successively younger ages[25] (see Figure 1). Cohort studies from Australia and Canada indicate that offspring exposed to maternal T2D in-utero face greater risks of adverse outcomes than those exposed to gestational diabetes mellitus (GDM), likely due to sustained hyperglycaemia throughout pregnancy[27-29]. However, GDM exposure also increases offspring metabolic risk compared to pregnancies with normal glucose levels[30-32].

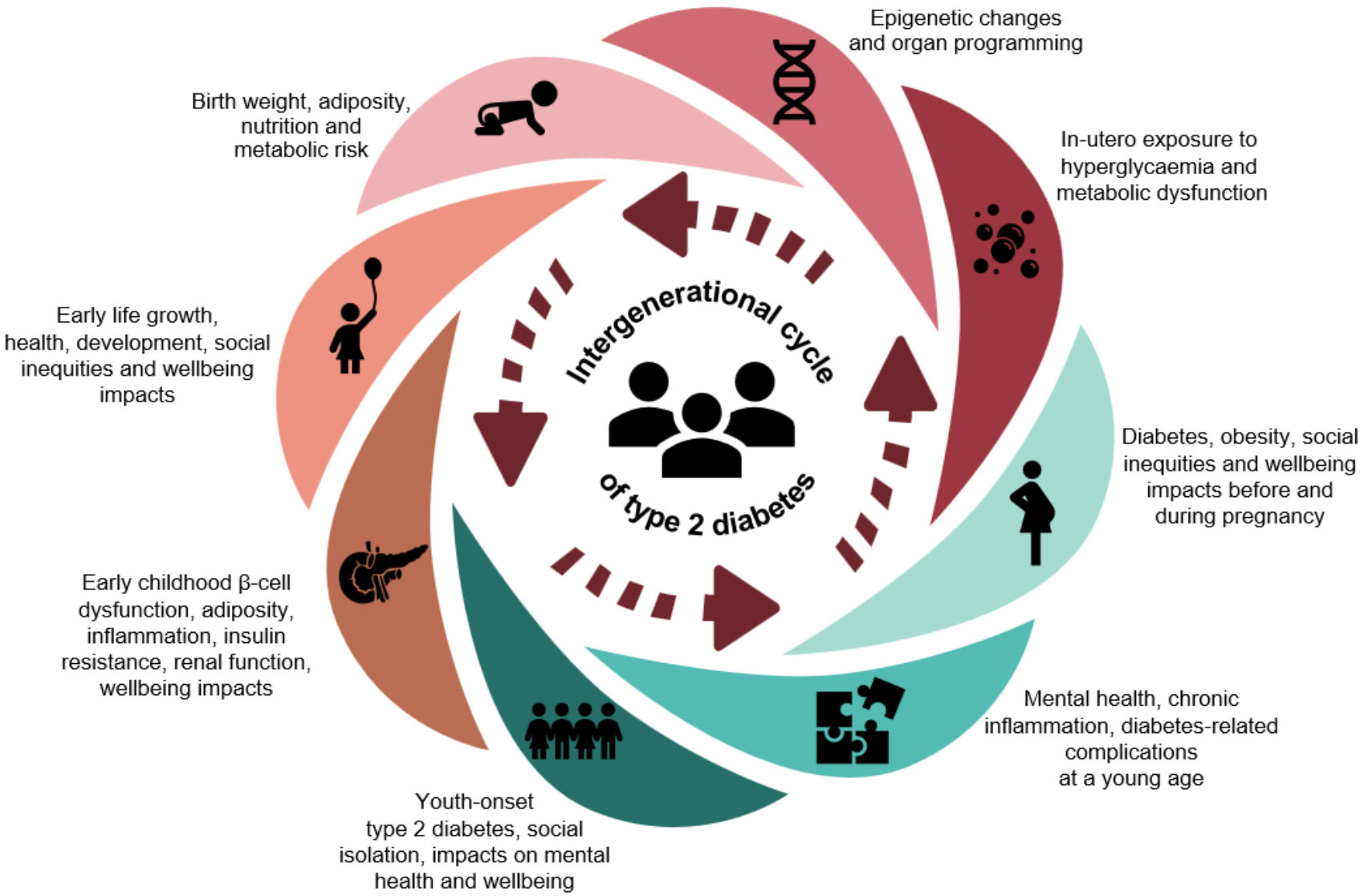
Genetic predisposition and environmental exposures, including in-utero hyperglycaemia, maternal smoking[33] and early nutrition (including early breastfeeding cessation) during critical developmental periods[34] may cause epigenetic changes and in-utero organ programming in offspring which may increase YOT2D risk[33 35, 36]. These changes, though not fully elucidated due to limited longitudinal data, likely underpin the intergenerational transmission of YOT2D. Most studies have been conducted in populations at high-risk for T2D, and the impact of recent societal changes and advances in management of maternal hyperglycaemia in pregnancy remains uncertain. Critical unknowns include timing of the vulnerable period of exposure to hyperglycaemia, the role of concurrent in-utero factors such as hyperinsulinaemia and dyslipidaemia, and the impact of maternal T2D treatment on offspring risk.

Children exposed to a double burden of both nutritional deficiency and dietary excess during pregnancy and their life course also have increased cardiometabolic risk compared to unexposed children. T2D may also occur at a lower BMI than unexposed peers. This phenotype, characterised by increased adiposity compared to lean mass, further exemplifies the risk linked to concurrent exposure to intrauterine hyperglycaemia and compounded by maternal malnutrition and nutrient imbalance[37-39]. Broader factors including maternal wellbeing and safety also influence child outcomes[40-42]. Diabetes counselling for females should consider holistic maternal health that emphasises not only glycaemic management but also wellbeing, nutrition, physical activity, and social support in order to optimise long term outcomes for both mother and child.

Clinical practice:

Clinicians should recognise the higher risk of YOT2D in offspring of mothers with hyperglycaemia in pregnancy, particularly those with pre-existing T2D. Additionally, youth with T2D should be counselled on the increased risk of congenital malformations linked to hyperglycaemia in early pregnancy.

Figure 1: The intergenerational cycle of type 2 diabetes



Social determinants and impact on type 2 diabetes

Social determinants of health, the conditions into which youth are born, live and work[43], profoundly impact the development, management and outcomes of YOT2D[44, 45]. Social determinants of health influence equity of access to health care, and can have an impact at an individual, family, and community level. These determinants include education, income, food and housing security, structural, commercial and political forces, including the experience of ongoing racism and discrimination. Their influence can extend beyond individual behaviours or health-care quality, accounting for up to 55% of health outcomes[46]. YOT2D disproportionately affects marginalised populations[3, 4] and has been described as an avoidable 'disease of poverty'[3]. In Australia and Aotearoa New Zealand, young people who are Aboriginal, Torres Strait Islander, Māori, Pacific Peoples, Asian, Middle Eastern, or from some refugee communities, experience a higher prevalence of YOT2D[1, 2, 5, 6, 47-51] and are hereafter defined as 'high-risk ethnicities'. Social and economic inequities may contribute to a higher prevalence of YOT2D[3]. Political, economic and structural barriers to culturally safe healthcare, food security, safe housing and opportunities to participate in research and clinical trials are also important[52, 53].

Social determinants of health not only contribute to the risk of development of T2D but also to the progression of complications[54, 55]. They also shape the extent to which health systems enable equitable access to care and support effective lifestyle change. Enhancing understanding of these influences, increasing representation of diverse communities among health professionals, policymakers and researchers, and promoting culturally safe practices are critical to advancing health equity (Figure 3). Equally essential is partnering with consumers and ensuring that the voices of young people, families and communities meaningfully shape service design, research priorities and models of care for YOT2D. It is also important to understand that collective thinking and behaviours regarding health within some communities are influenced by social determinants. This also requires explicit recognition of the ongoing impacts of colonisation, the commercial determinants of health (e.g., widespread availability of sugar-sweetened beverages and ultra-processed foods), and the ways in which poverty and food insecurity constrain health behaviours and access to care. Clinicians, policymakers and researchers must be aware of these structural forces and work in partnership with communities to implement culturally responsive primordial prevention strategies that address and overcome these systemic barriers.

More research and advocacy are needed to improve understanding of how social determinants influence YOT2D and to inform effective interventions to improve health outcomes and ameliorate the effects of social determinants. This work must be undertaken in genuine partnership with communities, led by community-controlled organisations, and grounded in shared decision-making. Strengthening the evidence base in this way will support the development of culturally safe, community-driven strategies that improve health outcomes and address the underlying social determinants of health.

Clinical practice:

Encourage person-centred care processes that address social context. Engage appropriate cultural safety support services early, acknowledging that social determinants affect the whole of health, especially mental health. Explore financial stability, food and housing insecurity, parental employment and education status of the family or household, acknowledging these social determinants as possible markers of health disparity. Consider these issues when formulating education and treatment plans.

Screening for type 2 diabetes

Evidence for screening (or case finding) for T2D in both youth and adults with risk factors remains limited[20, 56-58]. Early detection is particularly important in female youth of reproductive age, given the intergenerational impact of hyperglycaemia on offspring outcomes[59]. While no studies have thus far directly assessed the benefits or harms of risk-factor based screening in youth[60], in low-risk populations, where T2D remains rare pre-puberty[61, 62], screening youth with overweight or obesity alone, without other risk factors for T2D, is unlikely to be cost-effective due to low diagnostic yield[63]. Current recommendations support targeted screening from ≥10 years in youth with overweight or obesity plus at least one additional risk factor. However, in very high-risk populations, screening for T2D using annual HbA1c from the age of 8 years should be considered according to the presence of any risk factor[19, 20] (see Figure 2). HbA1c is the recommended primary screening test, though sensitivity is limited in paediatric populations and thus some affected youth may be missed[64]. Please see Diagnosis section for details as to diagnostic tests.

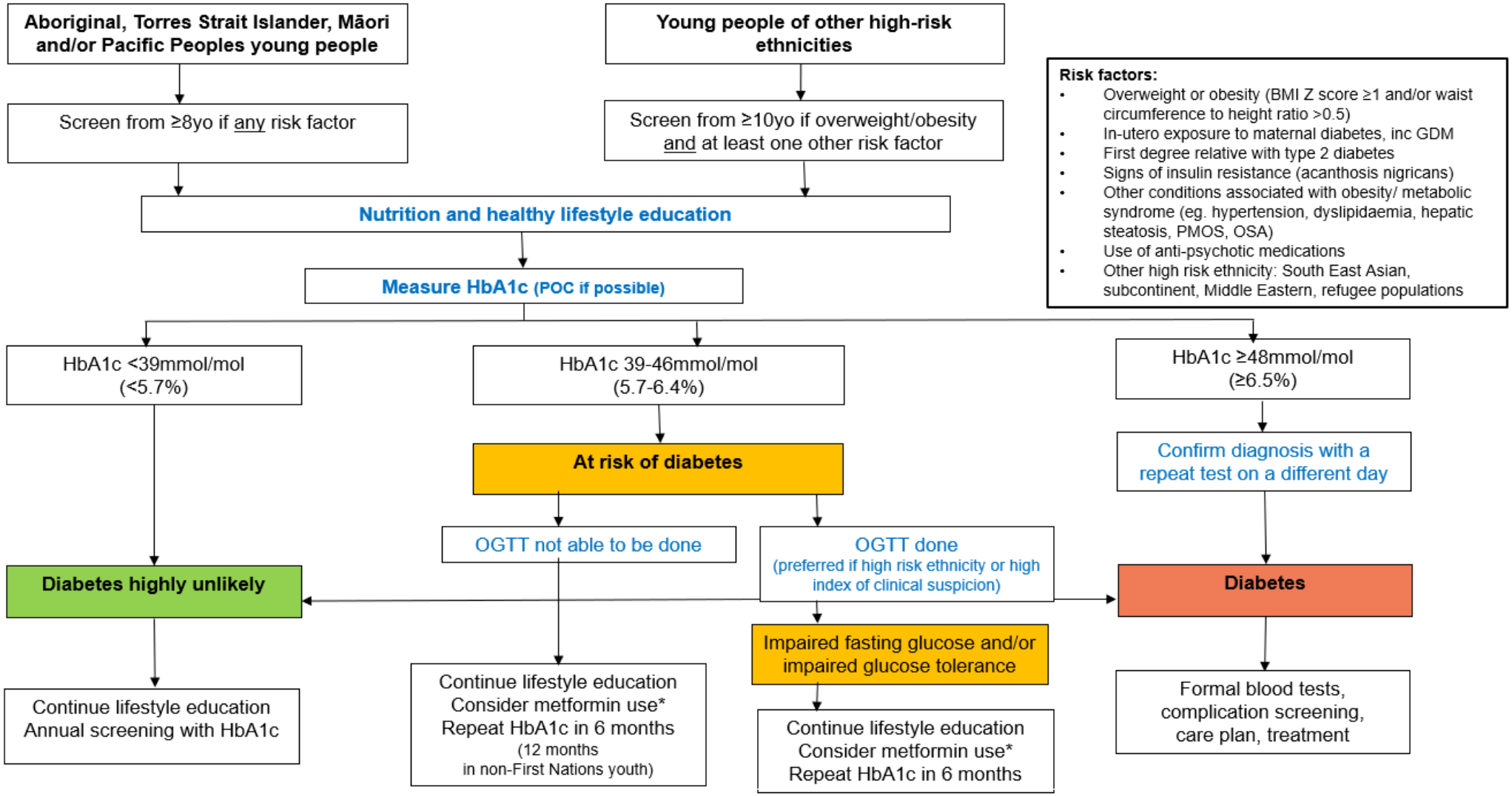
Aboriginal, Torres Strait Islander, Māori and Pacific Peoples communities have a very high background prevalence of T2D and known intergenerational transmission of cardiometabolic conditions. Body mass index (BMI) may underestimate risk in these populations[65, 66], and therefore use of waist-to-height ratio should be considered in addition to BMI[19]. Special consideration should be given to early screening of youth with high-risk genetic conditions (eg. Trisomy 21, Turner, Prader Willi and Bardet-Biedl Syndromes)[20].

The term 'pre-diabetes' is not yet universally accepted in youth despite its wide use in adult guidelines[20, 56] as diagnostic thresholds for pre-diabetes have not been validated in youth and data on the accuracy of various risk assessment tools to help estimate the risk of T2D among youth with pre-diabetes remain inadequate[56]. Consequently, this guideline does not address diagnosis of pre-diabetes in children or adolescents and uses the term 'at risk of diabetes' instead.

Clinical practice:

Screening should begin from 8 years of age in Aboriginal, Torres Strait Islander, Māori and/or Pacific Peoples young people if any risk factors. Risk factors include overweight/obesity, in-utero exposure to GDM or T2D, first degree relative with T2D, presence of acanthosis nigricans, presence of other conditions associated with obesity/ metabolic syndrome, use of anti-psychotic medications. Young people of other high-risk ethnicities should be screened from 10 years of age if overweight/obesity and at least one other risk factor.

Figure 2: Recommendations for type 2 diabetes screening in youth across Australia and Aotearoa New Zealand



BMI = body mass index; HbA1c = glycated haemoglobin; OGTT = oral glucose tolerance test; PCOS = polycystic ovarian syndrome; POC = point of care; * = See pharmacotherapy section
N.B. Consideration should be given to early screening of youth with high-risk background conditions, such as Trisomy 21, Turner, Prader Willi and Bardet-Biedl syndromes

Clinical presentation and diagnosis of T2D in children and adolescents

YOT2D is secondary to both insulin resistance - in the liver, adipose tissue and peripheral tissues - and pancreatic β-cell dysfunction, leading to progressive insulin deficiency[11]. Compared to adult-onset T2D, YOT2D exhibits more rapid β-cell decline following diagnosis[10,67], while insulin resistance appears to remain relatively stable[10, 68, 69]. This accelerated β-cell dysfunction may underly the rapid glycaemic deterioration, higher HbA1c, and 'severe aggressive phenotype' observed in YOT2D[4]. Co-existent insulin resistance due to obesity and physical inactivity, may exacerbate this condition by increasing insulin secretion, and accelerating β-cell failure. Pubertal insulin resistance likely also contributes[70], which may explain the relative rarity of T2D in pre-pubertal youth, and the earlier onset of YOT2D in girls, whose puberty typically occurs earlier than boys[71].

We note that none of the diagnostic criteria for diabetes have been specifically validated in youth and are all extrapolated from adult definitions[71, 72]. Diagnostic criteria for YOT2D are the same as adult-onset T2D. Diagnosis in asymptomatic youth should be confirmed using a different test on the same sample, or with a repeat test on the following day. Clinical presentations and diagnostic investigations are summarised in Box 1 and Box 2. Features assisting in the exclusion of other diabetes types (eg. T1D and monogenic diabetes) are described in Box 2.

Box 1: Clinical presentation of T2D in children and adolescents

Clinical presentation	Proportion of youth	Symptoms, signs and investigations
Asymptomatic	33%[4]	• Diagnosis on screening or random testing
Symptomatic	67%[4]	• Polyuria • Polydipsia • Weight loss • Fatigue • Absent to mild ketosis • Skin infections
Diabetic ketoacidosis*	up to 10%[73-75]	• Plasma glucose >11mmol/L • Venous pH <7.3 • Serum bicarbonate <18mmol/L • Ketosis • Vomiting, abdominal pain, Kussmaul breathing, drowsiness
Hyperglycaemic hyperosmolar state	up to 2%[75, 76]	• Plasma glucose >33.3mmol/L • Venous pH >7.25 • Serum bicarbonate >15mmol/L • Absent to mild ketosis • Effective serum osmolality >320mOsm/kg • Altered consciousness, seizures

*Note increase in DKA prevalence observed during COVID-19 pandemic[74]

Box 2: Diagnostic criteria for type 2 diabetes

Symptoms	Criteria	Issues to consider
SYMPTOMATIC	Classic symptoms of hyperglycaemia and fasting plasma glucose (FPG) ≥7mmol/L, or random plasma glucose ≥11.1mmol/L, or HbA1c ≥48mmol/mol (6.5%)	Hyperglycaemia may be transient during physiological stress (eg. acute infection, trauma, surgery, respiratory distress, circulatory compromise) and should not be considered diagnostic of diabetes in these circumstances.
ASYMPTOMATIC **Diagnostic glucose measurements**[20] *(N.B. Need to confirm diagnosis using a different test from the same sample, or with repeat test on the following day)*	HbA1c ≥48mmol/L (6.5%)*, or	Reproducibility is higher for HbA1c than fasting glucose or OGTT in youth[77]. HbA1c may vary between ethnic populations and should be interpreted with caution, particularly if haemoglobinopathies or anaemia. HbA1c should be performed in a laboratory using a method certified by the National Glycohaemoglobin Standardization Program and standardised to Diabetes Control and Complications Trial (DCCT) assay. Marked discordance between HbA1c and plasma glucose levels raises the possibility of HbA1c assay interference[20].
	Fasting plasma glucose ≥7mmol/L*, or	FPG correlates with hepatic insulin resistance. In YOT2D, post-prandial hyperglycaemia develops earlier, often leading to normal FPG values despite meeting diagnostic criteria for T2D with HbA1c and/or OGTT. Additionally, FPG is influenced by stress and illness, and glucose levels can reduce by 5-7% in a sample in the time between phlebotomy and analysis. (Sources of variation include patient preparation for fasting glucose, intra-individual variation and analytic variation. Pre-analytic variation is due to sample being taken (venous plasma, venous serum, capillary whole blood), time delay until separation from red cells, temperature, and glycolysis inhibitory agents used.)
	2-hour plasma glucose ≥11.1mmol/L during OGTT*	OGTT may be required if cannot diagnose on fasting, random or postprandial criteria. OGTT has poor reproducibility in adolescents[20, 77] and should be repeated if the youth does not have hyperglycaemic symptoms. OGTT should include a glucose load containing equivalent 1.75 g/kg anhydrous glucose dissolved in water to maximum 75 g and a high carbohydrate intake 3 days before the test. HbA1c, FPG and OGTT are equally effective in identifying glycaemic abnormalities but identify differing glycaemic patterns[78]. Note that sample glucose levels can reduce by 5-7% in the time between phlebotomy and analysis.
Other features at diagnosis to help determine diabetes type	Patient history	Features on history which increase probability of T2D include family history of T2D at a young age, high-risk ethnicity, and in-utero exposure to diabetes in pregnancy (GDM or T2D)
	Clinical examination	Features increasing probability of T2D include presence of acanthosis nigricans, elevated waist to height ratio (>0.5), elevated BMI (Z score ≥1), and presence of cardiovascular comorbidities such as hypertension

Symptoms	Criteria	Issues to consider
Other features at diagnosis to help determine diabetes type (cont)	Comorbidities	Cardiometabolic complications occur much more frequently in children and adolescents with T2D than T1D (eg. Proteinuria, hepatic steatosis, dyslipidaemia, hypertension) – FBC, EUC, LFTs, lipids and urine ACR should be tested at diagnosis. Comorbidities more typically associated with T2D than T1D include obstructive sleep apnoea, polycystic ovarian syndrome, and obesity (see Table 4). Autoimmune comorbidities increase the probability of T1D.
	Ketones	The presence of blood ketones ≥1.0 mmol/L in the context of hyperglycaemia increases the likelihood of type 1 diabetes in youth, and likely appropriate to treat as per T1D guidelines until alternative diagnosis is confirmed[20].
	Antibody testing	Consider diabetes autoantibody testing in all paediatric patients with the clinical phenotype of T2D due to higher prevalence of T1D — GAD, IA2, ZnT8 and IAA where available (GAD and IA2 most commonly available). Autoantibodies are present in 10-20% of youth clinically diagnosed with T2D, number and titre of positive antibodies is important in interpretation[79-81]. Discuss significance of antibody positivity with paediatric diabetes services.
	C-peptide	C-peptide should be paired with BGL and fasting status noted. Although evidence for routine use is limited, it is helpful in the nuanced consideration of diagnosis by paediatric/youth diabetes services[82], particularly when the phenotype is atypical. (see phenotype section) Measuring insulin levels is not recommended.
	Consider possibility of rare forms of diabetes	Approximately 2.5-6.5% individuals diagnosed with T2D may have monogenic or mitochondrial diabetes[83]. Genetic testing should be considered when appropriate, in consultation with paediatric diabetes services. Features suggestive of other forms of diabetes include an autosomal dominant or maternal inheritance pattern, absence of typical T2D phenotype or metabolic comorbidities, negative autoantibodies, and atypical features (eg pancreatic insufficiency, renal cysts and other urogenital abnormalities)[20].

HbA1c = glycated haemoglobin; OGTT = oral glucose tolerance test; FPG = fasting plasma glucose; FBC= full blood count; EUC = electrolytes, urea, creatinine; LFT = liver function test; ACR = urinary albumin creatinine ratio; GAD = glutamic acid decarboxylase; IA2 = islet tyrosine phosphatase 2; ZnT8 = Zinc Transporter 8 (ZnT8); IAA = Insulin autoantibodies

* Note that asymptomatic youth without ketosis should have diagnosis confirmed using a different test from the same sample, or with a repeat test on the following day.

Clinical practice:

Diagnostic criteria for YOT2D are the same as those for adult-onset T2D. Diagnosis in asymptomatic youth should be confirmed using a different test on the same sample, or with a repeat test on the following day. Consider diagnostic possibility of other forms of diabetes in discussion with paediatric/youth diabetes services.

Phenotypes of youth-onset T2D

The classical phenotype for T2D includes high BMI, central adiposity and clinical evidence of insulin resistance. There are varying phenotypes of YOT2D, however, and not all affected youth have overweight or obesity[66, 84]. These atypical phenotypes occur more frequently in some populations. In Japan, up to 13% of those with YOT2D do not have overweight or obesity[85], in contrast to only 3% of youth with T2D globally[4]. Youth with atypical phenotypes tend to have lower insulin secretion and milder, but evident, insulin resistance at diagnosis. Atypical phenotypes may also be associated with earlier onset. Australian Aboriginal youth diagnosed before the age of 15 years have a lower prevalence of overweight or obesity (87%), compared to those diagnosed with T2D between 15 and 24 years (97%)[5].

These differing phenotypes are still poorly understood, and require further research to develop targeted therapies to preserve β-cell function[84]. Personalised medicine will become increasingly important as we better understand the diverse phenotypes and their differing complication risk and treatment responses[81].

In-utero exposures likely contribute to these phenotypes, including epigenetic changes and in-utero organ programming that affect β-cell function, potentially reducing the age of onset and resulting in a lower BMI at diagnosis from what has been seen historically[37, 66]. In these contexts, waist-to-height ratio, as a marker of central adiposity, may be of more use clinically than BMI[86, 87]. Maternal malnutrition in pregnancy and nutritional deficiencies in early childhood likely increase risk through multiple interconnected pathways, including changes in the gut microbiome, chronic inflammation, altered insulin signaling and metabolic parameters[38].

Youth who are diagnosed pre-pubertally tend to have in-utero exposure, greater metabolic abnormalities and/or come from higher-risk populations[71]. Internationally, genetic factors, including HNF-1α gene polymorphisms can also influence insulin secretion, leading to T2D occurring at a lower BMI than in youth without these polymorphisms[88]. More than 65 other genetic variants have been identified to increase T2D risk, including 7 in YOT2D[89]. However, these newer variants are thought to only have a small impact on risk and are not yet part of routine clinical practice.

Clinical practice:

Consider the possibility of YOT2D even in youth with atypical phenotypes, and discuss atypical presentations with a paediatric/youth diabetes service. It is important to consider in-utero exposures and family history.

Diabetes education

Initial education

At diagnosis, youth and family should receive individualised multidisciplinary diabetes self- management education that is developmentally appropriate, informed by cultural safety and promotes family-centred behavioural change (see Appendix Table 3). An interpreter should be used for families when English is not their first language. Culturally tailored, written, pictorial and audio-visual educational material in their first language should be used and provided if available. Unfortunately, educational materials for paediatric T2D are limited (See Appendix, Resources section).

Assessment of health beliefs, cultural understandings, social inequities, attitudes, learning styles and psychosocial factors will help to identify barriers that enhance learning and promote sustained engagement (Figure 3). Success in maintaining lifestyle self- care changes and reaching therapeutic goals involves initial and regular diabetes self- management education, including collaborative goal setting for diet, activity, blood glucose and/or continuous glucose monitoring (CGM), and medications.

Diagnostic uncertainty is common in YOT2D. Therefore, to reduce confusion or anxiety for family it is important that messaging is consistent among the health team and that education is holistic and culturally appropriate, and reflects the goals of both youth and the health team[20].

Education should be facilitated by a specialised multidisciplinary team (eg, a credentialled diabetes educator, or a diabetes nurse specialist in Aotearoa New Zealand; a dietitian with specialist youth diabetes experience; a psychologist and/or social worker; a cultural health worker; and ideally a physiotherapist or exercise physiologist) considering the family sociocultural background. Families benefit from coordinated, culturally grounded, multidisciplinary support that builds on strengths, addresses practical needs, and promotes safety, wellbeing, and engagement[90]. Additional services may be indicated, for example, Social Work for support to address financial stressors, food insecurity, family dysfunction and housing instability (see Social Determinants and Engagement sections). Prioritise involvement of cultural health workers and sharing of lived experience. Prioritise collaboration and integration of care with primary health care services.

Follow-up

Specialised multidisciplinary paediatric follow-up should occur at least monthly for the first 3 months to adjust medications to achieve target glucose levels and review lifestyle modifications, then at least 3 monthly. A 'shared care' model between primary health care services and the multidisciplinary diabetes team is recommended. In areas where access to multidisciplinary diabetes teams are limited, appropriate models of care and adequate resourcing are critical to facilitate shared clinical responsibility with primary health care.

Providing ongoing follow-up by telehealth services may be a feasible option for families in regional and remote settings to facilitate the establishment and maintenance of ongoing therapeutic relationships with health professionals but needs careful consideration of resourcing and effectiveness of engagement[91]. This should not exclude access to face-to-face outreach services, integrated with primary health services and community based workers. Schools should also be involved in the management of diabetes to facilitate ongoing support.

Primary health care services continue to provide a critical role and enable close collaboration between families, tertiary and local services. This includes liaison with diabetes services, ongoing medication prescribing, monitoring for comorbidities and complications, holistic support of social and emotional

wellbeing, contraception, and continuity of care to enhance engagement. All diabetes services should audit the clinical care and outcomes of youth with T2D, preferably benchmarking to other services[92, 93].

Clinical practice:

Culturally safe multidisciplinary support and education, including holistic social support as needed, is essential. Close follow-up is required. Diabetes services should audit the clinical care and outcomes of youth with T2D.

Considerations for enhancing engagement

Diabetes management should consider the complex interplay of factors influencing engagement and health-related behaviours (Figure 3).

Figure 3: Strategies and issues to consider in engagement of youth living with type 2 diabetes across Australia and Aotearoa New Zealand

Glycaemic monitoring

The frequency and method of self-monitoring of blood glucose needs to be individualised according to treatment type, family circumstances and need to improve glycaemia. Aim for blood glucose levels 4–6 mmol/L (fasting) and 4–8 mmol/L (2 hours postprandial). A target glycated haemoglobin (HbA1c) of 48 mmol/mol or below (≤ 6.5%) is recommended[20] due to the anticipated longer disease duration, the accelerated rate of developing complications[12] and the higher mortality compared with T1D[13] and adult-onset T2D[4]. This target should be achieved without hypoglycaemia and/or undue treatment burden. In the TODAY study, hypoglycaemia rates, even for patients taking insulin, were lower in youth with T2D[94]. HbA1c should be assessed every 3 months.

Continuous glucose monitoring systems (CGMS) are recommended for youth with T2D who require insulin, in whom there is inadequate finger-prick blood glucose level monitoring for safety, for youth with suboptimal diabetes management, where there is suspected hypoglycaemia, and when there are treatment changes[20, 21]. We also note the negative impact of hypoglycaemia on the developing brain in youth living with T2D who are on insulin, similar to those with T1D. CGMS has been shown to reduce the risk of hypoglycaemia[95]. Among adults with T2D, CGMS significantly improve the time that glucose levels are in target range[96, 97], and this is now also shown in youth[98]. CGMS may also increase engagement, behavioural changes and diabetes self-management in youth[99]. CGMS report parameters such as time in range, time in tight range, glycaemic variability (%), glucose management indicator (estimated HbA1c) and estimated average glucose are all useful in assessment of glycaemia and treatment response, noting that there are as yet no defined normal ranges for individuals with T2D.

Clinical practice:

Individualise frequency and method of blood glucose level monitoring. Target blood glucose levels are 4–6 mmol/L (fasting) and 4–8 mmol/L (2 hours postprandial), with a recommended glycated haemoglobin (HbA1c) target of 48 mmol/mol or below (≤ 6.5%). CGMS is recommended when there are treatment changes, youth on insulin, if HbA1c is above target range, or there are safety concerns.

Nutrition, physical activity and daily living

Since the 2020 guidelines, there has been a shift towards multi-component interventions[20] that consider all aspects of life and emphasise the importance of community support and collaboration with a multidisciplinary team, including a specialised diabetes dietitian and exercise physiologist.

Global trends of increasing youth obesity are multifactorial, influenced by changes in food intake, decreased physical activity, and the obesogenic environment, all contributing to a positive energy imbalance in recent decades[100]. It is also important to consider the ongoing impacts of colonisation and discrimination in First Nations youth[101] and the impact of food insecurity[102].

These guidelines serve as a starting point and should be tailored to meet the unique needs of youth, as well as their families and communities (Figure 3). Youth with health-related motivations for weight loss and positive family influences are more likely to adopt protective eating habits and effective weight management strategies[103]. While there is limited evidence specific to YOT2D, current recommendations are also derived from research on both youth obesity and adult-onset T2D.

Current nutrition recommendations and behavioural interventions (Table 2) aim to:

- Enhance glycaemic outcomes
- Reduce the risk of complications and co-morbidities (e.g. central adiposity, dyslipidaemia)
- Improve overall quality of life
- Facilitate weight management

Youth living with T2D are encouraged to follow the Australian Guide to Healthy Eating and the New Zealand Food and Nutrition Guidelines for Healthy Children and Young People (aged 2–18 years)[104, 105]. Healthcare professionals guiding dietary and lifestyle modifications should use positive language when delivering recommendations (see Appendix Table 1). Individual empowerment and motivation are crucial for successfully implementing health-enhancing changes (Figure 3). Physiological, social and environmental factors influence an individual's ability to change. Engaging youth by addressing their perceptions of diabetes and barriers to adherence can enhance self-care engagement and improve health.

Education and interventions should be practical, age- and culturally appropriate, and tailored to the individual and family needs, ensuring sustainability in implementation. This includes knowledge of carbohydrates, label reading, and portion control. While carbohydrate counting may be beneficial, the potential burden and health literacy of the individual and family should be considered.

Dietary recommendations should emphasise whole foods and nutrient-rich options. Consuming foods rich in whole grains, vegetables, fruits, lean meats or alternatives (e.g. legumes and nuts), and low in salt, free sugars, and unhealthy fats (especially saturated and trans fats) provides the foundation for optimising health, well-being, growth and development in youth and their families. There is insufficient evidence regarding time-restricted eating (e.g. intermittent fasting) in youth with T2D[106]. Short-term dietary adaptations[106, 107] should only be implemented under the guidance of a specialised diabetes dietitian to prevent nutrient deficiencies.

In addition to dietary adaptations, a holistic approach addressing youth health factors, including sedentary behaviour, sleep, and risk-taking behaviours is crucial. Reducing sedentary behaviour should focus on improving sleep routines and minimising screen time. Research indicates that children with as little as 40 minutes less sleep per night tend to consume approximately 500 calories more daily, often from poorer food choices and highly processed, calorie-dense foods[108, 109] while physical activity patterns remain unchanged.

Youth should be encouraged to increase physical activity to a minimum of one hour daily, consisting mainly of moderate to vigorous intensity aerobic activity[20]. This assists with weight management, enhances insulin sensitivity and cardiometabolic function, and reduces subcutaneous and visceral fat[110]. Muscle strengthening resistance training is also important in improving insulin sensitivity, lean mass, blood pressure, bone mineral density and dyslipidaemia. Physical activity may also enhance mental health and wellbeing[110].

<u>Clinical practice:</u>

Need to consider food security and access to safe physical environments. Avoid stigmatising language and use a holistic and culturally grounded approach to optimise health. Tailor education and health goals to the needs of the young person and their family.

Table 2: Strategies for nutrition, physical activity and healthy living interventions

Nutritional Interventions				
	Strategy	**Features**	**Recommendations**	**Considerations**
Family and home environment	**Food Security**	Youth living with T2D may be more at risk of household food insecurity than the general population[111].	Offer practical advice, strategies and support services for youth to achieve nutrition and weight management recommendations, acknowledging the challenges faced by families experiencing household food insecurity[112].	Household food insecurity may negatively impact the ability for youth with overweight or obesity to change their BMI z-score with multicomponent lifestyle interventions[113].
	Traditional Food and Culture	Nutrition advice and education must be culturally safe, and adapted to the meet the needs of the family and the developmental stage of youth living with diabetes[114].	Acknowledge the cultural systems informing conceptualisations of food and its relationship with health[115]. Provide nutritional resources that include traditional foods where possible[116, 117].	Appreciate the significance of traditional foods and intergenerational cultural knowledge that supports positive relations with food.[115]
	Family Based Therapy	A holistic approach that engages family members and the community to support long-term behaviour change and cardiovascular risk reduction is recommended[112].	Encourage the family to establish a regular meal pattern where they sit and eat nutritious meals together.	See above – Food Security.
Diet composition	**Food Quality**	Dietary recommendations for youth with T2D are based on healthy eating guidelines suitable for all youth and their families[105, 118]. A lower HbA1c is associated with diet lower in high fat foods for youth with T2D[119].	Recommend a balanced diet comprising of nutrient rich foods including whole grains, fruits and vegetables. Carbohydrates should provide between 40-50% of total energy intake (TEI)[112] Nutritious high fibre and lower GI carbohydrates should be considered over higher GI carbohydrates Reduce saturated fat to <7% and total dietary fat to 25–35% TEI Reduce sugar sweetened beverages (soft drinks, energy drinks, juices and fruit drinks) Limit foods high in saturated fats such as takeaway, fast foods or ultra-processed foods	Geographical location and cultural attributes should be acknowledged, and consider variety of foods available e.g. including meals provided in schools The amount of carbohydrate consumed at a meal has more of an impact than GI alone

	Strategy	Features	Recommendations	Considerations
	Body Composition	Weight reduction of ≥7% has shown a small but significant benefit in cardiometabolic status for youth with T2D[120]	Decrease total energy intake to promote weight reduction. Waist circumference and waist/height ratios may be a more useful predictor than BMI of metabolic or cardiovascular risk in some population groups.[121]	Other measures for monitoring change in body composition (BI, WC, DEXA) should be used in addition to weight Weight management is multi-component , requiring nutrition and physical activity adaptations Body composition goals should consider pubertal stage to ensure linear growth is not adversely impacted[107]
Medical Nutrition Therapy Due to dietary restrictions, the following interventions should only be considered short term (due to risk of micronutrient deficiency) and under the guidance of a specialist diabetes dietitian. Note: no difference in weight loss has been described between a VLED and LCD, suggesting that an overall decrease in energy intake, irrespective of macronutrient break-down, is most important for achieving weight loss.	**Very Low Energy Diet (VLED)**	The use of VLED may be considered, particularly during early stages of T2D diagnosis[122, 123] VLED is used to achieve rapid weight loss, and should only occur under dietitian guidance[122]	Prior to implementation, appropriate screening of disordered eating should occur[124, 125] Short term (10-12 weeks) calorie restriction of between 600-800kcal/day[123] Meal replacement therapy or food restriction can be used A daily multivitamin should be introduced at the commencement of a VLED Maintain adequate hydration with individuals aiming for 2L/day	Due to macronutrient restrictions, including carbohydrate intake, VLED can induce a mild state of ketosis The use of dietary restrictions within this population can increase risk of subsequent disorder eating and body image issues Potential increase in DKA risk in patients on SGLT2-Is if put on VLED
	Low Carbohydrate Diet (LCD)	Low carbohydrate diets have been used successfully within the adult population[126] particularly in those with comorbidites[127]	Classification of carbohydrate restriction is as follows[107] • Low 26% to 44% EER • Very Low <26% EER (~20-50g carbohydrates) • Ketogenic <20g carbohydrates (Note: in CYP, ketogenic diet is solely used in treatment for drug-resistant epilepsy)	Carbohydrate reduction should occur through decreasing added sugars and processed foods intake Growth and development should be monitored when carbohydrate restriction occurs[107]

Daily Living				
	Strategy	**Features**	**Recommendations**	**Considerations**
	Sleep	Sleep duration and sleep quality are associated with HbA1c in adults with T2D[128]. Daytime sleepiness is associated with higher HbA1c in overweight/obese youth with, or at risk for, T2D[129]. Interventions aimed at improving sleep could be beneficial for enhancing dietary intake and weight status in children[130]	Recommended sleep durations are as follows:[131] • 9-11 hours per day for school-aged children • 8-10 hours for teenagers • 7-9 hours per day for young adults. Establish improved sleep hygiene, such as regular sleep and wake times, and reduce screen time prior to bedtime[132].	Recognise the presence of gender and cultural differences in habitual sleep patterns in youth[133, 134]. Consistent sleep routines avoids risk of day-time napping, which can have significant impact on weight. Loss of sleep is linked to increased energy intake, regardless of changes in sedentary behaviour and physical activity[130].
	Driving	Driving and fine motor skills can be impaired by chronic hyperglycaemia, as well as risk of hypoglycaemia.	Discuss driving risks related to hyperglycaemia, and also need for glucose monitoring if on medications that can cause hypoglycaemia. Consider safety for licence.	
Youth Health Factors				
	Strategy	**Features**	**Recommendations**	**Considerations**
	Smoking & Vaping & Illicit Drugs	Cannabis use is associated with changes in appetite and eating behaviours, inconsistent glucose monitoring and insulin administration youth and adults with T1D[135, 136]. Smoking has been linked to increase risk of complications in adults with T2D.	Education about the health and behavioural risks associated with using cannabis, tobacco, vaping and illicit drugs and discuss strategies to minimize risks. With the consent of youth, referral to local community cessation programs should occur.	The minimum legal age to buy or consume tobacco in Australia and New Zealand is 18 years old. People under 18 years need a prescription to access vapes.
	Alcohol	Raise awareness with youth and their families about the risk-taking behaviour associated with alcohol and substance use[112, 137].	Alcohol consumption is considered harmful and not recommended for children with diabetes. For those that do consume alcohol, aim to reduce harm by providing education about the risks of alcohol use and discuss strategies to minimize risks.	The minimum legal age to buy or drink alcohol in Australia and New Zealand is 18 years old.

	Strategy	Features	Recommendations	Considerations
	Active Lifestyle	Physical activity helps lower glucose levels and blood lipids in youth with diabetes[138].	Activity recommendations are based on the general guidelines suitable for all youth[139] e.g. at least one hour of physical activity every day. Muscle and bone strengthening activities should be included 3 days per week Limit sedentary behaviours, such as television viewing, video games, and computer use	Movement should be encouraged, and individual abilities may vary.
	Screen Time	Limiting screen time in youth has been associated with[140]: - reduced risk of excess adiposity - a higher intake of healthy foods, like fruit and vegetables and dairy products	Screen time should be limited to <2 hours per day	Youth's screen use in bed impairs sleep, especially when screen time is interactive e.g. gaming, or involves multi-tasking[132, 141] The increasing use of social media platforms and ease of access of electronic devise/screens will likely impact sedentary behaviours for youth

BI= bioimpedance, WC = waist circumference, DEXA = bone mineral densitometry, EER = estimated energy expenditure

Psychological health

Psychosocial distress among youth is common across Australia and Aotearoa New Zealand[142, 143]. YOT2D disproportionately affects youth facing structural disadvantage[75, 144, 145] (see Social Determinants section), where diabetes-related distress and mental health issues frequently co-occur[146-149]. Assessing for these comorbidities and ensuring care is holistic and adapts to cultural and family-based strengths (Figure 3) may optimise outcomes for youth living with T2D[150].

A holistic approach to diabetes care emphasises the overall impact diabetes has on an individual and their family's wellbeing. Integrating physical and mental wellbeing aligns with First Nations' frameworks of health, including Māori, Pacific Peoples, Aboriginal, and Torres Strait Islander peoples[151-155]. Within these frameworks, health is more than physical wellbeing alone and includes mental wellbeing, connectedness to family, spiritual and cultural identity. The inclusion of these relational factors is essential to improving equity and health outcomes for First Nations people[153, 156, 157].

Depression affects 15% to 38% of youth with T2D[147, 148]. Among adults, a bidirectional relationship between diabetes and depression has been reported[158]. The SEARCH study and Pediatric Diabetes Consortium have independently found that youth with T2D reported significantly higher levels of depression and lower quality of life compared to their peers with T1D[147]. Up to one in five youth (19%) with T2D have a neuropsychiatric diagnosis, including depression, attention deficit hyperactivity disorder, or neurodevelopmental disorders, at the time of their diabetes diagnosis[159]. Use of psychotropic medications including atypical antipsychotic medications may increase risk of diabetes (see Screening, Figure 2).

Approximately one-third of adolescents with T2D report elevated diabetes distress[160]. In comparison to depression and mental wellbeing, diabetes distress in youth with T2D endures[16, 161]. In Australia, 53% of adolescents with T2D reported moderate to severe diabetes distress, and 58% experienced depressive symptoms[16]. Youth identified stigma, management burden and mental health challenges as significant concerns, calling for empathetic, supportive care for well-being and diabetes self-management[16, 117]. In regional Australia, diabetes distress has been linked to suboptimal glycaemia[162].

Among adults with T2D, 80% experience stigma, and 20% experience discrimination, with a recent consensus statement calling for an end to diabetes stigma and discrimination[163]. Aboriginal youth face similar challenges[164]. Psychological factors can contribute to inconsistent medication adherence[165], disordered eating[166, 167], needle anxiety[168], reduced readiness to adopt self-care behaviours[169] and adversely impact sleep[170]. These may contribute to higher rates of complications and morbidity associated with diabetes. The association between mental health comorbidities and suboptimal glycaemia among youth[149, 162] and increased hospitalisation and mortality among adults with T2D[171], emphasises the importance of mental health support for improving outcomes in YOT2D.

Body image concerns, particularly binge-eating, are common in this population[166, 172], yet remain under-researched. Youth with a higher weight demonstrated increased risk for developing unhealthy relationships with their body and with food[173], making this an important focus of clinical inquiry.

Annual psychological health screening is recommended in youth with T2D from 12 years of age[174]. Screening for psychological distress (Box 3) highlights to youth and their family that physical and mental health are interconnected, reflecting the importance of a holistic model of health. Literacy levels should not be a reason not to screen for psychosocial distress. For youth, there are both diabetes-specific screening tools available with parent-proxy versions, as well as generic mental wellbeing screens. Each of these screening tools provide cut-off scores indicating the need for further support and can help identify youth with T2D who have

higher levels of stress and distress. The HEAASDSSS framework can be introduced over multiple clinical reviews and assists in building rapport. Other tools can be used as determined by clinical need.

At-risk youth should be offered appropriate mental health services, either in conjunction with the diabetes clinic or through community-based mental health programs or could be encouraged to seek a mental health treatment plan (if available) through their primary health care provider. Screening for psychological comorbidities among youth with T2D, including anxiety, diabetes distress, disordered eating, low mood should occur at diagnosis and at regular follow-up intervals[175]. Low English literacy should not be a barrier to assessment.

Box 3: Screening tools for psychological health in youth-onset T2D

TOOL	Age range	Measures, Considerations	Notes/ Scoring
HEEADSSS framework[176]	10 years +	Approach exploring the many domains of young people's lives. Can help to build rapport and identify risk and protective behaviours. Should be completed with young person alone and may take several encounters to complete. Designed to start with less threatening topics. Home Education and employment Eating, exercise, sleep, physical health Activities, peer relationships, social media Drugs and alcohol Sexuality and gender Suicide, self-harm, mood Spirituality and safety	Recommended to use in all young people
Problem Areas in Diabetes – Teen Version (PAID-T)[177]	11-19 years	Diabetes distress, recommended in all youth aged 11-19 years[178]	A score ≥ 44 is the preliminary cut-off score.
Diabetes Distress Scale (DDS)[179]	10 years +	Questions focus on overall diabetes care, with less focus on the burden of insulin injections. DDS also captures distress relating to interactions with health systems.	A score ≥2 defines clinically significant distress
World Health Organization – Five Well-Being Index (WHO-5)[180]	9 years +	Measure of general emotional wellbeing	A score on the WHO-5 ≤50 indicates lower wellbeing and is used to screen for depression.
Kessler Psychological Distress Scale (K6, K10)[181]	11 years +	Measures non-specific psychological distress	A score ≥ 13 indicates higher probability of psychological distress*
Patient Health Questionnaires (PHQ-2, PHQ-9)[182, 183], youth version of PHQ-9 (PHQ-A)[184]	12 years + (PHQ-2) 13 years + (PHQ-9) 11-17 years (PHQ-A)	Depression symptoms PHQ-9 has been adapted and validated for use among Aboriginal adults (aPHQ-9) and may be considered for use among young adults[185-187].	A score ≥ 12 (PJHQ-9) indicates depression symptoms in people with diabetes (≥3 for PHQ-2, ≥10 for PHQ-A, ≥10 for aPHQ-9)

TOOL	Age range	Measures, Considerations	Notes/ Scoring
SCOFF[188]	12 years +	Assesses eating behaviour challenges	A score ≥ 2 indicates further assessment is required.
mSCOFF[189]	11 years +	Assesses eating behaviour challenges for those using insulin	A score ≥ 1 indicates further assessment is required.
Aboriginal and Islander Mental health initiative (AIMhi)[190]	No age specified	Social and emotional wellbeing assessment tool for Aboriginal and Torres Strait Islander youth	Variety of screening resources available at: https://www.menzies.edu.au/page/Research/Projects/Mental_Health_and_wellbeing/AIMhi_NT_-_Australian_Integrated_Mental_Health_Initiative/
Strengths and Difficulties Questionnaire[191]	3-17 years	25 item measure of clinically significant emotional and behavioural difficulties, highlights areas of difficulty in emotional regulation requiring further investigation across five domains: emotional symptoms, conduct problems, hyperactivity, peer problems, and prosocial behaviour or strengths.	Validated to identify 'at-risk' children through total strengths and difficulties score, derived by summing 20 of the 25 SDQ items (0-13 low risk, 14-16 moderate risk, 17-40 high risk of clinically significant difficulties).

*Australian scoring https://www.abs.gov.au/ausstats/abs@.nsf/lookup/4817.0.55.001Chapter92007-08

Therapeutic approaches should consider the cultural, socioeconomic, and psychological factors that influence health-related behaviours. The historical legacy of colonisation and past discriminatory policies continue to adversely affect health and health-related behaviours among First Nations youth[192, 193]. Cultural strengths and self-determination, however, are powerful protective factors and may form a foundation for positive change and adaptability[194]. The intergenerational impact of diabetes diagnosis is important to consider, as family and community members are often a source of support[157]. Lower stress levels and greater resilience are associated with improved readiness for self-care behaviours[195] and a sense of mastery[196], which can protect against depression and anxiety. Interventions such as mindfulness, cognitive behavioural therapy, and resilience-building have shown promise in improving mental health and health behaviours among female youth at risk for T2D[197-199]. Future therapeutic approaches should prioritise addressing the psychological well-being of youth with T2D, recognising its potential to enhance diabetes management and overall health outcomes. Person-centred and family focused care may enhance engagement and improve outcomes for youth with T2D[175, 200, 201] (Figure 3).

Care for youth with T2D ideally involves a multidisciplinary team. Household food security, housing stability, safety of the physical environment (eg. animal management in local area) and family financial resources should be considered when developing treatment strategies in collaboration with the youth and their family. Healthcare providers are advised to avoid stigmatising language[163] (Appendix Table 1). Positive relationships with health care providers are valuable for engagement and improving outcomes[157]. Promoting a comprehensive understanding of the multifaceted nature of YOT2D, which includes psychosocial health and extends beyond lifestyle behaviours is encouraged (see Figure 3). Recognising the individual's strengths and those associated with family, spiritual and cultural identity will enhance the diabetes treatment plan.

Clinical practice:

Consider importance of psychological health in optimising overall health for youth living with T2D and the complex interplay of culture, socioeconomic and psychological factors. Consider strengths, spirituality and culture. Aim for person-centred, empathetic care, avoiding stigmatising language. Address diabetes distress and screen for psychological comorbidities. Explore eating behaviours. Engage mental health and culturally embedded supports early in management.

Pharmacotherapy (see Appendix Table 4 for detailed information on each medication class)

Initial therapy will depend on clinical presentation at diagnosis (Figure 4)[20]. Recommended medications for treatment of YOT2D are summarised in Table 3 and a detailed list is in Appendix (Table 4).

1. Medications

Metformin should be commenced in all cases at diagnosis except for those presenting with DKA or HHS (metformin should be commenced after resolution of these complications[202, 203]). This should not be delayed awaiting paediatric review. It should also be considered in youth with impaired fasting glucose and/or impaired glucose tolerance, in addition to lifestyle changes, though there is no definitive evidence for its use in this setting[204]. It may improve metabolic parameters beyond hyperglycaemia.

Metformin should be avoided in youth who have intolerable side effects, known allergy, or renal insufficiency (GFR <50ml/min). The recommended starting dose is metformin extended release (XR) 500mg after the evening meal or 250mg immediate release (IR) once daily if youth less than 10 years or weight <40kg. Metformin liquid (available as 100mg/ml and 1000mg/5ml) should be limited to younger children due to high cost. Increase metformin up to 2000mg/day (once daily XR, 1000 mg twice daily IR) over 3-4 weeks as tolerated[205]. Discontinue metformin 48 hours prior to elective surgery, radiological studies involving iodinated contrast media and during gastrointestinal illness. Gastrointestinal side effects are seen in 40% with initial metformin use. Slow dose escalation, avoidance of intermittent adherence, use of extended release formulation, and administration with food reduce or prevent these symptoms. A minority of youth (8%) are unable to tolerate metformin[202, 206]. Metformin is not associated with hypoglycaemia and may provide weight loss and reduce cardiovascular risk[120].

Insulin is the first line medication in children/adolescents presenting with metabolic decompensation at diagnosis and in YOT2D at any stage of their diabetes if they are experiencing metabolic decompensation. It is used as 'rescue' medication and where possible is used only for short periods of time. (see Figure 4)[114, 207].

Insulin therapy achieves rapid improvement in glycaemia, with low rates of symptomatic hypoglycaemia[206, 208]. Long-acting insulin can be commenced at 0.25-0.5 units/kg/day, titrating up to a maximum of 1.5 units/kg/day[20], at diagnosis or if metformin alone does not meet glycaemic targets. Prandial insulin may be considered if glucose targets are not achieved but acknowledging this may be unsustainable for families to continue. Where possible, if glycaemic targets are met, insulin should be reduced by 30 to 50% weekly until ceased, transitioning to metformin monotherapy (see Figure 4). This will minimise insulin's undesirable side effect of weight gain. Insulin pump therapy, shown to benefit adults in the OpT2mise study, may be considered for youth unable to be weaned from insulin but is not currently funded for T2D in Australia or Aotearoa New Zealand[209, 210].

HbA1c ≥64mmol/mol (8.5%) at diagnosis or prolonged diabetes duration increases likelihood of sustained insulin requirement. The TODAY study showed that over 50% of youth on metformin monotherapy required insulin within 5 years and that HbA1c ≥43mmol/L (6.3%) three months after initiation of metformin was a predictor of eventual rise in HbA1c irrespective of treatment arm[203, 211]. However, the RISE study showed that short-term insulin therapy in newly diagnosed YOT2D did not prevent the decline in β cell function in YOT2D[212].

Clinicians should ensure that appropriate medication storage is possible in the home setting and engage support persons to aid with medication administration. Youth on several medications may benefit from a dosage administration aid. The family should be provided with clear accessible medication information, and consider pictorial guidance for families with low literacy. Their general practitioner should be notified regarding any medication changes.

Regular follow-up should occur at least quarterly to ensure medication adjustment, review modifiable lifestyle measures and assess glycaemia. Supportive, non-blaming approaches should be used to address barriers in achieving glycaemic targets. Treating to target HbA1c improves long-term glycaemic management[207]. Education regarding injection technique and management of hypoglycaemia is required.

2. Second line agents

When glycaemic targets are not met within 3 months of diagnosis (acknowledging HbA1c at diagnosis) or during a clinical review, a second agent should be commenced under the supervision of a paediatric endocrinologist, as per Figure 4/ Table 3. A wide range of diabetes medications are TGA approved for adults with T2D. Many diabetes medications have proven efficacy in youth-onset T2D with safety profiles similar to adults. (See Appendix Table 4)[213].

If glycaemic targets are not met (see Figure 4) a glucagon-like peptide-1 receptor agonist (GLP-1RA) and/or sodium-glucose cotransporter 2 inhibitor (SGLT-2i) should be commenced. Choice of medication should target weight optimisation and metabolic risk reduction in addition to glycaemia, as well as other potential barriers such as individual preferences, cost, route of administration, dosing regimen and potential side effects. In light of the larger effect on HbA1c with GLP-1RAs than SGLT2is, GLP-1RAs would be recommended for most youth[21,22], however, careful consideration of phenotype and accurate diagnosis of YOT2D is required before use. SGLT2is may be of particular benefit in youth with renal impairment. Contraception is required for female youth, and those assigned female at birth. GLP1-RAs may improve fertility and therefore increase the risk of unplanned pregnancy. There are unknown effects on the foetus.

GLP-1RA have been used for 20 years in adult T2D, meta-analysis of safety in GLP-1RA have shown no increase in pancreatitis or pancreatic cancer, no impact on suicide and GLP-1RA demonstrate cardiovascular and renal benefits in addition to glycaemia benefits. In intentional or accidental overdose with GLP-1RA, adverse effects are minor and no fatalities have resulted[214, 215]. GLP-1RAs can be self-administered, and weekly injections may improve adherence for some youth. As administration technique has been shown to be poor, clear instructions and injection technique demonstration is needed to ensure ongoing good technique[216].

SGLT2is, including empagliflozin and dapagliflozin, are administered once daily orally and are available in combined tablets with metformin. They have been shown to be efficacious in lowering HbA1c in YOT2D. There are additional renal and cardiovascular benefits making these a suitable drug choice in YOT2D and the combined therapies may improve adherence for some youth due to reducing cost and volume of medication.

3. Medications not recommended in youth

As DPP4 inhibitors or sulfonylureas are not as efficacious in YOT2D as in adult-onset T2D, and thiazolidinediones have been shown to have unacceptable safety concerns (see Appendix Table 4), these medications should be avoided.

Clinical practice:

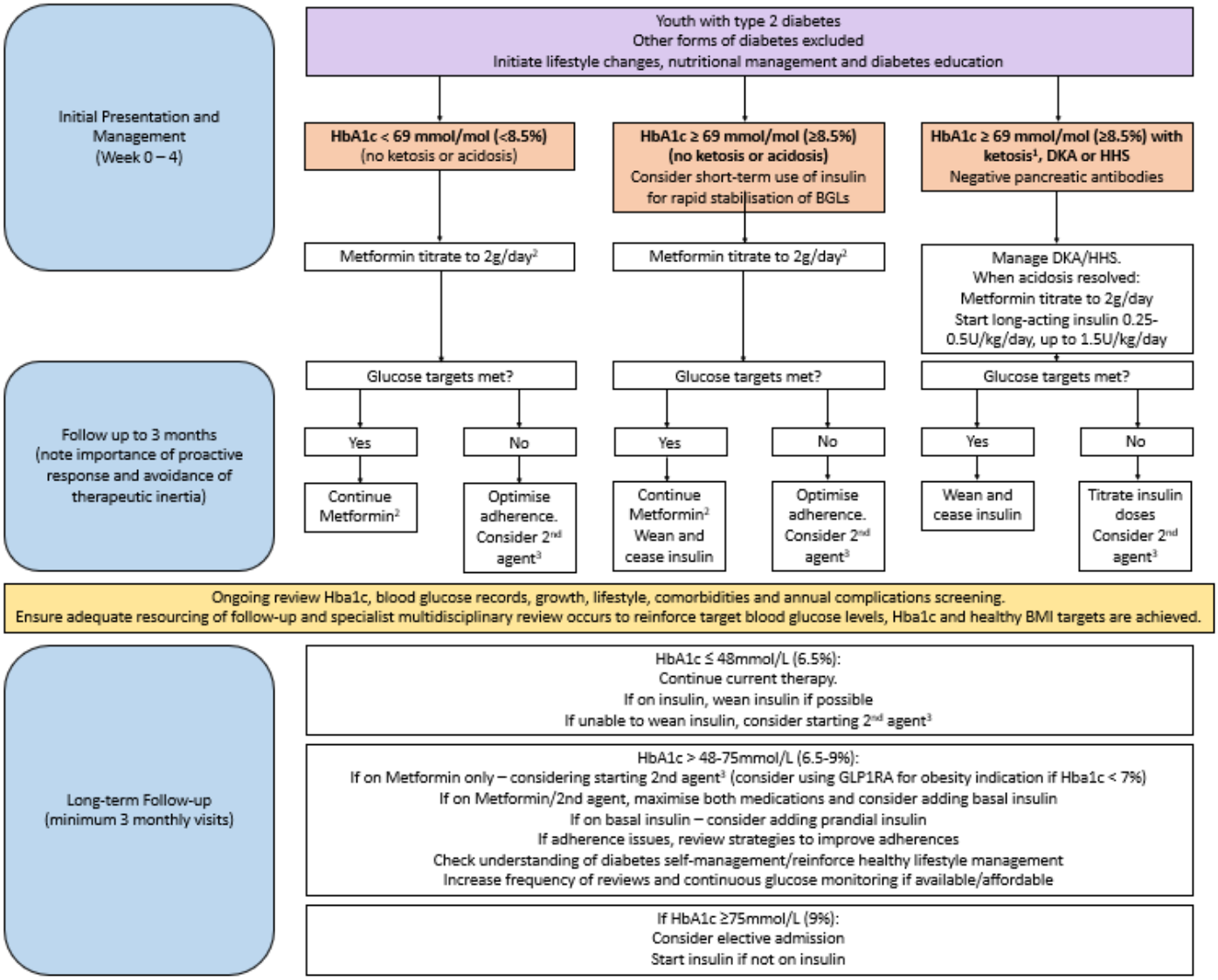
Initial therapy will depend on clinical presentation at diagnosis. Metformin should be commenced in all cases at diagnosis except for those presenting with DKA or HHS. Metformin should not be delayed awaiting paediatric review. When glycaemic targets are not met within 3 months of diagnosis or during a clinical review, a second line agent should be commenced under the supervision of a paediatric endocrinologist.

Table 3: Pharmacotherapies for youth-onset T2D – summary

Drug Class	Medication	Dose	Side effects	Glycaemic efficacy	Potential metabolic benefits
First line agent					
Biguanide	Metformin	250-2000 mg/day	GI: abdominal pain, nausea, diarrhoea, bloating, loss of appetite; headache, B12 deficiency. Rare: Lactic acidosis with concurrent illness or dehydration Pregnancy safety: TGA category C Side effects may recur after a period of non-adherence	HbA1c reduction -0.7 to -1.2%	Improved lipids, reduced BMI and adiposity
	Metformin XR	500-2000 mg/day			
Insulin	Glargine	0.25-1.5U/kg/day	Hypoglycaemia, weight gain, hypersensitivity at injection site Pregnancy safety: TGA category B	HbA1c reduction -0.61-5.22%	
Second line agents					
GLP-1RA	Semaglutide	0.25-1 mg/week	GI: nausea, vomiting, diarrhoea, heartburn Rare: acute pancreatitis, gallstones, cholecystitis, gastroparesis, bowel obstruction, acute kidney injury Mild hypoglycaemia. Potential increased risk of medullary cell carcinoma. Pregnancy safety: TGA category D	No published data yet in adolescents with T2D	BMI reduction greater in youth with obesity than YOT2D Improved MASLD, dyslipidaemia and hypertension, anti-inflammatory, reduced endothelial dysfunction, renoprotective
	Dulaglutide	1.5 mg/week		HbA1c: -0.6% (0.75mg), -0.9% (1.5 mg) vs +0.6% (26w)	
	Liraglutide	0.6-1.8 mg/week		HbA1c: -0.64% vs +0.42% (26w) HbA1c: -0.5% vs +0.8% (52w)	
SGLT2i	Empagliflozin	5-25 mg/day	Dehydration, GU infections (UTI, thrush), headache, nasopharyngitis, hypoglycaemia (when used with insulin). Rare: euglycaemic diabetic ketoacidosis. Pregnancy safety: TGA category D	HbA1c: -0.17% vs +0.68% (26w) Weight: NS	Renoprotective No change in BMI Decreased intracellular lipids Reduction in cardiovascular risk Anti-inflammatory
	Dapagliflozin	5-10 mg/day		HbA1c: -0.26% vs +0.5% (24w NS)	

GLP-1RA Glucagon-like peptide 1 receptor agonist
GI Gastrointestinal
TGA Therapeutic Goods Administration (Australia)
SGLT2i Sodium-glucose cotransporter 2 inhibitor
GU Genitourinary

Figure 4: Treatment algorithm for type 2 diabetes in youth



1. Ketone testing
Daily ketone test to avoid missed early T1D. If ketone ≥1, admit to start insulin (basal vs MDI up to clinician depending on BGL)

2. Metformin XR Titration:
Start at 500-1000 mg once daily and titrate by 500-1000 mg weekly to 2000 mg once daily
If side effects, consider reducing by 500 mg and slower titration
Metformin immediate release may be required in younger patients who require smaller doses and/or are not able to swallow tablets.

3. 2nd agent
Start other antidiabetes medications under the supervision of a paediatric endocrinologist within a multidisciplinary unit if the child is older than 10 years old. Weekly GLP1RA is the preferred choice (after careful consideration of phenotype and accurate diagnosis), with SLGT2 inhibitor treatment to be be considered if GLP1RA is contraindicated or not tolerated, may be particularly considered in renal impairment..

DKA = diabetic ketoacidosis; HHS = hyperglycaemic hyperosmolar state

Complications and comorbidities

The evaluation and management of complications and comorbidities are summarised in Table 4. Cardiovascular disease (not included in Table 4) is another diabetes complication that will manifest with cardiovascular events in adulthood. Cardiovascular risk factors such as increased weight, increased waist circumference, dyslipidaemia, hypertension, diet, activity and glycaemia should be addressed early to prevent cardiovascular disease later in life. Waist-to-height ratio and elevated urinary albumin creatinine ratio are useful markers to identify youth with higher cardiovascular risk factors[217].

Complications and/or comorbidities are often present at diagnosis. In a large cohort of youth with T2D in Hong Kong, 37.9% had MASLD, 35.3% dyslipidaemia, 22.5% hypertension and 12.8% microalbuminuria at diagnosis[218]. Similarly, the TODAY study found that 13.6% had hypertension, 79.8% had a low high-density lipoprotein level, 10.2% had high triglycerides and 13.0% had microalbuminuria just 7 months after diagnosis[219]. Data from the TODAY follow-up study at 15 years showed that 60.1% had at least one complication[12].

The SEARCH observational study revealed higher prevalence rates for albuminuria, retinopathy, neuropathy, arterial stiffness and hypertension in adolescents with T2D compared to those with T1D of similar diabetes duration (7.9 years) [19.9 vs 5.8%, 9.1 vs 5.6%, 17.7 vs 8.5%, 47.4 vs 11.6% and 21.6 vs 10.1% respectively][13].

Despite the documented increased multiple comorbidities/complications in YOT2D and the availability of several published management guidelines[19, 20, 64, 114], screening for comorbidities/ complications continues to be suboptimal[220].

Retinopathy

A meta-analysis of 27 studies (5924 individuals diagnosed with T2D before 21 years of age with a range of diabetes duration from 1-15 years) found a 7% prevalence of diabetic retiopathy (DR). This increased with diabetes duration (1% at <2.5 years, 9% at 2.5- 5 years and 28% at >5 years) with no differences in prevalence according to gender, race or obesity.

The SEARCH study found a 56% prevalence of DR in youth with T2D after 12.5 years of follow-up [221]. Similar prevalence of DR in YOT2D was shown in the TODAY study after 12 years of follow-up [222]. Risk factors associated with progression of DR include higher HbA1c, blood pressure, triglycerides, presence of hypertension or albuminuria, decreased c-peptide and longer diabetes duration[221, 222], with the most important predictor being HbA1c[221, 223].

Nephropathy

Microalbuminuria was found in 21% of adolescents with T2D in a meta-analysis, and macroalbuminuria in 3.9%, with higher prevalence in Pacific Peoples, First Nations American and Asian populations[224]. Similarly higher incidence of albuminuria was reported in Indigenous Australians and the highest incidence of macroalbuminuria and end stage kidney disease was reported in the Pima Indian population in the United States[225]. The TODAY study showed a 54.8% cumulative incidence of albuminuria after 15 years[226]. Albuminuria during adolescence is predictive of the future risk of chronic kidney15 and cardiovascular disease[227].

Peripheral neuropathy

The prevalence of peripheral neuropathy was reported as 31.5%[226] in a meta-analysis while a smaller Australian cohort study reported a prevalence of 47% only 1.7 years after diagnosis[227]. The TODAY study found neuropathy increased from 1% to 32.4% over 15 years follow-up[228]. Prevalence also increased from 19% to 36% according to diabetes duration (5-10 years vs >10 years duration) in the SEARCH study[229].

Cardiac autonomic neuropathy

There is no unifying definition of cardiac autonomic neuropathy and prevalence varies between studies, with the SEARCH study reporting a 17% in youth with T2D[230] while the TODAY study reported 8% at 7.7 years post-diagnosis[231]. Although these measures are typically used only in research studies, cardiac autonomic neuropathy has been associated with aortic stiffness in youth with T2D and an increased risk of cardiovascular events across all diabetes subtypes[232, 233].

Overweight and obesity

Obesity is a major contributor to T2D, with up to 97% of youth with T2D having obesity[234]. Obesity is defined as BMI Z score ≥2 though various adiposity measures are being explored in different ethnic groups. Weight management is essential for managing YOT2D to optimise glycaemia and overall health, with lifestyle management critical (see Nutrition section). GLP1-RAs are effective for weight loss in youth[235]. Bariatric surgery may also offer long-term benefits, with studies showing high remission rates for YOT2D[236-238] and associated comorbidities including kidney disease (Appendix Table 5). However, considerations to take into account include consent issues, adequate family support, ongoing multivitamin supplementation, contraception advice for females, lack of multidisciplinary teams with expertise in paediatric bariatric surgery attached to transition services in Australia and Aotearoa New Zealand and availability in the public sector[239].

Female reproductive health: Polyendocrine metabolic ovarian syndrome and contraception

There is a bidirectional relationship between polyendocrine metabolic ovarian syndrome (PMOS)[240] and T2D. PMOS is more prevalent in adolescents with T2D (2-5 times higher than in youth without T2D)[241-243]. It is important to screen pubertal girls with T2D for PMOS, especially if they have irregular menstrual cycles[19]. Additionally, there is increased risk of T2D in women with PMOS, regardless of BMI, thereby necessitating regular glycaemic screening[244]. PMOS diagnosis during adolescence should be based on the presence of both menstrual irregularities (including primary amenorrhoea) >3 years post menarche and hyperandrogenism (hirsutism, severe acne and/or total/free testosterone) after exclusion of other conditions that mimic PMOS[244-246]. Pelvic ultrasound or anti-mullerian hormone levels are not recommended for diagnosis of PMOS in youth within 8 years post menarche[244-246]. Youth that present with only one of these features (menstrual irregularities or hyperandrogenism) can be considered "at risk" for PMOS and require management of symptoms and ongoing follow-up[244].

Contraception should be discussed as part of regular review and is especially required when starting any potential teratogenic medications (ACE inhibitors, ARB, statins, SGLT2-i, GLP1-RA). It is also important to consider that ovulatory function may improve with metformin and/or weight loss and cause unplanned pregnancy. Any hormonal contraception method can be prescribed for youth with T2D without severe obesity, diabetes complications or other cardiovascular risk factors. For reliability, long-acting reversible contraceptives such as subdermal implant or intrauterine devices are recommended. Progestin-only

methods (pills, implants, intrauterine devices) or non-hormonal intrauterine devices may be used if the youth has severe obesity (BMI>35 kg/m2) and/or have severe hypertension[247]. Pregnancy and diabetes risks should be discussed at every visit in female youth with T2D.

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)

Previously known as non-alcoholic fatty liver disease (NAFLD), is characterised by hepatic steatosis in addition to one of; excess adiposity, presence of prediabetes or T2D, or evidence of metabolic dysregulation (high triglycerides, low HDL cholesterol, high systolic blood pressure)[248].

A meta-analysis reported a 45% prevalence in paediatric obesity clinics and 35.3% in youth with overweight or obesity[249]. Children with MASLD have higher prevalence of glucose abnormalities and increased risk of T2D in young adulthood[250, 251 252].

GLP1-RAs are of benefit, improving liver function and steatosis and possibly reducing fibrosis progression[253]. Metformin improves liver enzymes but effect on liver histology is inconclusive[253]. Pioglitazone is increasingly used off-label in YOT2D[53] in light of possible improvement in insulin sensitivity and MASLD potentially outweighing the theoretical risks of heart failure and reduced bone mineral density previously seen in adults with T2D.

Obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is prevalent in adults with prediabetes and T2D, but data in adolescents is limited. Poor sleep in adolescents with T2D is associated with worse metabolic control[129, 254], and should be monitored. Weight management is recommended, and use of GLP1-RAs may assist[255].

Hypertension and dyslipidaemia

Hypertension is common in youth with T2D, with 34% prevalence reported in a meta-analysis[224] and 19.2% prevalence at baseline in the TODAY study[228]. The TODAY study showed a 67.5% and 51.6% cumulative incidence over 15 years of hypertension and dyslipidaemia respectively[228]. Deteriorating glycaemic management is associated with deteriorating lipid profile[256, 257].

Clinical practice:

Complications and comorbidities are common at diagnosis, and young people are at high risk for development within a short timeframe from diagnosis. Complications should be screened for annually, more frequently if there is clinical concern.

Table 4: Evaluation and management of complications and comorbidities in youth living with T2D

Complication/ Comorbidity	Evaluation at diagnosis (thereafter yearly unless abnormal or specified)	Management	Management goals	Other considerations
Retinopathy	Dilated pupil exam or retinal photograph with a non-mydriatic camera by optometrist or ophthalmologist. (note early stages are asymptomatic).	According to retinal findings by optometrist/ ophthalmologist	Reduce/stop progression of proliferative retinopathy Preservation of vision	Optimise diabetes and weight management; and treatment of dyslipidaemia/ hypertension if present
Nephropathy Micro-albuminuria and macroalbuminuria	Proper collection of 3 early morning urine samples for albumin creatinine ratio (random samples if unable to do morning). Review if any interference with urine sample (contamination, menstruation, prior exercise, orthostatic proteinuria or infections). Renal function assessed by estimated glomerular filtration rate. (Of note early stages are asymptomatic).	Angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB) after confirming 2 urine samples >30 mg/g [3 mg/mmol] (microalbuminuria) Refer to renal if significant albuminuria (>300mg/g - macroalbuminuria) or hypertension to evaluate different aetiologies Consider adding SGLT2Is for diabetes management	Maintain normal renal function, urine albumin creatinine ratio and blood pressure	Optimise diabetes and weight management; and treatment of dyslipidaemia/ hypertension if present
Peripheral neuropathy	Foot examination, ankle reflexes, vibration sensation testing using128-Hz tuning fork pin prick sensation, and 10 g monofilament pressure (distal plantar aspect of great toes and metatarsal joints) Michigan neuropathy screening instrument	Foot care education and podiatry input Refer to neurologist for further evaluation if abnormal neurological signs (note painful neuropathy is rare in youth)	Individualised according to symptoms and signs	Optimise diabetes and weight management; and treatment of dyslipidaemia/ hypertension if present
Cardiac autonomic neuropathy	Review heart rate for resting tachycardia and blood pressure and postural hypotension (a drop of at least 20 mm Hg in systolic blood pressure and 10 mm Hg in diastolic blood pressure within 3 minutes of standing)	Individualized according to symptoms and signs Referral to cardiology	Individualised according to symptoms and signs	Optimise diabetes and weight management; and management of other cardiovascular risk factors if present
Overweight and obesity	Family history of obesity and other modifiable risk factors including environment. Plot BMI according to	Healthy lifestyle and family involvement See also section on Nutrition Reinforce adherence to metformin and GLP-1RA.	Small changes in BMI can improve comorbidities and are easier to achieve. BMI closer to	Possible use of newer treatments for T2D that improve weight Bariatric surgery in

Complication/ Comorbidity	Evaluation at diagnosis (thereafter yearly unless abnormal or specified)	Management	Management goals	Other considerations
	age and gender (Overweight BMI 85-95 percentile and obese BMI>95 percentile). * Identify other comorbidities related to obesity (also listed below) *	If on insulin optimize management to avoid further weight gain and consider other medications for treatment of diabetes Treat other obesity comorbidities	healthy range is ideal, as weight has impact on other comorbidities	selected cases with special considerations (see text)
Psychosocial Depression Diabetes distress Diabetes stigma High risk behaviours Eating disorders See also section on Psychological health	Quick screening tools for depression and/or referral for formal evaluation if suspected using PHQ-2 or HEEADSSS assessment for adolescents [H (Home environment), E (Education and employment), E (Exercise and healthy eating), A (Activity, include screen/ awareness of electronic device use; and peers), D (Drugs, smoking, vaping and alcohol), D (Depression and suicide ideation), S (sexuality and sexual health), S (Sleep)] Evaluation every visit according to general impression on review *	Early referral to mental health professionals, community health workers and school counselling Discuss involvement with peer support groups	Should be individualised but aiming to improve general emotional well-being and social functioning (school/family)	Presence of psychosocial disorders affects medication adherence and increases overall morbidity
Reproductive health Polyendocrine Metabolic Ovarian Syndrome Contraception	Menstrual cycle regularity according to time post menarche in pubertal girls * If menstrual irregularities are present evaluate hyperandrogenism: Clinical hyperandrogenism (hirsutism using Ferriman Gallwey score and/or moderate to severe acne, and/or androgenic alopecia) Biochemical hyperandrogenism (Testosterone levels, Free Androgen Index and Sex Hormone Binding Globulin) Diagnosis of adolescent PMOS requires 2 criteria menstrual irregularities and hyperandrogenism (clinical and/or biochemical).	Treat according to symptoms and recent international evidence-based guidelines for PMOS. Combined oral contraceptive if irregular menstrual cycles and/or hirsutism but consider BMI when assess benefit/risk. Metformin if not on it Cosmetic therapies for treating hirsutism	Individualized according to main complaining symptoms	Weight management Discuss/educate about contraception. Contraception is required if on potential teratogenic medications (ACE inhibitors, ARB, statins, newer oral /injectable agents). Also consider potential for improved ovulatory function with weight loss/ metformin and possible unplanned pregnancy.

Complication/ Comorbidity	Evaluation at diagnosis (thereafter yearly unless abnormal or specified)	Management	Management goals	Other considerations
	If only one criterion is present, consider adolescent at risk of PMOS Pelvic ultrasound or antimullerian hormone are not required for diagnosis of PMOS during adolescence Discuss pregnancy risk related to hyperglycaemia in pregnancy			
Metabolic Dysfunction-Associated Steatotic Liver Disease MASLD	Assess liver enzymes (Aspartate aminotransferase, gamma-glutamyl transferase and alanine aminotransferase)	Refer to gastroenterologist if liver enzymes > 3 times upper limit of normal Consider adding GLP1-RA if not on this medication for diabetes	Individualized according to liver enzymes	Optimize diabetes and weight management
Obstructive sleep apnoea Sleep quality	Evaluate symptoms of obstructive sleep apnoea (snoring, morning sleepiness and stop breathing episodes) *	Refer to respiratory team for oximetry and/or sleep study	Individualized according to sleep study results	Weight management Sleep apnoea is associated with dyslipidaemia, hypertension and insulin resistance. Consider use of GLP1-RA.
Hypertension	Blood pressure (BP) using appropriate cuff * Plot BP in centile charts for age, gender, height: Prehypertension (BP 90-94.9th centile if < 13 years or 120/80 to129/80mm Hg if ≥13 years) Hypertension Stage 1 (BP ≥95th or 130/80 mm Hg to <95th plus 12 mm Hg if < 13 years or 130/80 to 138/89 mm Hg if ≥13 years) Hypertension Stage 2 (BP ≥95th plus 12 mm Hg if < 13 years or ≥140/90 mm Hg if ≥13 years- whichever is lower) Ambulatory 24-hour BP monitors can be useful to confirm hypertension and assist with medication management	Healthy lifestyle and family involvement Use Dietary Approaches to Stop Hypertension (DASH) including Restriction of Sodium intake to about 1500 mg/day (< 1tsp/day) Start ACE inhibitors or ARB if hypertension is confirmed - starting at lower doses and ensuring contraception in sexually active adolescent girls.	BP < 90 percentile for age and gender if younger than 13 years or <120/80 if older than 13 years	Weight management Renal referral if hypertension stage 2, other pathology is suspected and/or treatment goals are hard to achieve. Review other risk factors for cardiovascular disease and manage accordingly

Complication/ Comorbidity	Evaluation at diagnosis (thereafter yearly unless abnormal or specified)	Management	Management goals	Other considerations
Dyslipidaemia High LDL cholesterol Low HDL cholesterol High triglycerides	Family history of dyslipidaemia Lipid profile (LDL [>3.36 mmol/L], HDL [<0.9 mmol/L] and triglycerides [>1.7 mmol/L) ideally fasting and when lower HbA1c is achieved	Healthy lifestyle and family involvement Reduce total dietary fat <35% and saturated fat <7% of total energy intake. Avoid trans-fat intake and limit saturated fats to be < 7% Introduce plant sterols into diet (if unable to achieve with whole foods consider stanol ester supplementation 2g/day) Increase fibre in diet through inclusion of vegetables, and complex carbohydrates Consider use of soluble fibre supplements, such as psyllium (Dose: 2-12yrs: 6g/day and >12yrs:12g/day) Statins if high LDL (>3.4 mmol/L) cholesterol despite improved lifestyle starting at a lower dose. Fish oil/fibrates if high triglycerides (>4.6 mmol/L)	Lipid profile 12 weeks after starting treatment to review levels: LDL cholesterol <2.6 mmol/L HDL cholesterol >0.9 mmol/L Triglycerides <1.7 mmol/L	Weight management Review other risk factors for cardiovascular disease and manage accordingly. If treatment targets not met on treatment, consider the possibility of familial hypercholesterolaemia. Acknowledge as yet limited evidence for lipid targets in this age group.

* Evaluation should occur at each visit

Transition to adult services

Although there may be differences across services and countries, transition from paediatrics to adult services typically occurs by 15 to 18 years, with possible further transition from a young adult service around age 25 years.

The transition period is a time of vulnerability for youth with T2D, with increasing independence, geographical relocation, high risk behaviours and competing social, financial and/or educational demands impacting on disease self-management[258].

For youth with T2D, the transition period has been associated with worsening glycaemia, low attendance at health care appointments and loss to follow-up[259]. Compared to youth with T1D, those with T2D have poorer clinic attendance both before and after transition, are more likely to be admitted to hospital, and are less likely to transition successfully. In high income countries, T2D in youth is often associated with socioeconomic disadvantage, lower rates of high school completion and higher rates of pregnancy during the transition period, factors that are likely to add to the complexity of the transition process[260].

There can be marked differences between paediatric and adult care[259]. Given that T2D is an aggressive disease in youth[12], transition should occur to age-appropriate specialist care, with the transition process started early in adolescence[20]. Where possible, emerging adults should be transitioned to a diabetes team with appropriate training and exposure to young adults and ideally comprised of an endocrinologist, dietitian, diabetes educator, psychologist and exercise physiologist[19, 20, 22, 259]. This is in contrast to older adults with T2D who are typically managed in primary care. The recommendation to transition to a specialist service does not preclude the role of the General Practitioner who provides critical additional support and ensures continuity of care during transition periods. The General Practitioner plays an important role in coordination of care, ongoing medication prescribing, monitoring for comorbidities and complications, supporting social and emotional wellbeing, and building trust and engagement with health services. For youth who live in remote areas without access to a multidisciplinary team, we recommend telehealth and/or case conferencing with a specialist service.

It is important that the transition process is culturally, socially and developmentally safe, noting that certain groups may be over-represented in YOT2D[1, 2, 5, 6, 47, 49]. Use of First Nations/ cultural health workers or health workers from relevant ethnic or community groups may improve the transition process. In addition, the needs of youth with developmental and/or intellectual disability should be recognised and appropriately accommodated.

Loss to follow-up is one of the greatest issues in transition[259, 260] and should be addressed in the transition model of care. It is also important for paediatric services to develop systems to monitor outcomes of young people after transition to other services and plan for a regular primary health care provider and pharmacy supplier. It is also important to continue to address pregnancy education and planning in all women. Given the aggressive nature of T2D in youth, even a seemingly short period out of care may significantly impact outcomes.

To improve engagement, we recommend considering novel models of care that include technology and support improved access to technology, particularly in rural and remote areas where this may be an issue. We recommend an emphasis on social support, including family members or key support figures. Web-based peer support programs are considered enablers in improving transition in youth with T1D and could be considered[261]. Where practical, the adult treating team should provide consistency of care, with experience managing issues relevant to youth. Pharmacological treatment options for T2D expand considerably after the age of 18 years and the transition process needs to support these changes with culturally and

developmentally appropriate education and follow-up. It is important to recognise the impact of mental health on the transition process, and the use of psychosocial screening or HEADSS assessments may identify areas to address and help teams to stratify resources. It is also important to consider strategies which overcome diabetes related stigma, address priorities of youth and improve engagement – examples include addressing PMOS and clinical hyperandrogenism in women and focusing on strength training and weight management in young men.

In future, improved understanding of the facilitators and barriers to transition of youth with T2D, along with collaboration between adult and paediatric care teams and development of policies and guidelines, will better inform and support the transition process.

Clinical practice:

Plan transition to adult services well, ideally to a multidisciplinary team with expertise in YOT2D. Consider strategies to maximise engagement and minimise loss to follow-up.

Standards of care

Effective implementation of these consensus guidelines relies on services being able to meaningfully review their practices and outcomes. All services should develop data collection and monitoring systems that allow regular audits of clinical care and outcomes of youth living with T2D. Clinical registries can be used to facilitate standardisation of care across regions through quality improvement initiatives aimed at improving models of care, collaboration between primary health care and paediatric diabetes services, and diabetes outcomes. Benchmarking to other services is an important continuous quality improvement activity and allows sharing of knowledge and experience between centres. Audits and benchmarking are iterative processes and assist in identifying opportunities for change. They have been shown to be effective in improving health outcomes of young people living with T1D[262-264], limited data as yet available in T2D. Benchmarking assists in reducing variability in clinical practice and allows identification of key transferable processes and practices that could lead to improved outcomes[265].

There are still large evidence gaps in understanding risk, phenotypes and trajectories of YOT2D, as well as targeting of pharmacological management for individual youth. Benchmarking across services may assist in establishing this evidence and improve models of care in YOT2D. It will also be important in identifying areas of inequity and disparity to allow a better understanding of need and facilitate effective advocacy for enhanced resources. All youth living with T2D in Australia and Aotearoa New Zealand deserve equitable and high-quality care, regardless of where they live.

Clinical practice:

All services should audit the clinical care and outcomes of youth with T2D, preferably benchmarking to other services.

Conclusion

These Australian and Aotearoa New Zealand guidelines for youth living with YOT2D and aged <18 years represent the first consensus guidelines from a collaboration of paediatric, diabetes, primary health care, consumer and research organisations. They offer comprehensive recommendations for health care providers on screening, diagnosis, diabetes education, monitoring (including targets), healthy lifestyle, pharmacotherapy, complications and comorbidity management, and transition of care.

The guidelines emphasise the unique challenges of caring for these youth, including strategies to enhance engagement and improve access to health care, and considerations related to newer medications, atypical phenotypes, social determinants, psychological health and wellbeing and intergenerational transmission of risk. Critically, they underscore that YOT2D is a distinct entity from adult-onset type 2 diabetes, with differing management needs, risk profiles and future trajectories. Recommendations are graded based on current evidence, highlighting the need for high quality, targeted research into YOT2D and benchmarking across services. To improve future health outcomes, the lived experience and voices of young people must meaningfully shape service design, research priorities and models of care.

APPENDIX

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Table 1: Terminology

Ethnicity	Where possible, the guidelines refer to evidence or recommendations relating to specific ethnicities. In reflection of evidence internationally regarding YOT2D risk and trajectories, the term First Nations is also used at some points to respectfully refer to young people of Aboriginal, Torres Strait Islander, Māori and/or Pacific Peoples ethnicity. The term 'high-risk ethnicities' is also used at some points to refer to young people of First Nations, South East Asian, Indian subcontinent, Middle Eastern or refugee backgrounds.	
Youth living with diabetes	The 2021 Diabetes Australia consensus statement[266] clearly highlights the impact of language in the diabetes trajectory of those living with T2D. In particular, we emphasise the importance of avoiding language that contributes to shame, stigma, disrespect, judgement and blame[116, 117]. It is also important to choose language that facilitates collaborative and positive relationships[266].	
	Instead of:	***Try:***
	Diabetic	Young person living with diabetes
	Patient	Young person
	Difficult/challenging patient	Has other priorities right now... Has concerns about... Finding it difficult...
	In denial	Finding it difficult...
	Obese	Living with obesity
	Adherence/ compliance	Describe the specific behaviours, understandings and solutions
	Control/ poorly controlled	Manage glucose levels Outside target range High glucose levels
	Good/bad	Numbers
	Failed on...	Medication X did not achieve target levels
	Testing	Monitoring or checking glucose levels
	Disease	Condition
Hyperglycaemia in pregnancy	Differing definitions of hyperglycaemia in pregnancy exist, with current definitions based on risk of adverse perinatal outcomes, as compared to subsequent risk of maternal type 2 diabetes as used previously[267, 268]. In 2025, the Australasian Diabetes in Pregnancy Society (ADIPS) published consensus recommendations for classification of gestational diabetes[269], acknowledging the evidence that the frequency of adverse pregnancy outcomes increase at glucose levels below the threshold for diagnosis of GDM[270, 271]. Regardless of gestation, gestational diabetes mellitus is defined as one or more of the following during 2 hour OGTT: fasting glucose level 5.3-6.9mmol/L; one hour glucose level ≥ 10.6mmol/L; and/or a two hour level ≥ 9-11mmol/L[269]. Regardless of gestation, overt diabetes in pregnancy is diagnosed if one or more of the following are met: a fasting glucose level ≥ 7.0mmol/L; two hour glucose level ≥ 11.1mmol/L on OGTT; and/or HbA1c ≥ 6.5% (≥ 48mmol/mol)[269].	

*Adapted from Speight J, et al. 2021. Our language matters: Improving communication with and about people with diabetes. A position statement by Diabetes Australia. DRCP; 173: 108655.

Table 2: National Health and Medical Research Council (NHMRC) and Grading of Recommendations Assessment, Development and Evaluation (GRADE) levels of evidence

GRADE definition and NHMRC levels of evidence	A	B	C	D
	Excellent	**Good**	**Satisfactory**	**Poor**
Definition	Body of evidence can be trusted to guide practice	Body of evidence can be trusted to guide practice in most situations	Body of evidence provides some support for recommendations but care should be taken in its application	Body of evidence is weak, and recommendation should be applied with caution
Evidence base	Several level I or II studies with low risk of bias	One or more level II studies with low risk of bias	Level III studies with low risk of bias, or level II studies with moderate risk of bias	Level IV studies or level I to III with high risk of bias
Consistency	All studies consistent	Most studies consistent and inconsistencies may be explained	Some inconsistency reflecting genuine uncertainty around clinical question	Evidence is inconsistent
Clinical impact	Very large	Substantial	Moderate	Slight or restricted
Generalisability	Populations studied in body of evidence are the same as the target population for the guideline	Populations studied in body of evidence are similar to the target population for the guideline	Populations studied in body of evidence differ to target population for guideline, but it is clinically sensible to apply this evidence to target population	Populations studied in body of evidence differ from target population and it is hard to judge whether it is sensible to generalise to target population
Applicability	Directly applicable to Australian health care context	Applicable to Australian health care context with few caveats	Probably applicable to Australian health care context with some caveats	Not applicable to Australia health care context
GRADE quality of evidence and definition	High quality. Further research is unlikely to change our confidence in the estimate of effect	Moderate quality. Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate	Low quality. Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate	Very low quality. Any estimate of effect is very uncertain

Table 3: Diabetes self-management education content

Education - at diagnosis and ongoing	Continuing curriculum
Simple explanation of how the diagnosis was made and the cause of symptoms. Reassure that treatment will help the child regain health and energy quickly	Pathophysiology, epidemiology and classification
Explore feelings of guilt, shame or blame and discuss the uncertain cause of diabetes	Explore understanding of youth as they mature
Normalise grief, anger and loss reaction to the diagnosis	Address psychological health and diabetes burnout/distress
Discuss risk for siblings and other family members; and interventions available to minimise risk	Revise as needed
Simple explanation of glucose and the relationship between food, blood glucose, metformin and insulin	Explain other sources of glucose, that is, liver glucose
Simple explanation of the effect of metformin, insulin (long and short acting), GLP1-RA and SGLT2Is on blood glucose levels	Metformin action and profile, Insulin action and profiles, other medications action and profiles. Adjustment of insulin If applicable, simple explanation of other available oral or injectable hypoglycaemic agents
Discuss the role and responsibility of family in the delivery and supervision of self-management tasks and expectation for frequency of follow-up. Explore family and collective views regarding health and diabetes.	Review individual roles at each visit and encourage active parental involvement Explore barriers to clinic attendance if missed appointments
Establish clear blood glucose levels and HbA1c targets; and goals of treatment	Self-monitoring blood glucose - monitoring frequency, HbA1c Weight loss, balanced diet Exercise/screen time/sleep Goal-setting focus on goals that are SMART: Specific, measurable, achievable, realistic, and time-based Micro- and macro-vascular complications, screening protocol, and prevention
Focus on basic "survival" skills needed to manage the diabetes from day one Accomplishment of these skills will increase the carers/youth's confidence in their ability to manage. Assess competence in: • Blood glucose self-monitoring • Taking metformin and/or other oral medications • Insulin devices: injection, pen • Insulin injection sites/techniques, storage • GLP1-RA injection sites/technique • Diabetes diary: what to record and how often • Insulin bolus calculator • Carbohydrate awareness, including portion sizes, and glycaemic index • CGMS if applicable	Revise these skills • As new devices or technologies are introduced • As youth take on self-management tasks • If diabetes needs stabilisation • In response to episodes of ketosis or severe hypoglycaemia • When new carers are introduced to the family • When child/adolescent is planning school camp/excursion • During transition to adult services • Whenever there are admissions other than due to diabetes
Basic dietetic advice including importance of healthy eating, portion sizes, and meal-time routines. Promotion of healthy body weight. Link between food and diabetes. Carbohydrate awareness and glycaemic index if on prandial insulin	Explain effects on blood glucose levels of different food components including proteins, fats, fibre, and glycaemic index; and discuss management strategies to optimise postprandial blood glucose levels Revise nutritional skills as the child grows and develops Adapt nutritional interventions in response to new diagnoses Screen for disordered eating

Education - at diagnosis and ongoing	Continuing curriculum
Explanation of hyperglycaemia and diabetes ketoacidosis (symptoms, prevention, management) If on insulin, explanation of hypoglycaemia (symptoms, prevention, management), identity cards, necklace, bracelets, and other equipment	Revise with introduction of new activities and new carers Practice reconstitution of glucagon Precautions with alcohol Precautions, recognising and treating hypoglycaemia when driving
Diabetes during illness Advice not to omit insulin and to call the diabetes team for advice May need to stop metformin, SGLTI2s or GLP1RA if dehydrated, ill or require surgery	Effects of intercurrent illness, hyperglycaemia, ketosis, and prevention and identification of diabetes ketoacidosis Diet and fluids on sick days Sick day management plan
Integration of diabetes self-management tasks into family life, social activities, sporting activities, and school	Problem-solving and adjustments to treatment in everyday life, motivation, and coping with unexpected glucose fluctuations Review and revise school management plan annually Exercise, holiday planning and travel, including educational holidays and camps
Address questions about impact on future aspirations for youth, that is, career, having children	Sexuality, pregnancy planning, contraception, employment Teratogenic effects of oral/injectable agents, especially newer medications for management of type 2 diabetes
Membership of a diabetes association, joining National Diabetes Service Scheme, Medic Alert bracelet	Explore opportunities for peer support and family support, connections to others with lived experience
Details of emergency telephone contacts and follow-up arrangements	Update as required

Adapted from ISPAD Clinical Practice Consensus Guidelines 2018 (ISPAD Clinical Practice Consensus Guidelines 2018: Diabetes education in children and adolescents. Phelan H, Lange K, Cengiz E, Gallego P, Majaliwa E, Pelicand J, Smart C et al. Pediatr Diabetes. 2018; 19 Suppl 27:75-83)

Available resources for youth living with type 2 diabetes

- Education materials and a lifestyle program used in Treatment Options for type 2 Diabetes in Adolescents and Youth (TODAY) study:

  https://portal.bsc.gwu.edu/web/today/tsdematerials

- Resources for youth to help promote lifestyle changes in type 2 diabetes:

  https://www.childrens.health.qld.gov.au/__data/assets/pdf_file/0027/382491/diabetes-wont-stop-me-healthy-kids-booklet.pdf

- Resources for Aboriginal and Torres Strait Islander youth living with type 2 diabetes, including lived experience and explanatory videos, as well as educational resources for health professionals:

  https://diabeteslifecourse.org.au/youth-diabetes/

- Resources for Aboriginal and Torres Strait Islander adults living with type 2 diabetes:

  https://healthinfonet.ecu.edu.au/learn/health-topics/diabetes/resources

- Nutritional resources for type 2 diabetes in children:

  https://www.kidshealth.org.nz/diabetes/type-2-diabetes-in-children-general-guidelines-on-nutritional-management

Table 4: Studies of glucose lowering pharmacotherapeutic agents for treatment of YOT2D

Note - Only clinical trials where the primary aim is to determine pharmacokinetics/ pharmacodynamics of the agent or glycaemic efficacy in YOT2D are presented. Studies in pre-diabetes or where the primary outcome is weight loss/BMI change or co-morbidity changes are not presented. NCT- Clinical Trials government identified

Drug class	BIGUANIDES
Formulation	Metformin[120, 202, 206, 211, 212, 272-279] Immediate Release (IR) Extended Release (XR) Dose 500mg - 2g daily
Route of administration	Oral. IR twice daily, XR once daily with food.
Mechanism of action	Decreases hepatic glucose output by inhibiting gluconeogenesis. Inhibits hepatic lipogenesis and increases fatty acid oxidation. Increases glucose uptake and fatty acid oxidation in skeletal muscle and adipose tissue. Decreases absorption of glucose from intestine. Promotes secretion of GLP-1 and PYY concentrations from the intestine[280]. Influences gut microbiome by promoting the growth of beneficial species. Excreted unchanged via kidneys.
Pharmacokinetic / pharmacodynamic studies in youth	Metformin 500mg twice daily (n=32) bioavailability 60%. Metformin 500mg, AUC 54% of that reported in adults. [273] *NHMRC Level II evidence* Single dose metformin in T2D (n=12)showed PK similar to healthy adults (n=12)[281]. *NHMRC Level III-2 evidence* Metformin dose 750mg daily (n=6) 9 year old non obese girls with low birth weight, PK similar to adults[282]. *NHMRC Level IV evidence*
Precautions	Not to be used in eGFR of less than 30 ml/min/1.73 m^2. Withhold before surgery. Withhold before and 48hrs after radiographic contrast. Withhold in acute illness, risk of dehydration, hypoxemia. Safe during pregnancy Safety profile like adults Slow escalation of dose to minimize side effects.
Side effects	Gastrointestinal; abdominal pain/nausea/diarrhoea/bloating and loss of appetite- common in both IR and XR formulations.[206, 279] Headache B12 deficiency, risk increases with duration of exposure [283]. Lactic acidosis with concurrent illness or dehydration (rare).
Clinical trials in T2D and HbA1c outcomes	RCT, 16 weeks (n=82), Metformin 1grm twice daily versus placebo. HBA1c difference between metformin and placebo was -1.2% in favour of metformin p<0.001[273], *NHMRC Level II evidence* TODAY study pre-randomization phase of 2 months of metformin (n=927) starting HbA1c mean 7.7±2.2, 77.9% achieved <7% at 2 months, 8.5% intolerant of metformin. [202] *NHMRC Level IV evidence* TODAY study RCT (n=699), mean 3.8 years. Metformin alone versus, metformin plus lifestyle versus metformin plus rosiglitazone.49.3% of youth on metformin alone and 53.4% on metformin plus intensive lifestyle maintained HbA1c <8.5% (12). *NHMRC Level II evidence* RCT (n=285) of glimepiride vs metformin 26 weeks, Reduction of HbA1c on metformin group -0.71% p=0.0002, no difference between metformin and glimepiride [274] *NHMRC Level II evidence*
Impact on BMI, weight, metabolic	Reduction in BMI and body weight[273]. 30% of youth in TODAY on metformin alone or metformin plus lifestyle had >7% weight loss[278]. Improved lipids[273]
Trials currently underway	Phase I, PRECISE_T2D study comparing metformin 1000mg twice daily with 1350 mg twice daily. ECD Dec 2025. NCT06120881

Drug class	BIGUANIDES
TGA, PBS Medsafe	TGA approved ≥10 years T2D PBS general schedule Pregnancy category C
Recommended use in YOT2D	Initial treatment and ongoing. Can be used in combination with all other drug classes.

TGA Therapeutic Goods Administration (Australia) PK pharmacokinetic PD pharmacodynamic

Drug class	INSULIN
Formulation	Glargine (2 strengths)[212] Detemir[208] Aspart +aspart protamine [284] Aspart Isophane Degludec / aspart Glulisine Lispro+ lispro protamine Dose variable
Route of administration	S/C injection, Single or multiple times daily CSII
Mechanism of action	Binds to insulin receptors to increase glucose uptake and to inhibit hepatic glucose output. Insulin metabolized by liver and cleared by the kidneys.
PK PD studies in youth	
Precautions	Reduce insulin dose to prevent symptomatic hypoglycaemia when new agents are commenced.
Side effects	Hypoglycaemia, severe hypoglycaemia rare[208, 285]. Weight gain, younger age higher weight gain[286] Oedema Hypersensitivity at injection site Generalised hypersensitivity to insulin (rare)
Clinical trials in T2D and HbA1c outcomes	RCT (n=91) of glargine (3mths) followed by metformin (9mths) versus metformin (12mths). Neither treatment halted β-cell decline [212]. *NHMRC Level II evidence* Pilot study (n=18) pre-mix 30/70 insulin started mean 2 months after diagnosis, for a mean of 8.7±4.3 weeks HbA1c decreased from 12.81% to 7.59%, improvement persisted for at 12 months after ceasing insulin [284]. *NHMRC Level IV evidence* Detemir or NPH insulin +metformin (n=42) 26 weeks Detemir HbA1c -0.61% and -0.84% in NPH group, no difference between groups [208]. *NHMRC Level II evidence*
Impact on BMI, weight, metabolic	BMI increase in insulin group compared to metformin across the 12-month intervention (p=0.008) RISE Pediatric Medication Study[212]. No significant change in BMI [284].
Trials currently underway	Phase I trial long-acting Insulin (Insulin Icodec). ECD Jan 2026. NCT05790681 Phase I, PK study of LY3209590. ECD Nov 2026. NCT06362265
TGA, PBS Medsafe	TGA approved PBS general schedule Pregnancy category B
Recommended use in YOT2D	Initial treatment in DKA or HHS and as rescue treatment in times of glycaemic decompensation. In insulin deplete T2D.

Drug class	GLP-1 RECEPTOR AGONISTS (GLP1-RA)[287]
Formulation	Exenatide[288, 289] - discontinued Liraglutide [213, 290] s/c 0.6-1.8mg daily Dulaglutide [291] s/c 1.5 mg wkly Lixisenatide[292]s/c 10-20 µg daily Semaglutide s/c 0.25-1mg wkly/ oral 3-14 mg daily
Route of administration	s/c inj daily, weekly semaglutide oral daily (not available in Aus or NZ)
Mechanism of action	Glucagon-like peptide-1 (GLP-1), stimulates glucose dependent insulin secretion. Increases intracellular Ca2+concentration through calcium channels on the endoplasmic reticulum enhancing insulin secretion. Suppresses glucagon directly and indirectly via α-cell inhibition and stimulating somatostatin secretion. Promotes glycogen synthesis in liver cells. Activating multiple nuclei of hindbrain and hypothalamus suppressing appetite. Delays gastric emptying, slows peristalsis of gastrointestinal tract results in increased satiety. Degraded by DPP-4 and other enzymes and eliminated by the kidneys.
PK PD studies in youth	Exenatide (n=13) single dose 2.5, 5 µg (n=13). Dose-dependent increases in plasma concs. 2.5 µg dose achieved low plasma concs, 2.5µg may not be a high enough dose in T2D. [293]. *NHMRC Level III-1 evidence* Liraglutide 0.3- 1.8 mg (n=14). PK was comparable to adults at all doses. Lower exposure with higher weight, lower exposure in males[290]. *NHMRC Level II evidence* Lixisenatide (n=23) PK PD, once daily in escalating doses over 6 weeks 5-20 µg showed dose-dependent drug levels rise. 70% developed anti-drug antibodies [292] *NHMRC Level II evidence*
Precautions	Anaesthetic services should be informed in cases of elective endoscopic or surgical procedures regarding the use of GLP-1RA. GLP-1RA may be withheld for some procedures/surgeries. Use with caution if history of pancreatitis. Reduction of insulin dose may be required when starting GLP-1 agonist. Family or personal Hx of MEN2A, MEN2B or medullary thyroid cancer GLP-1RA contraindicated. Safety in pregnancy unknown Safety profile like adults
Side effects	Acute pancreatitis GI effects (nausea, vomiting, diarrhoea). Diarrhoea more prevalent than nausea[294]. Mild hypoglycaemia. Potential increased risk of medullary cell carcinoma.
Clinical trials in T2D and HbA1c outcomes	RCT (n=83) 24 weeks of Exenatide 2mg s/c once weekly versus placebo +/- metformin, sulphonylurea or insulin. Resulted in difference of HbA1c -0.85% (p=0.012) in favour of exenatide.[289] *NHMRC level II evidence* Ellipse RCT trial (n=134) liraglutide + metformin versus placebo at 26 and 52 weeks. Difference in HbA1c change at 26 weeks was -1.1% in favour of Liraglutide (p<0.001) Liraglutide group 63.7% compared to 36.5% of control group achieved an HbA1c less than 7.0% (p<0.001)[213]. *NHMRC level II evidence* RCT Liraglutide versus placebo (n=19) 5 weeks. Difference in HbA1c change was -0.9 % in favour of Liraglutide. (p=0.0007)[294]. No major side effects[294]. *NHMRC level III-1 evidence* AWARD-PEDS RCT (n=154) 26 weeks Dulaglutide 0.75 mg, 1.5 mg versus placebo, participants on metformin +/- basal insulin. Difference between groups was -1.4 % HbA1c in favour of dulaglutide pooled data (p<0.001). HbA1c of ≤6.5% met by 47% of dulaglutide group and 13% of placebo [291]. *NHMRC level II evidence*

Drug class	GLP-1 RECEPTOR AGONISTS (GLP1-RA)[287]
Impact on BMI, weight, metabolic	Exenatide, Dulaglutide not superior to placebo for BMI reduction[289, 291] Liraglutide, superior BMI reduction seen in ELLIPSE trial at 52 weeks [295]*. Systematic review of GLP-1 agonists found Liraglutide BMI reduction is greater in youth obesity than T2D[296]. Improvement in lipid profile with dulaglutide and liraglutide (26 weeks) [213].
Trials currently underway	Phase III trial of oral semaglutide versus placebo. Both in Combination with Metformin and/or basal Insulin. ECD Mar 2026. NCT04596631. Semaglutide, s/c weekly. Effect on Beta-cell preservation and reduction of intrahepatic triglyceride content in obese youth with IGT/early T2D and NAFLD. *NCT05067621* ECD Jan 2027. Phase III trial of Dulaglutide 3.0 mg, 4.5mg. ECD Jul 2027 NCT06739122
TGA, PBS Medsafe	Exenatide, Semaglutide, Liraglutide, Dulaglutide all, TGA approved for T2D ≥ 18 years (*Semaglutide TGA approved for obesity ≥12 yrs)* Semaglutide, Dulaglutide on PBS for T2D, criteria based, authority required Pregnancy category D
Recommended use in YOT2D	Second line agent when glycaemic targets not met. Not to be used in combination with a DPP-4 inhibitor.

*Daily Liraglutide studies in T2D use a maximum dose of 1.8 mg weekly, doses of up to 3.0 mg are used in weight loss studies in youth [297]. In addition 50% of participants in the ELLIPSE study did not reach maximum dose due to dose adjustment based on achieving fasting glucose target, this may have prevented additional weight loss in the liraglutide group and explain the better weight loss seen in youth with obesity compared to T2D [213]. Long term data is lacking in children to determine long-term efficacy and adverse effects. Studies in adults show favourable effect on cardiovascular and renal outcomes, with TGA approval in adults for these benefits.

Drug class	DUAL INCRETINS
Formulation	Tirzepatide 2.5-15 mg
Route of administration	s/c injection weekly
Mechanism of action	Dual incretin with GLP-1 agonist and GIP. GLP-1 effect same as GLP-1 agonist. GIP activates the GIP receptor stimulating insulin secretion from the pancreas lowering blood glucose and promoting satiety by slowing stomach emptying. Degraded by DPP-4 and other enzymes and eliminated by the kidneys.
PK PD studies in youth	
Precautions	Same as GLP-1RA
Side effects	Acute pancreatitis GI effects (nausea, vomiting, diarrhoea) Potential increased risk of medullary cell carcinoma
HbA1c outcomes	SURPASS-PEDS[298] compared to placebo of metformin and/or insulin. At week 30, tirzepatide superior to placebo in reducing HbA1c, with mean reduction of 2.28% in pooled tirzepatide group (95% CI −2.87 to −1.69; p<0.0001). Glycaemic efficacy sustained up to 52 weeks.
Impact on BMI, metabolic	Significant reductions in BMI of 7.4% (5mg) and 11.2% (10mg) at 30 weeks, vs 0.4% in placebo. At week 52, BMI decreased by 8.9% (5 mg) and 15.1% (10 mg). Waist circumference, triglycerides, LDL, and blood pressure improved relative to placebo.
Trials currently underway	SURPASS-PEDS Phase III trial of Tirzepatide versus placebo[298]. (Clinicaltrials.gov NCT05260021)
TGA, PBS Medsafe	TGA approved for T2D, obesity ≥18 years (note FDA approval in ≥10 years for T2D). Not PBS listed Pregnancy category D
Recommended use in YOT2D	Not as yet

Drug class	SGLT-2 INHIBITORS (SGLT2i)
Formulation	Empagliflozin[299, 300] 5-25mg daily Canagliflozin[305]100-300mg daily Dapagliflozin[301-304] 5-10mg daily Ertugliflozin 5-15 mg daily
Route of administration	Oral once daily with or without food (except canagliflozin – taken in morning before food)
Mechanism of action	Blocks the sodium-glucose cotransporter 2 protein in the proximal convoluted tubules of the kidney, preventing the reabsorption of glucose at the proximal tubule. The glucose is excreted in the urine along with sodium and an accompanied diuresis. Undergo biotransformation by UGT, excreted via the kidneys.
PK PD studies in youth	Dapagliflozin single dose 2.5, 5 or 10mg (n=24) PK/PD like adults.[301]. *NHMRC level III-1 evidence* Empagliflozin 5, 10 and 25mg dose (n=27). Maximum plasma level lower in youth for both 10 and 25 mg doses. AUC slightly lower in 10mg and slightly higher for 25 mg in youth compared to adults[299]. *NHMRC level evidence* Canagliflozin 100-300mg (n=17) PK PD like adults. Renal threshold reductions achieved consistent with reductions seen in adults[305]. *NHMRC level III-1 evidence*
Precautions	Do not use in states of volume depletion. Withhold before surgery, prolonged fasting, critical medical illness. Use with caution with history of urinary tract infections. Use with caution in renal insufficiency. Not safe in pregnancy Safety profile like adults
Side effects	Dehydration Euglycaemic diabetic ketoacidosis (rare)[306] Genitourinary infections Headache Nasopharyngitis Hypoglycaemia when used with insulin
HbA1c outcomes	DINAMO RCT Linagliptin, empagliflozin, versus placebo 26 weeks and safety extension 52 weeks (n=158). Difference in HbA1c between pooled empagliflozin data and placebo -0.84% p=0.012 in favour of empagliflozin.[300] *NHMRC level II evidence* Dapagliflozin versus saxagliptin or placebo, on metformin +/-insulin (26 weeks) (n=245). HbA1c difference at week 26 -1.03 % p<0.001, at 52 weeks difference sustained -1.19 % p<0.001 in favour of dapagliflozin[303]. *NHMRC level II evidence* Dapagliflozin T2NOW versus placebo RCT 24 weeks, (n=72) double blind and 28-week open label. HbA1c difference at 24 weeks -0.75% p=0.101. In per-protocol population difference -1.13% p=0.012[304]. *NHMRC level II evidence*
Impact on BMI, metabolic	No BMI, weight or BP change with dapagliflozin[304]. It is unclear as to whether SGLT2 inhibitors will confer similar renal and cardiac benefits as observed in adults.
Trials currently underway	Phase III trial of Ertugliflozin versus placebo. ECD Jul 2025. NCT04029480
TGA, PBS Medsafe	Empagliflozin T2D ≥10 years. Dapagliflozin T2D ≥18 yrs. Also approved for chronic heart failure (CHF) and chronic kidney disease (CKD) in ≥18 yrs. Empagliflozin, Dapagliflozin PBS listed for T2D, CHF, CKD criteria based authority required Pregnancy category D
Recommended use in YOT2D	Second or third line when glycaemic targets not met. Particularly consider if renal insufficiency.

UGT uridine 5-diphosphate-glucuronosyltransferases

Drug class	DPP-4 INHIBITORS[287]
Formulation	Sitagliptin[307-309] 100 mg Alogliptin[310, 311] 25 mg Saxagliptin[303] 5mg Linagliptin[300] 5mg Vildagliptin 50-100mg
Route of administration	Oral once daily with or without food
Mechanism of action	DPP-4 inhibitors, block the action of the enzyme DPP-4 that degrades GLP-1 and GIP, raising the levels of these incretin hormones. Do not have the same effect on satiety, gastric emptying and weight loss as GLP-1 agonists Eliminated by the kidneys, except saxagliptin which is metabolized by the liver cytochrome P450.
PK PD studies in youth	Sitagliptin single dose 50, 100, 200mg (n=35). PK comparable to adult data. DPP-4 inhibition lower in youth than adults, excretion comparable, no adverse events[307]. *NHMRC level II evidence* Alogliptin single dose 12.5, 25 mg (n=23 youth, 22 adults). 25mg dose, peak plasma conc 26% and AUC 23% lower in youth than adults, maximum DPP-4 inhibition effect same as adults. No safety concerns. 12.5 mgs dose suboptimal in youth.[310] *NHMRC level III-1 evidence* Linagliptin single dose 1, 5mg n=39, mean DPP-4 inhibition 38% and 79% with 1 and 5mg, higher serum levels of drug found but more bound compared to adults, DPP-4 inhibition activity lower than in adults[312].* *NHMRC level II evidence*
Precautions	Dose adjustment required for moderate or severe renal insufficiency. Do not use in combination with GLP-1RA Not safe in pregnancy Safety profile like adults
Side effects	Acute pancreatitis GI effects, nausea, vomiting
HbA1c outcomes	Sitagliptin versus placebo RCT (n=220) added to metformin +/- insulin, 54 weeks. HbA1c difference -0.49% p=0.018 in favour of sitagliptin at 20 weeks, not sustained at 54 weeks.[308] *NHMRC level II evidence* Sitagliptin vs placebo RCT (n=190) as first line treatment for 20 and 54 weeks. HbA1c difference btwn groups -0.29% in favour of sitagliptin (but NS)[309] *NHMRC level II evidence* Alogliptin versus placebo RCT (n=152) treatment naïve or added to metformin +/- insulin, 26 weeks. HbA1c difference between groups +0.1% in favour of placebo but not significant[311]. *NHMRC level II evidence* Saxagliptin, dapagliflozin or placebo RCT (n= 245) 26 and 52 weeks HbA1c difference between saxagliptin and placebo -0.44 % in favour of saxagliptin but not significant[303]. *NHMRC level II evidence* DINAMO RCT n=158 Linagliptin 5mg, empagliflozin 10, 25 mg versus placebo for 26 weeks and safety extension 52 weeks. Linagliptin HbA1c difference from placebo -0.34% not significant[300]. *NHMRC level II evidence* Linagliptin 1, 5 mg versus placebo (n=39) 12 weeks. HbA1c difference in favour of linagliptin -0.63% for 5mg and -0.48% with 1 mg, both not significant[312]. *NHMRC level II evidence*
Impact on BMI, metabolic	Sitagliptin and linagliptin, BMI, weight changes. No BP changes[308, 312].
Trials currently underway	
TGA, PBS, Medsafe	Sitagliptin, Alogliptin, Saxagliptin, Linagliptin, VildagliptinTGA approved T2D≥18 years Sitagliptin, Alogliptin, Saxagliptin, Linagliptin, Vildagliptin PBS listed criteria based, authority required Pregnancy category B
Recommended use in YOT2D	Efficacy limited in T2D, not recommended as first or second line. Not to be used in combination with GLP-1 agonist

*With GLP-1RA showing superior HbA1c lowering compared to DPP4 inhibitors it is suggested that a much higher and sustained increase in GLP-1 concentrations is needed than what the DPP4 inhibitors can achieve.

Drug class	THIAZOLDINEDIONES (GLITAZONES)
Formulation	Rosiglitazone[120, 206, 211, 275-278] 2-8mgs daily Pioglitazone[313] 15-45 mg daily
Route of administration	Oral once daily with or without food
Mechanism of action	Improves insulin sensitivity in adipose tissue, muscle and liver via activation of the peroxisome proliferator-activated receptors (PPARγ). Metabolised in the liver by CYP 2C8 and CYP 3A4.
PK PD studies in youth	Rosiglitazone single dose of 2, 4 mgs, predicted AUC based on twice daily dosing same as adults [314]. *NHMRC Level II evidence* Pioglitazone single and multiple dose 15, 30, 45 mg (n=36). PK similar to adults.[313].
Precautions	Increased risk of myocardial infarction and increased risk of congestive heart failure[315]. Increased risk of fractures in adults. Contraindicated in moderate and severe hepatic impairment. Not to be used in patients with bladder cancer or a history of bladder cancer. Cytochrome inducers and inhibitors use with caution. Not safe for use in pregnancy. Safety profile good in youth with T2D*
Side effects	Gastrointestinal; abdominal pain/nausea/diarrhoea Weight gain Fluid retention Anaemia Lowering of accrual of bone mineral content. Increased risk of heart failure. Increased risk of bladder cancer.[206]
HbA1c outcomes	TODAY study RCT (n=699) 4 years, Metformin and rosiglitazone resulted in a lower failure rate of 39%, compared with 52% with Metformin alone or 47% metformin plus lifestyle 47% R HbA1c lowering of -0.49%, Short term improvement in IR with M+R (6 months)[68, 278] . *NHMRC level II evidence*
Impact on BMI, metabolic	Rosiglitazone resulted in BMI increase[206]
Trials currently underway	
TGA, PBS Medsafe	Rosiglitazone TGA boxed warning Pioglitazone TGA approved ≥18 yrs PBS rosiglitazone- withdrawn Pioglitazone PBS restricted benefit Pregnancy category B
Recommended use in YOT2D	Not recommended due to risk profile in adults and lack of studies of Pioglitazone in T2D. Emerging evidence re potential role for pioglitazone in MASLD.

*Due to the short duration of YOT2D studies no safety concerns that have been identified in adults have as yet been identified in youth.

Drug class	ALPHA-GLUCOSIDASE INHIBITORS
Formulation	Acarbose 25-100mg three times daily
Route of administration	Oral with food
Mechanism of action	Inhibits breakdown of oligosaccharides which delays absorption of carbohydrates and reduces post-prandial hyperglycaemia. Metabolized in gastrointestinal tract and excreted by kidneys and in faeces.
PK PD studies in youth	None
Precautions	Reduce dose or avoid in renal or liver dysfunction. Not recommended during pregnancy
Side effects	Flatulence, abdominal pain, diarrhoea. Hypoglycaemia when in combination with other agents.
HbA1c outcomes	No data in T2D
TGA, PBS Medsafe	TGA approved T2D ≥18 yrs PBS listed restricted benefit Pregnancy category B
Recommended use in YOT2D	Not recommended

Drug class	SULFONYLUREAS
Formulation	Glimepiride[274] 1-8 mg daily Glyburide[316] 2.5-20mg daily Glipizide 2.5- 40 mg daily
Route of administration	Oral once or twice daily with food
Mechanism of action	Bind to sulfonylurea receptor of the ATP- sensitive potassium channel on beta cell, closing the channel to stimulate insulin release. Metabolized in the liver by cytochrome P450 2C9 isoenzyme. Metabolites are excreted in urine.
PK PD studies in youth	Glimepiride single 1mg dose (n=30), PK like adults (Sanofi product information). Glyburide/metformin n=28 single dose 1.25/250mg (n=28), PK like adults.[317] *NHMRC level II evidence*
Precautions	Not safe in pregnancy Safety profile like adults
Side effects	Hypoglycaemia Weight gain Possible acceleration of beta-cell loss
HbA1c outcomes	Glimepiride versus metformin RCT 26 weeks(n=285). HbA1c -0.54% from baseline p=0.001. Not superior to metformin.[274] *NHMRC level III-1 evidence* RCT (n=167) of glyburide +/- metformin showed combination therapy was not superior to monotherapy with either agent[316] *NHMRC level II evidence*
Impact on BMI, metabolic	BMI and weight gain with glimepiride[274].
Trials currently underway	
TGA, PBS Medsafe	Glimepiride, Glipizide TGA approved T2D ≥18 years Glimepiride, Glipizide PBS listed, restricted benefit Pregnancy category C
Recommended use in YOT2D	Not recommended

Table 5: Metabolic surgery in YOT2D

Metabolic Surgery	Mechanism of action	Side effects/ complications	Clinical trials in T2D and diabetes remission	Impact on BMI, weight, metabolic	Trials currently underway	ANZMOSS criteria for metabolic surgery for adolescents with T2D in Australia and New Zealand
Laparoscopic Sleeve Gastrectomy (LSG)	A minimally-invasive bariatric procedure that is performed laparoscopically. A large volume of the stomach is removed and indirectly restricts food intake. In addition there is an alteration in the absorption of macro and micronutrients, changes in gut hormones with a reduction in ghrelin and increase in GLP-1 and PYY.	Vitamin and mineral deficiencies requiring ongoing supplements	Non-randomised, retrospective study. LSG & LRYGB versus medical treatment (n=202). 2 years diabetes remission 76.1% versus 6.5% in favour of surgery (p<0.001). Surgery group requirement for medication reduced from 88.1% baseline to 23.9% at 2 years. [318] *NHMRC level III-3 evidence* IMPROVE-T2D study, longitudinal observational study (n=12). 3 months HbA1c decreased from 6.6 ± 0.2% to 5.7 ± 0.2% p = .003, with 86% of participants no longer requiring diabetes medications. [319] *NHMRC level III-3 evidence* Prospective cohort study 263/2504 with T2D at entry. Diabetes remission in 74% at 10-year follow-up.[320] *NHMRC level III-3 evidence*	At 2 years BMI reduced more in LSG&LRYGB group compared to controls, and more in LRYGB than LSG p=0.03. Mean TWL 24.8% in surgical and 5.4% medical p<0.001.[318] At 3 months weight and BMI decreased 19% and 18%, respectively, P <.001. Body fat decreased 4.5%, P = .012.[319] 10-year follow-up 59% remission of dyslipidaemia and 64% hypertension. [320]	Phase 4 Surgical or Medical Treatment (ST2OMP) in T2D. comparing vertical sleeve gastrectomy with medical therapy. EDC Dec 2025. Observational study of impact of sleeve gastrectomy on glucose metabolism in Chinese obese and type 2 patients (age 16-65 yrs) EDC Dec 2026 NCT05894811 Phase 1 Phase 2 IMPROVE-T2D, Vertical sleeve gastrectomy impact on pancreatic, renal and cardiovascular health. EDC Oct 2024 NCT03620773	BMI > 35 kg/m2 or 120% of 95th percentile. With a comorbidity including type 2 diabetes, hypertension, MAFLD, intracranial hypertension or obstructive sleep apnoea. May be considered at age 14 in certain circumstances. Required to be Tanner stage 4 or 5, attained final or near final adult height. Persistence of obesity despite lifestyle modification and pharmacotherapy for a minimum of 6 months. Must be able to provide informed consent.

Metabolic Surgery	Mechanism of action	Side effects/ complications	Clinical trials in T2D and diabetes remission	Impact on BMI, weight, metabolic	Trials currently underway	ANZMOSS criteria for metabolic surgery for adolescents with T2D in Australia and New Zealand
Laparoscopic Roux-en-Y Gastric bypass (LRYGB)	Reduces the size of the stomach to a small pouch and attaches the pouch to the small intestine. Stomach capacity is restricted and food bypasses most of the stomach (malabsorptive) and upper part of the small intestine.	Re-operation secondary to gastrojejunal anastomotic stricture, reoperation, leak. Vitamin and mineral deficiencies requiring ongoing supplements	Teen-LABS n=30 (24 RYGB, 6 LSG) versus TODAY n=63. Diabetes remission at 5 years in Teen-LABS versus TODAY was 85.4% versus no remission p<0.001.[321] *NHMRC level III-2 evidence*			
Adjustable gastric band (AGB)	A synthetic adjustable band is placed around the top of the stomach. A port for adjustment of the band is placed in the anterior abdominal wall. The inflated band constricts the stomach creating a small pouch. The procedure restricts food intake and slows gastric emptying.	High re-operation rates, including band slipping. 26% of band removal by 5 years. Heartburn and vomiting common. Dysphagia and diarrhoea less common.[322]	Prospective observational study total n=137 with 5 with T2D at entry. 3 of 5 had diabetes remission at 5 years.[322] *NHMRC level III-3 evidence* Retrospective study of 50 AGB with 5/50 with T2D, all had remission of T2D at 5 years.[323] *NHMRC level III-3 evidence*	18% with >50% excess weight loss at 5 years.[322]		

Endorsing organisations for the consensus guidelines

Australian and New Zealand Society for Paediatric Endocrinology and Diabetes (ANZSPED)

Australian Diabetes Educators Association (ADEA)

Australian Diabetes Society (ADS)

Child & Youth Health National Clinical Network for Children and Young People with Diabetes (CYHNCN)

Diabetes Australia

Diabetes New Zealand

Menzies School of Health Research

Murdoch Children's Research Institute (MCRI)

National Aboriginal Community Controlled Health Organisation (NACCHO)

New Zealand Society for the Study of Diabetes (NZSSD)

Royal Australian College of General Practitioners (RACGP)

Royal New Zealand College of General Practitioners (RNZCGP)

The Kids Research Institute Australia (KRIA)

Table 6: Members of guideline working group

Name of Participant	Conflict of Interest	Organisation	Expertise	Institution	Geographical location	Member's role
Lisa Amato MBBS, FRACP, MMed	None	Australia and New Zealand Society for Paediatric Endocrinology and Diabetes (ANZSPED)	Paediatric Endocrinologist	Royal North Shore Hospital	Sydney, New South Wales	Chair of transition section
Maria Craig MBBS, MMedSc (ClinEpid), PhD, FRACP	None	ANZSPED	Paediatric Endocrinologist	St George Hospital/ The Children's Hospital at Westmead	Sydney, New South Wales	Contributor to all sections of guidelines
Jacqueline Curran MChB, FRACP	None	ANZSPED	Paediatric Endocrinologist	Perth Children's Hospital	Perth, Western Australia	Co-chair of pharmacotherapy section
Elizabeth Davis MBBS, FRACP, PhD	None	ANZSPED The Kids Research Institute Australia	Paediatric Endocrinologist	Perth Children's Hospital, The Kids Research Institute Australia	Perth, Western Australia	Contributor to all sections of guidelines
Gary Deed MBBS, FRACGP	None	Royal Australian College of General Practitioners Australian Diabetes Society	General Practitioner	Royal Australian College of General Practitioners	Brisbane, Queensland	Contributor to all sections of guidelines from Australian primary health care perspective. Chair Diabetes Specific Interest Group RACGP and Member of ADS Guidelines/ Clinical Standards Committee
Amelia Fisher GCertDiabEd, BfoodSc&Nutr(Hons), MDiet	None	ANZSPED	Dietitian	Royal Children's Hospital	Melbourne, Victoria	Contributor to nutrition and daily living section
Kung-Ting Kao MBChB, FRACP, DMedSc	None	ANZSPED	Paediatric Endocrinologist	Royal Children's Hospital	Melbourne, Victoria	Co-chair of pharmacotherapy section
Timothy Kenealy PhD, MBChB, FRNZCGP	None	Royal New Zealand Society of General Practitioners	General Practitioner	University of Auckland	Auckland, New Zealand	Contributor to all sections of guidelines from New Zealand primary health care perspective
Danielle Longmore B Med (Hons), MPHTM, FRACP, PhD	None	ANZSPED Murdoch Children's Research Institute	Paediatric Endocrinologist	Royal Children's Hospital	Melbourne, Victoria	Co-chair of intergenerational and mental health sections Chair of social determinants section

Name of Participant	Conflict of Interest	Organisation	Expertise	Institution	Geographical location	Member's role
Joanna McClintock PGDipClinPsych, PhD	None	Child and Youth Health National Clinical Network for Children & Young People with Diabetes NZ Society for the Study of Diabetes	Clinical Psychologist	Te Whatu Ora – Health New Zealand Waikato District	Waikato, New Zealand	Co-chair of mental health section Contributor to all sections of guidelines from NZ and mental health perspective
Deanne Minniecon MHSc, Grad Dip (Hlth Prom)	None	Diabetes Australia	Group Executive, Aboriginal and Torres Strait Islander Engagement, Diabetes Australia	Diabetes Australia	Brisbane, Queensland	Contributor to all sections of guidelines from Australian consumer and First Nations perspective
Emily Papadimos MBBS, FRACP	None	ANZSPED	Paediatric Endocrinologist	Children's Health Queensland, Menzies School of Health Research	Brisbane, Queensland	Chair of evidence grading section Editing of all sections
Meredith Paterson BExSc(Hons), BBus, MNutrDiet, GCertDiabEd	None	ANZSPED	Dietitian	Royal Children's Hospital	Melbourne, Victoria	Chair of nutrition and daily living section
Alexia Peña MD, FRACP, PhD	None	ANZSPED	Paediatric Endocrinologist	The University of Adelaide Women and Children's Hospital	Adelaide, South Australia	Chair of complications and comorbidities section
Matire Ropiha	None	Diabetes New Zealand	Head of Engagement, Diabetes NZ	Diabetes New Zealand	Wellington, New Zealand	Contributor to all sections of guidelines from Aotearoa NZ consumer and Māori perspective
Anwyn Pyle MBBS AFPHM MPH	None	National Aboriginal Community Controlled Health Organisation	Public Health Physician	NACCHO	Perth, Western Australia	Contributor to all sections of guidelines from Aboriginal community-controlled health sector perspective
Shelley Rose BSc, PGDip Diet, MSc, PhD	None	Child and Youth Health National Clinical Network for Children & Young People with Diabetes ANZSPED	Dietitian	Palmerston North Hospital	Palmerston North, New Zealand	Contributor to all sections of guidelines from NZ perspective

Name of Participant	Conflict of Interest	Organisation	Expertise	Institution	Geographical location	Member's role
Michelle Scerri MBBS (Hons), FACRRM	None	Diabetes across the Lifecourse: Northern Australia Partnership, Menzies School of Health Research Australian College for Rural and Remote Medicine	General Practitioner	Menzies School of Health Research	Katherine, Northern Territory	Contributor to all sections of guidelines from Australian rural/remote primary health care perspective.
Mark Shah NP, CDE	None	ANZSPED Australian Diabetes Educators Association (ADEA)	Diabetes nurse practitioner	Perth Children's Hospital	Perth, Western Australia	Chair of diabetes education section
Phoebe Stewart BMedSci, MBBS (Hons), MBioethics, FRACP	None	ANZSPED	Paediatric Endocrinologist	Royal Hobart Hospital	Hobart, Tasmania	Chair of methods section
Angela Titmuss MBBS (Hons), MPH, FRACP, PhD	None	ANZSPED Diabetes across the Lifecourse: Northern Australia Partnership, Menzies School of Health Research	Paediatric Endocrinologist	Royal Darwin Hospital, Menzies School of Health Research	Darwin, Northern Territory	Chair, guideline working group Chair of screening, diagnosis, phenotype and engagement sections Co-chair of intergenerational section
Esko Wiltshire MBChB, MD, FRACP	None	ANZSPED	Paediatric Endocrinologist	University of Otago, Wellington Health New Zealand Capital, Coast and Hutt Valley	Wellington, New Zealand	Contributor to all sections of guidelines
Jencia Wong BHB, MBChB, FRACP, PhD	None	Australian Diabetes Society	Adult Endocrinologist	Royal Prince Alfred Hospital, The University of Sydney	Sydney, New South Wales	Contributor to all sections of guidelines from adult endocrinology perspective Chair of Australian young adult T2D group
Helen Woodhead MBBS, FRACP, PhD	None	ANZSPED	Paediatric Endocrinologist	Sydney Children's Hospital	Sydney, New South Wales	Contributor to all sections of guidelines

Search strategy

Search strategies used for each section, including databases searched, are listed below. Searches were done until 31 March 2026.

Intergenerational risk

Database: Ovid MEDLINE(R) 1946 to Mar 31, 2026

1. Diabetes Mellitus, Type 2/ and intergenerational.mp
2. Diabetes Mellitus, Type 2/ and offspring.mp

Social determinants of health

Database: Ovid MEDLINE(R) 1946 to Mar 31, 2026

1. type 2 diabetes Diabetes Mellitus, Type 2/ and (Adolescent/ or Child/ or Infant/) and "Social Determinants of Health"/
2. "Social Determinants of Health"/ and Diabetes Mellitus, Type 2/ and (Adolescent/ or Child/ or Infant/)

Screening

Database: Ovid MEDLINE(R) 1946 to Mar 31, 2026

1. exp Diabetes Mellitus, Type 2/di
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. exp Mass Screening
6. screen*.ti,ab,kw.
7. (diagnos* adj3 (test* or tool* or approach* or accuracy)).ti,ab,kw.
8. or/5-7
9. Adolescent
10. exp Child
11. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
12. or/9-11
13. 4 and 8 and 12
14. 13 not (Animals/ not (Animals/ and Humans/))
15. limit 14 to yr="2019 -Current"
16. limit 15 to english language

Database: Embase -Search Strategy:

1. non insulin dependent diabetes mellitus/di [Diagnosis]
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. exp mass screening/
6. screen*.ti,ab,kw.
7. (diagnos* adj3 (test* or tool* or approach* or accuracy)).ti,ab,kw.
8. or/5-7
9. adolescent/
10. exp child/
11. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
12. or/9-11
13. 4 and 8 and 12
14. 13 not ((exp animal/ or nonhuman/) not exp human/)
15. limit 14 to yr="2019 -Current"
16. limit 15 to english language

Cochrane Central Register of Controlled Trials and Cochrane Database of Systematic Reviews

ID Search Hits

1. MeSH descriptor: [Diabetes Mellitus, Type 2] this term only and with qualifier(s): [diagnosis -DI]
2. (diabetes NEAR/3 (type2 or "type 2" or "type ii")):ti,ab
3. ("non insulin dependent diabetes" or NIDDM or T2DM or T2D):ti,ab
4. #1 or #2 or #3
5. MeSH descriptor: [Mass Screening] explode all trees
6. screen*:ti,ab
7. (diagnos* NEAR/3 (test* or tool* or approach* or accuracy)):ti,ab
8. 5 or 6 or 7
9. MeSH descriptor: [Adolescent] this term only
10. MeSH descriptor: [Child] explode all trees
11. (adolescen* or child* or "school-age*" or schoolage* or preschool* or "pre-school*" or youth* or juvenile* or pediatric* or paediatric* or girl* or boy*):ti,ab
12. 9 or 10 or 11
13. 4 and 8 and 12

Clinical presentation and diagnosis

Database: Ovid MEDLINE(R) ALL

1. exp Diabetes Mellitus, Type 2/di
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. exp diagnosis
6. diag*.ti,ab,kw.
7. (diagnos* adj3 (test* or tool* or approach* or accuracy)).ti,ab,kw.
8. or/5-7
9. Adolescent
10. exp Child
11. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
12. or/9-11
13. 4 and 8 and 12
14. 13 not (Animals/ not (Animals/ and Humans/))
15. limit 14 to yr="2019 -Current"
16. limit 15 to english language

Database: Embase -Search Strategy:

1. non insulin dependent diabetes mellitus/di [Diagnosis]
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. exp diagnosis
6. diag*.ti,ab,kw.
7. (diagnos* adj3 (test* or tool* or approach* or accuracy)).ti,ab,kw.
8. or/5-7
9. adolescent/
10. exp child/
11. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
12. or/9-11
13. 4 and 8 and 12

14. 13 not ((exp animal/ or nonhuman/) not exp human/)
15. limit 14 to yr="2019 -Current"
16. limit 15 to english language

Phenotypes

Database: Ovid MEDLINE(R) ALL

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. phenotype
6. clinical presentation
7. 5 or 6
8. (youth* or adolescent* or child*)
9. 4 and 7 and 8
10. limit 9 to yr="2019 -Current"
11. limit 10 to english language

Diabetes education

Database: Ovid MEDLINE(R) ALL

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. Education/
6. Health Education
7. Health Promotion/
8. Learning/
9. Counselling/
10. Patient Education as Topic/
11. Health Knowledge, Attitudes, Practice/
12. ((patient* or client* or participant* or individual* or adolescen* or child* or family or families) adj3 (train* or educat* or teach* or learn* or instruct* or inform or counsel* or empower*)).ti,ab,kw.
13. or/5-12 (518489)
14. Adolescent/
15. exp Child/
16. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or teen* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
17. or/14-16
18. 4 and 13 and 17
19. 18 not (Animals/ not (Animals/ and Humans/))
20. limit 19 to yr="2019 -Current"
21. limit 20 to english language

Nutrition and healthy lifestyle

Database: Ovid MEDLINE(R)

Diet:

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4

6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. exp Diet/
12. exp Diet Fads/
13. exp Diet Therapy/
14. exp Food/
15. exp Feeding Behavior/
16. (diet* or food* or nutrition*).ti,ab,kw.
17. 11 or 12 or 13 or 14 or 15 or 16
18. 10 and 17
19. therapeutics/ or *behavior control/ or *glycemic control/ or *obesity management/ or *remission induction/
20. 18 and 19

Diet: VLED Specific

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. exp Diet/
12. exp Diet Therapy/
13. exp Food/
14. exp Feeding Behavior/
15. (diet* or food* or nutrition*).ti,ab,kw.
16. 11 or 12 or 13 or 14 or 15
17. 10 and 16
18. exp Caloric Restriction/
19. 17 and 18

Diet: Intermittent Fasting Specific

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. exp Diet/
12. exp Diet Therapy/

13. exp Food/
14. exp Feeding Behavior/
15. (diet* or food* or nutrition*).ti,ab,kw.
16. 11 or 12 or 13 or 14 or 15
17. 10 and 16
18. exp Fasting/ or exp Intermittent Fasting/
19. 17 and 18

Diet: Low carbohydrate Specific

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. exp Diet/
12. exp Diet Therapy/
13. exp Food/
14. exp Feeding Behavior/
15. (diet* or food* or nutrition*).ti,ab,kw.
16. 11 or 12 or 13 or 14 or 15
17. 10 and 16
18. exp Dietary Carbohydrates/
19. exp Diet, Carbohydrate-Restricted/ or exp Diet, Ketogenic/
20. 18 and 19
21. 17 and 20

Diet: Glycaemic Index Specific

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. exp Diet/
12. exp Diet Therapy/
13. exp Food/
14. exp Feeding Behavior/
15. (diet* or food* or nutrition*).ti,ab,kw.
16. 11 or 12 or 13 or 14 or 15
17. 10 and 16
18. exp Glycemic Index/
19. 17 and 18

Physical Activity and Sedentary Behaviour

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. 9 and 10
12. exp Exercise/ or exp Exercise Therapy/ or exp Circuit-Based Exercise/ or exp Cool-Down Exercise/ or exp Post-Exercise Recovery Techniques/
13. exp Sedentary Behavior/
14. 12 or 13
15. 11 and 14
16. therapeutics/ or *behavior control/ or *glycemic control/ or *obesity management/ or *remission induction/
17. 15 and 16

Screen Time:

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. 9 and 10
12. exp screen time/
13. exp Social Media/
14. 12 or 13
15. 11 and 14

Sleep

1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. 9 and 10

12. sleep/ or *sleep deprivation/ or *sleep duration/ or exp sleep hygiene/ or *sleep latency/
13. 11 and 12

Illicit Drugs
1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. 9 and 10
12. exp Illicit Drugs/
13. exp Drug-Seeking Behavior/
14. 12 or 13
15. 11 and 14

Smoking
1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. exp obesity/
5. 1 or 2 or 3 or 4
6. exp adolescent/ or *child/
7. (adolescen* or teen* or child* or school-age* or schoolage* or pre?school* or youth* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw.
8. 6 or 7
9. 5 and 8
10. limit 9 to (english language and yr="2020 -Current")
11. 9 and 10
12. smoking/ or exp smoking reduction/ or exp smoking, non-tobacco products/ or exp tobacco smoking/ or exp vaping/
13. exp Smoking Cessation/
14. 12 or 13
15. 11 and 14

Psychological health

Database(s): Ovid MEDLINE(R) 1946 to Mar 31, 2026
1. type 2 diabetes.mp. or Diabetes Mellitus, Type 2/
2. limit 1 to "all child (0 to 18 years)"
3. mental health.mp. or Mental Health/
4. psychological.mp. or Psychological Distress/ or Psychological Well-Being/ or Stress, Psychological/
5. Anxiety/ or Anxiety Disorders/
6. Depression/ or depression.mp.
7. "Feeding and Eating Disorders"/
8. diabetes distress.mp.
9. Psychotic Disorders/
10. 3 or 4 or 5 or 6 or 7 or 8 or 9
11. 2 and 10

Pharmacotherapy

Database(s): Ovid MEDLINE(R) 1946 to Mar 31, 2026

1. ((Type 2 diabetes mellitus or Type 2 diabetes) and (biguanides or metformin or insulin or sulphonylurea or thiazolidinediones or glucagon-like peptide or GLP-1 or DPP4 or dipeptidyl-peptidase IV inhibitors or SGLT2 or sodium-glucose transporter 2 inhibitors or glycoside hydrolase inhibitors)).mp. [mp=title, book title, abstract, original title, name of substance word, subject heading word, floating sub-heading word, keyword heading word, organism supplementary concept word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier, synonyms, population supplementary concept word, anatomy supplementary concept word]
2. limit 1 to (yr="2000 -current" and "all child (0 to 18 years)")
3 . 1 and 2
4. Abstract review of 4,627 from 3, keep 4-5, 13, 22, 29, 35, 50, 55-56, 58, 102, 113, 120, 127, 129, 137, 152, 155, 170, 174, 179, 184, 188, 193, 202, 237, 255, 257, 263, 270, 302, 308, 326, 330, 340, 347, 351, 355, 367, 387, 391, 395, 410, 412, 437, 440, 445-446, 454, 458, 469, 492, 495, 497-498, 517, 526-527, 563, 586, 612, 617, 649, 652, 656, 677, 693, 708, 719, 728, 754, 770, 776, 796, 853, 866, 874, 904, 951, 981, 1003, 1018, 1042, 1080, 1084, 1169, 1204, 1218, 1286, 1294, 1301, 1316, 1325, 1339, 1357, 1388, 1403, 1410, 1412, 1480, 1491, 1521, 1546-1547, 1613, 1624,1628, 1630, 1632, 1684, 1686, 1732, 1787, 1818, 1865

115 (full article review)

In addition, the following sources were used:

ISPAD Clinical Practice Consensus Guidelines 2022: Type 2 diabetes mellitus in youth

ISPAD Clinical Practice Consensus Guidelines 2024: Type 2 diabetes in Children and Adolescents

ClinicalTrials.gov accessed Mar 31, 2026

Comorbidities/complications

Database: Ovid MEDLINE(R) ALL < January 2019 to Mar 31, 2026>

1. exp Diabetes Mellitus, Type 2/ (180877)
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw. (189121)
3. (non insulin dependent diabetes or NIDDM or T2DM or t2D).ti,ab,kw. (66235)
4. 1 or 2 or 3 (250906)
5. exp Comorbidity/ (130243)
6. (comorbid* or co-morbid* or co-occur* or concurren*).ti,ab,kw. (281060)
7. 5 or 6 (398881)
8. Adolescent/ (2253042)
9. exp Child/ (2209796)
10. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or teen* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw. (2388618)
11. 8 or 9 or 10 (4230467)
12. 4 and 7 and 11 (924)
13. 12 not (Animals/ not (Animals/ and Humans/)) (919)
14. limit 13 to dt=20190101-20240601 (225)

Database: Embase <January 2019 to Mar 31 2026 >

1. non insulin dependent diabetes mellitus/ (358146)
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw. (293900)
3. (non insulin dependent diabetes or NIDDM or T2DM or t2D).ti,ab,kw. (107642)
4. 1 or 2 or 3 (420580)
5. exp Comorbidity/ (420515)
6. (comorbid* or co-morbid* or co-occur* or concurren*).ti,ab,kw. (824340)
7. 5 or 6 (961889)
8. adolescent/ (1845708)

9. exp child/ (3207726)
10. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or teen* or juvenile* or p?ediatric* or girl* or boy*).ti,ab,kw. (3045311)
11. 8 or 9 or 10 (4933747)
12. 4 and 7 and 11 (2449)
13. 12 not ((exp animal/ or nonhuman/) not exp human/) (2418)
14. limit 13 to dd=20190101-20240601 (346)

Database: Cochrane <Jan 2019 to Mar 31 2026>
ID Search
1. MeSH descriptor: [Diabetes Mellitus, Type 2] explode all trees
2. (diabetes NEAR/3 (type2 or "type 2" or "type ii")):ti,ab,kw
3. ("non insulin dependent diabetes" or NIDDM or T2DM or T2D):ti,ab,kw
4. 1 or 2 or 3
5. MeSH descriptor: [Comorbidity] explode all trees
6. (comorbid* or co-morbid* or co-occur* or concurren*)
7. 5 or 6
8. MeSH descriptor: [Adolescent] this term only
9. MeSH descriptor: [Child] explode all trees
10. (adolescen* or child* or school-age* or schoolage* or pre?school* or youth* or teen* or juvenile* or p?ediatric* or girl* or boy*):ti,ab,kw
11. 8 or 9 or 10
12. 4 and 7 and 11
13. 12

Transition
Database: Ovid MEDLINE(R) ALL
1. exp Diabetes Mellitus, Type 2/
2. (diabetes adj3 (type2 or type 2 or type ii)).ti,ab,kw.
3. (non insulin dependent diabetes or NIDDM or T2DM or T2D).ti,ab,kw.
4. or/1-3
5. Transition to Adult Care/
6. Transitional Care/
7. (transition* adj2 (care or health?care or practice* or policy or policies or plan*)).mp.
8. or/5-6
9. Adolescent/
10. Young Adult/
11. (adolescen* or young people or young person* or young adult* or young men or young women or young male* or young female* or youth* or teen*).ti,ab,kw.
12. or/9-11
13. 4 and 8 and 12
14. 13 not (Animals/ not (Animals/ and Humans/))
15. limit 14 to yr="2019 -Current"
16. limit 15 to english language

References

1. Lynch JL, Barrientos-Pérez M, Hafez M, et al. Country-Specific Prevalence and Incidence of Youth-Onset Type 2 Diabetes: A Narrative Literature Review. Ann Nutr Metab. 2020;76(5):289-96.
2. Perng W, Conway R, Mayer-Davis E, Dabelea D. Youth-Onset Type 2 Diabetes: The Epidemiology of an Awakening Epidemic. Diabetes Care. 2023;46(3):490-9.
3. McGavock J, Wicklow B, Dart AB. Type 2 diabetes in youth is a disease of poverty. The Lancet. 2017;390(10105):1829.
4. Viner R, White B, Christie D. Type 2 diabetes in adolescents: a severe phenotype posing major clinical challenges and public health burden. Lancet. 2017;389(10085):2252-60.
5. Titmuss A, Davis EA, O'Donnell V, et al. Youth-onset type 2 diabetes among First Nations young people in northern Australia: a retrospective, cross-sectional study. Lancet Diabetes Endocrinol. 2022;10(1):11-3.
6. Haynes A, Curran JA, Davis EA. Two decades of increasing incidence of childhood-onset type 2 diabetes in Western Australia (2000-2019). Med J Aust. 2021;214(6):285-.e1.
7. Smith L, Shi Hui S, Dug Yeo H, Alexander J, Jefferies C. Rising Incidence of Type 2 Diabetes (T2D) Among Children and Adolescents Under 15 Years Over a 28-Year Period (1995–2023): Insights From a Regional Diabetes Service in Auckland, New Zealand. Journal of Paediatrics and Child Health. 2025;61(11):1773-8.
8. Al-Rousan T, AlHeresh R, Saadi A, et al. Epidemiology of cardiovascular disease and its risk factors among refugees and asylum seekers: Systematic review and meta-analysis. International Journal of Cardiology Cardiovascular Risk and Prevention. 2022;12:200126.
9. Al-Saeed AH, Constantino MI, Molyneaux L, et al. An Inverse Relationship Between Age of Type 2 Diabetes Onset and Complication Risk and Mortality: The Impact of Youth-Onset Type 2 Diabetes. Diabetes Care. 2016;39(5):823-9.
10. Nadeau KJ, Anderson BJ, Berg EG, et al. Youth-Onset Type 2 Diabetes Consensus Report: Current Status, Challenges, and Priorities. Diabetes Care. 2016;39(9):1635-42.
11. Bjornstad P, Chao LC, Cree-Green M, et al. Youth-onset type 2 diabetes mellitus: an urgent challenge. Nat Rev Nephrol. 2023;19(3):168-84.
12. TODAY Study Group BP, Drews KL, Caprio S, Gubitosi-Klug R, Nathan DM, Tesfaldet B, Tryggestad J, White NH, Zeitler P. Long-Term Complications in Youth-Onset Type 2 Diabetes. N Engl J Med. 2021;385(5):416-26.
13. Dabelea D, Stafford JM, Mayer-Davis EJ, et al. Association of Type 1 Diabetes vs Type 2 Diabetes Diagnosed During Childhood and Adolescence With Complications During Teenage Years and Young Adulthood. JAMA. 2017;317(8):825-35.
14. Dart AB, Sellers EA, Martens PJ, Rigatto C, Brownell MD, Dean HJ. High Burden of Kidney Disease in Youth-Onset Type 2 Diabetes. Diabetes Care. 2012;35(6):1265-71.
15. McVoy M, Hardin H, Fulchiero E, et al. Mental health comorbidity and youth onset type 2 diabetes: A systematic review of the literature. Int J Psychiatry Med. 2023;58(1):37-55.
16. Spajic D, Curran J, Luu Y, et al. Diabetes Distress and Unmet Support Needs Hinder Optimal Care for Adolescents With Type 2 Diabetes: A Mixed Methods Study. Pediatr Diabetes. 2025;2025:5574666.
17. University of Manitoba. Diabetes Education Resource for Children & Adolescents (DER-CA) Annual Report. In: Child Health Program WRHA, editor. Winnipeg2016.
18. Sellers EAC, McLeod L, Prior HJ, Dragan R, Wicklow BA, Ruth C. Mental health comorbidity is common in children with type 2 diabetes. Pediatr Diabetes. 2022;23(7):991-8.
19. Peña AS, Curran JA, Fuery M, et al. Screening, assessment and management of type 2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines. Medical Journal of Australia. 2020;213(1):30-43.
20. Shah AS, Barrientos-Pérez M, Chang N, et al. ISPAD Clinical Practice Consensus Guidelines 2024: Type 2 Diabetes in Children and Adolescents. Hormone Research in Paediatrics. 2024;97(6):555-83.
21. National Institute for Health and Care Excellence. NICE Guideline: Diabetes (type 1 and type 2) in children and young people: diagnosis and management. London: National Institute for Health and Care Excellence (NICE); 2023.
22. Wong J, Ross GP, Zoungas S, et al. Management of type 2 diabetes in young adults aged 18-30 years: ADS/ADEA/APEG consensus statement. Med J Aust. 2022;216(8):422-9.
23. Dabelea D, Knowler WC, Pettitt DJ. Effect of diabetes in pregnancy on offspring: follow-up research in the Pima Indians. J Matern Fetal Med. 2000;9(1):83-8.
24. Pettitt DJ, Nelson RG, Saad MF, Bennett PH, Knowler WC. Diabetes and Obesity in the Offspring of Pima Indian Women With Diabetes During Pregnancy. Diabetes Care. 1993;16(1):310-4.

25. Dabelea D, Mayer-Davis EJ, Lamichhane AP, et al. Association of intrauterine exposure to maternal diabetes and obesity with type 2 diabetes in youth: the SEARCH Case-Control Study. Diabetes Care. 2008;31(7):1422-6.
26. Tomar AS, Tallapragada DS, Nongmaithem SS, Shrestha S, Yajnik CS, Chandak GR. Intrauterine Programming of Diabetes and Adiposity. Curr Obes Rep. 2015;4(4):418-28.
27. Maple-Brown L, Lee IL, Longmore D, et al. Pregnancy And Neonatal Diabetes Outcomes in Remote Australia: the PANDORA study-an observational birth cohort. Int J Epidemiol. 2019;48(1):307-18.
28. Longmore DK, Barr ELM, Lee IL, et al. Maternal body mass index, excess gestational weight gain, and diabetes are positively associated with neonatal adiposity in the Pregnancy and Neonatal Diabetes Outcomes in Remote Australia (PANDORA) study. Pediatr Obes. 2019:e12490.
29. Wicklow BA, Sellers EA, Sjharma AK, et al. Association of Gestational Diabetes and Type 2 Diabetes Exposure In Utero With the Development of Type 2 Diabetes in First Nations and Non–First Nations Offspring. JAMA Pediatrics. 2018;172(8):724-31.
30. Tam WH, Ma RC, Yang X, et al. Glucose intolerance and cardiometabolic risk in adolescents exposed to maternal gestational diabetes: a 15-year follow-up study. Diabetes Care. 2010;33(6):1382-4.
31. Yajnik CS. Fetal Programming of Diabetes: Still So Much to Learn! Diabetes Care. 2010;33(5):1146-8.
32. Donovan LE, Cundy T. Does exposure to hyperglycaemia in utero increase the risk of obesity and diabetes in the offspring? Diabetic Medicine. 2016;33:695-6.
33. Salama OE, Hizon N, Del Vecchio M, et al. DNA methylation signatures of youth-onset type 2 diabetes and exposure to maternal diabetes. Clin Epigenetics. 2024;16(1):65.
34. Blasetti A, Quarta A, Guarino M, et al. Role of Prenatal Nutrition in the Development of Insulin Resistance in Children. Nutrients. 2022;15(1).
35. Chen P, Piaggi P, Traurig M, et al. Differential methylation of genes in individuals exposed to maternal diabetes in utero. Diabetologia. 2017;60(4):645-55.
36. Montgomery SM, Ekbom A. Smoking during pregnancy and diabetes mellitus in a British longitudinal birth cohort. Bmj. 2002;324(7328):26-7.
37. Yajnik CS, Bandopadhyay S, Bhalerao A, et al. Poor In Utero Growth, and Reduced β-Cell Compensation and High Fasting Glucose From Childhood, Are Harbingers of Glucose Intolerance in Young Indians. Diabetes Care. 2021;44(12):2747-57.
38. Wells JC, Sawaya AL, Wibaek R, et al. The double burden of malnutrition: aetiological pathways and consequences for health. The Lancet. 2020;395(10217):75-88.
39. Yajnik CS. Nutrient-mediated teratogenesis and fuel-mediated teratogenesis: Two pathways of intrauterine programming of diabetes. International Journal of Gynecology & Obstetrics. 2009;104, Supplement:S27-S31.
40. Slomian J, Honvo G, Emonts P, Reginster JY, Bruyère O. Consequences of maternal postpartum depression: A systematic review of maternal and infant outcomes. Womens Health (Lond). 2019;15:1745506519844044.
41. Davis JA, Ohan JL, Bear N, et al. "Stop, pause and take a break": a mixed methods study of the longer-term outcomes of digital emotional wellbeing training for perinatal women. BMC Pregnancy Childbirth. 2024;24(1):811.
42. Séguin L, Potvin L, St-Denis M, Loiselle J. Chronic stressors, social support, and depression during pregnancy. Obstet Gynecol. 1995;85(4):583-9.
43. Marmot M, Allen J, Bell R, et al. WHO European review of social determinants of health and the health divide. Lancet. 2012;380(9846):1011-29.
44. Bacha F, Hannon TS, Tosur M, et al. Pathophysiology and Treatment of Prediabetes and Type 2 Diabetes in Youth. Diabetes Care. 2024;47(12):2038-49.
45. Titmuss A, Korula S, Wicklow B, Nadeau KJ. Youth-onset Type 2 Diabetes: An Overview of Pathophysiology, Prognosis, Prevention and Management. Curr Diab Rep. 2024;24(8):183-95.
46. World Health Organization. World report on social determinants of health equity. Geneva: World Health Organization; 2025.
47. Wu H, Patterson CC, Zhang X, et al. Worldwide estimates of incidence of type 2 diabetes in children and adolescents in 2021. Diabetes Res Clin Pract. 2022;185:109785.
48. Haynes A, Sanderson E, Smith GJ, Curran JC, Maple-Brown L, Davis EA. Demographic and clinical characteristics of a population-based pediatric cohort of type 1 and type 2 diabetes in Western Australia (1999-2019). Pediatr Diabetes. 2021;22(8):1102-7.
49. Mazahery H, Gammon CS, Lawgun D, Conlon CA, Beck KL, von Hurst PR. Pre-diabetes prevalence and associated factors in New Zealand school children: a cross-sectional study. N Z Med J. 2021;134(1531):76-90.
50. Omer Gilon M, Balmakov Y, Gelman S, Twig G. Adolescent Immigration and Type-2 Diabetes. Curr Diab Rep. 2021;21(12):60.

51. Papadimos ER, Claussen C, Magliano DJ, et al. Prevalence of youth type 2 diabetes in global Indigenous populations: a systematic review. Diabetologia. 2026;69(3):568-81.
52. Agarwal S, Wade AN, Mbanya JC, et al. The role of structural racism and geographical inequity in diabetes outcomes. Lancet. 2023;402(10397):235-49.
53. Kelsey MM, Zeitler PS, Nadeau KJ, Shah AS. Type 2 diabetes in youth: Rationale for use of off-label antidiabetic agents. Pediatr Diabetes. 2022;23(6):615-9.
54. Walker RJ, Smalls BL, Campbell JA, Strom Williams JL, Egede LE. Impact of social determinants of health on outcomes for type 2 diabetes: a systematic review. Endocrine. 2014;47(1):29-48.
55. Hill-Briggs F, Adler NE, Berkowitz SA, et al. Social Determinants of Health and Diabetes: A Scientific Review. Diabetes Care. 2020;44(1):258-79.
56. Mangione CM, Barry MJ, Nicholson WK, et al. Screening for Prediabetes and Type 2 Diabetes in Children and Adolescents: US Preventive Services Task Force Recommendation Statement. Jama. 2022;328(10):963-7.
57. Davidson KW, Barry MJ, Mangione CM, et al. Screening for Prediabetes and Type 2 Diabetes: US Preventive Services Task Force Recommendation Statement. Jama. 2021;326(8):736-43.
58. Duan D, Kengne AP, Echouffo-Tcheugui JB. Screening for Diabetes and Prediabetes. Endocrinol Metab Clin North Am. 2021;50(3):369-85.
59. TODAY Study Group. Pregnancy Outcomes in Young Women With Youth-Onset Type 2 Diabetes Followed in the TODAY Study. Diabetes Care. 2021;45(5):1038-45.
60. Jonas DE, Vander Schaaf EB, Riley S, et al. Screening for Prediabetes and Type 2 Diabetes in Children and Adolescents: Evidence Report and Systematic Review for the US Preventive Services Task Force. Jama. 2022;328(10):968-79.
61. Kevat D, Wilson D, Sinha A. A 5-year-old girl with type 2 diabetes. The Lancet. 2014;383(9924):1268.
62. Sawatsky L, Halipchuk J, Wicklow B. Type 2 diabetes in a four-year-old child. Cmaj. 2017;189(26):E888-e90.
63. Wallace AS, Wang D, Shin JI, Selvin E. Screening and Diagnosis of Prediabetes and Diabetes in US Children and Adolescents. Pediatrics. 2020;146(3).
64. White B, Ng SM, Agwu JC, et al. A practical evidence-based approach to management of type 2 diabetes in children and young people (CYP): UK consensus. BMC Med. 2024;22(1):144.
65. Titmuss A, Barzi F, Barr ELM, et al. Association between maternal hyperglycemia in pregnancy and offspring anthropometry in early childhood: the pandora wave 1 study. Int J Obes (Lond). 2023;47(11):1120-31.
66. Prasad RB, Asplund O, Shukla SR, et al. Subgroups of patients with young-onset type 2 diabetes in India reveal insulin deficiency as a major driver. Diabetologia. 2022;65(1):65-78.
67. RISE Consortium. Impact of Insulin and Metformin Versus Metformin Alone on β-Cell Function in Youth With Impaired Glucose Tolerance or Recently Diagnosed Type 2 Diabetes. Diabetes Care. 2018;41(8):1717-25.
68. TODAY Study Group. Effects of metformin, metformin plus rosiglitazone, and metformin plus lifestyle on insulin sensitivity and β-cell function in TODAY. Diabetes Care. 2013;36(6):1749-57.
69. Arslanian SA, El Ghormli L, Kim JY, et al. OGTT Glucose Response Curves, Insulin Sensitivity, and β-Cell Function in RISE: Comparison Between Youth and Adults at Randomization and in Response to Interventions to Preserve β-Cell Function. Diabetes Care. 2021;44(3):817-25.
70. RISE Consortium. Metabolic Contrasts Between Youth and Adults With Impaired Glucose Tolerance or Recently Diagnosed Type 2 Diabetes: I. Observations Using the Hyperglycemic Clamp. Diabetes Care. 2018;41(8):1696-706.
71. Shah AS, Nadeau KJ, Dabelea D, Redondo MJ. Spectrum of Phenotypes and Causes of Type 2 Diabetes in Children. Annu Rev Med. 2022;73:501-15.
72. Kapadia C, Zeitler P, Drugs, Therapeutics Committee of the Pediatric Endocrine S. Hemoglobin A1c measurement for the diagnosis of Type 2 diabetes in children. International Journal of Pediatric Endocrinology. 2012;2012(1):31.
73. Praveen PA, Hockett CW, Ong TC, et al. Diabetic ketoacidosis at diagnosis among youth with type 1 and type 2 diabetes: Results from SEARCH (United States) and YDR (India) registries. Pediatr Diabetes. 2021;22(1):40-6.
74. Magge SN, Wolf RM, Pyle L, et al. The Coronavirus Disease 2019 Pandemic is Associated with a Substantial Rise in Frequency and Severity of Presentation of Youth-Onset Type 2 Diabetes. The Journal of Pediatrics. 2022;251:51-9.e2.
75. Klingensmith GJ, Connor CG, Ruedy KJ, et al. Presentation of youth with type 2 diabetes in the Pediatric Diabetes Consortium. Pediatric Diabetes. 2016;17(4):266-73.
76. Tittel SR, Sondern KM, Weyer M, et al. Multicentre analysis of hyperglycaemic hyperosmolar state and diabetic ketoacidosis in type 1 and type 2 diabetes. Acta Diabetologica. 2020;57(10):1245-53.

77. Tjaden AH, Edelstein SL, Arslanian S, et al. Reproducibility of Glycemic Measures Among Dysglycemic Youth and Adults in the RISE Study. The Journal of Clinical Endocrinology & Metabolism. 2023;108(10):e1125-e33.
78. Chan CL, Pyle L, Newnes L, Nadeau KJ, Zeitler PS, Kelsey MM. Continuous glucose monitoring and its relationship to hemoglobin A1c and oral glucose tolerance testing in obese and prediabetic youth. J Clin Endocrinol Metab. 2015;100(3):902-10.
79. Klingensmith GJ, Pyle L, Arslanian S, et al. The presence of GAD and IA-2 antibodies in youth with a type 2 diabetes phenotype: results from the TODAY study. Diabetes Care. 2010;33(9):1970-5.
80. Reinehr T, Schober E, Wiegand S, Thon A, Holl R. Beta-cell autoantibodies in children with type 2 diabetes mellitus: subgroup or misclassification? Arch Dis Child. 2006;91(6):473-7.
81. Abbasi M, Tosur M, Astudillo M, Refaey A, Sabharwal A, Redondo MJ. Clinical Characterization of Data-Driven Diabetes Clusters of Pediatric Type 2 Diabetes. Pediatr Diabetes. 2023;2023.
82. Jones AG, Hattersley AT. The clinical utility of C-peptide measurement in the care of patients with diabetes. Diabetic Medicine. 2013;30(7):803-17.
83. Dabelea D, Pihoker C, Talton JW, et al. Etiological approach to characterization of diabetes type: the SEARCH for Diabetes in Youth Study. Diabetes Care. 2011;34(7):1628-33.
84. Guo J, Li Z, Carrillo-Larco RM, et al. Data-driven subtypes of newly diagnosed youth-onset type 2 diabetes in the USA. Journal of Diabetes and its Complications. 2026;40(1):109207.
85. Urakami T. Clinical characteristics in Japanese children with nonobese type 2 diabetes. Ann Pediatr Endocrinol Metab. 2018;23(3):113-8.
86. Sellers EA, Singh GR, Sayers SM. Large waist but low body mass index: the metabolic syndrome in Australian Aboriginal children. Journal of Pediatrics. 2008;153(2):222-7.
87. Rodriguez BL, Fujimoto WY, Mayer-Davis EJ, et al. Prevalence of cardiovascular disease risk factors in U.S. children and adolescents with diabetes: the SEARCH for diabetes in youth study. Diabetes Care. 2006;29(8):1891-6.
88. Sellers EAC, Triggs-Raine B, Rockman-Greenberg C, Dean HJ. The prevalence of the HNF-1alpha G319S mutation in Canadian aboriginal youth with type 2 diabetes. Diabetes Care. 2002;25(12):2202-6.
89. Srinivasan S, Chen L, Todd J, et al. The First Genome-Wide Association Study for Type 2 Diabetes in Youth: The Progress in Diabetes Genetics in Youth (ProDiGY) Consortium. Diabetes. 2021;70(4):996-1005.
90. Titmuss A. Type 2 diabetes in Aboriginal and Torres Strait Islander youth: inequity and intergenerational risk. Medicine Today. 2026;27:11-5.
91. Davy C, Cass A, Brady J, et al. Facilitating engagement through strong relationships between primary healthcare and Aboriginal and Torres Strait Islander peoples. Aust N Z J Public Health. 2016;40(6):535-41.
92. Williman J, de Bock M, Wheeler B, et al. The KiwiDiab First National Paediatric Diabetes Benchmarking Audit: A Report on Children and Adolescents With Diabetes in Aotearoa New Zealand 2023. Journal of Paediatrics and Child Health. 2025;61(12):1879-85.
93. Willmington C, Belardi P, Murante AM, Vainieri M. The contribution of benchmarking to quality improvement in healthcare. A systematic literature review. BMC Health Services Research. 2022;22(1):139.
94. TODAY Study Group. A Clinical Trial to Maintain Glycemic Control in Youth with Type 2 Diabetes. New England Journal of Medicine. 2012;366(24):2247-56.
95. Janapala RN, Jayaraj JS, Fathima N, et al. Continuous Glucose Monitoring Versus Self-monitoring of Blood Glucose in Type 2 Diabetes Mellitus: A Systematic Review with Meta-analysis. Cureus. 2019;11(9):e5634.
96. Aleppo G, Beck RW, Bailey R, et al. The Effect of Discontinuing Continuous Glucose Monitoring in Adults With Type 2 Diabetes Treated With Basal Insulin. Diabetes Care. 2021;44(12):2729-37.
97. Martens T, Beck RW, Bailey R, et al. Effect of Continuous Glucose Monitoring on Glycemic Control in Patients With Type 2 Diabetes Treated With Basal Insulin: A Randomized Clinical Trial. Jama. 2021;325(22):2262-72.
98. Chang N, Barber ROB, Llovido Alula J, Durazo-Arvizu R, Chao LC. Continuous Glucose Monitoring versus Standard of Care in Adolescents With Type 2 Diabetes: A Pilot Randomized Cross-Over Trial. J Diabetes Sci Technol. 2023;17(5):1419-20.
99. Manfredo J, Lin T, Gupta R, et al. Short-term use of CGM in youth onset type 2 diabetes is associated with behavioral modifications. Front Endocrinol (Lausanne). 2023;14:1182260.
100. World Health Organisation. Report of the Commission on Ending Childhood Obesity. Geneva: World Health Organisation; 2016.
101. National Aboriginal Community Controlled Health Organisation. Commonwealth Parliamentary Inquiry into Diabetes: Submission to the Standing Committee on Health, Aged Care & Sport Canberra: National Aboriginal Community Controlled Health Organisation; 2023.

102. National Indigenous Australians Agency. National Strategy for Food Security in Remote Aboriginal and Torres Strait Islander Communities: 2025-2035. Canberra: Commonwealth of Australia; 2025.
103. Biggs BK, Wilson DK, Quattlebaum M, Kumar S, Meek A, Jensen TB. Examination of Weight-Loss Motivators and Family Factors in Relation to Weight Management Strategies and Dietary Behaviors among Adolescents with Obesity. Nutrients. 2021;13(5).
104. National Health and Medical Research Council. Australian Dietary Guidelines. . Canberra: National Health and Medical Research Council; 2013.
105. Ministry of Health. Food and Nutrition Guidelines for Healthy Children and Young People (aged 2-18 years): A background paper - Revised February 2015. Wellington: Ministry of Health, New Zealand; 2015.
106. Zang BY, He LX, Xue L. Intermittent Fasting: Potential Bridge of Obesity and Diabetes to Health? Nutrients. 2022;14(5).
107. Neyman A, Hannon TS, Committee On N. Low-Carbohydrate Diets in Children and Adolescents With or at Risk for Diabetes. Pediatrics. 2023;152(4).
108. Morrison S, Galland BC, Haszard JJ, et al. Eating in the absence of hunger in children with mild sleep loss: a randomized crossover trial with learning effects. Am J Clin Nutr. 2021;114(4):1428-37.
109. Ward AL, Reynolds AN, Kuroko S, Fangupo LJ, Galland BC, Taylor RW. Bidirectional associations between sleep and dietary intake in 0-5 year old children: A systematic review with evidence mapping. Sleep Med Rev. 2020;49:101231.
110. Kanaley JA, Colberg SR, Corcoran MH, et al. Exercise/Physical Activity in Individuals with Type 2 Diabetes: A Consensus Statement from the American College of Sports Medicine. Med Sci Sports Exerc. 2022;54(2):353-68.
111. Malik FS, Liese AD, Reboussin BA, et al. Prevalence and Predictors of Household Food Insecurity and Supplemental Nutrition Assistance Program Use in Youth and Young Adults With Diabetes: The SEARCH for Diabetes in Youth Study. Diabetes Care. 2023;46(2):278-85.
112. Annan SF, Higgins LA, Jelleryd E, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Nutritional management in children and adolescents with diabetes. Pediatr Diabetes. 2022;23(8):1297-321.
113. Cyrenne-Dussault M, Sirois M, St-Pierre J, Drouin-Chartier JP. Household Food Insecurity and Changes in BMI Z-Score in a Pediatric Obesity Management Clinic. J Nutr. 2023;153(5):1323-9.
114. Arslanian S, Bacha F, Grey M, Marcus MD, White NH, Zeitler P. Evaluation and Management of Youth-Onset Type 2 Diabetes: A Position Statement by the American Diabetes Association. Diabetes Care. 2018;41(12):2648-68.
115. Burgess H, Koroi H. Intergenerational Intimicies: A whakapapa conceptualisation of kai. Toi Tangata: Tāmaki Makaurau. 2024.
116. Subramani G, Peña AS, Oxlad M. “I Don’t Want to Feel Judged”: A Qualitative Study of Adolescents’ Experiences of Living with Type 2 Diabetes. Canadian Journal of Diabetes. 2025.
117. Weaver E, Freeman N, Mack S, et al. "I Don't Really Know What Diabetes Is": A Qualitative Study Exploring the Experiences of Aboriginal and Torres Strait Islander Young People Aged 10 to 25 Years Living With Type 2 Diabetes in Northern and Central Australia. Can J Diabetes. 2022.
118. National Health and Medical Research Council. Australian Dietary Guidelines. Canberra: National Health and Medical Research Council; 2013.
119. Sauder KA, Stafford JM, The NS, et al. Dietary strategies to manage diabetes and glycemic control in youth and young adults with youth-onset type 1 and type 2 diabetes: The SEARCH for diabetes in youth study. Pediatr Diabetes. 2020;21(7):1093-101.
120. Marcus MD, Wilfley DE, El Ghormli L, et al. Weight change in the management of youth-onset type 2 diabetes: the TODAY clinical trial experience. Pediatr Obes. 2017;12(4):337-45.
121. Ludwig K, Craig ME, Donaghue KC, Maguire A, Benitez-Aguirre PZ, Group AS. Type 2 diabetes in children and adolescents across Australia and New Zealand: A 6-year audit from The Australasian Diabetes Data Network (ADDN). Pediatr Diabetes. 2021;22(3):380-7.
122. Gow ML, Pham-Short A, Jebeile H, Varley BJ, Craig ME. Current Perspectives on the Role of Very-Low-Energy Diets in the Treatment of Obesity and Type 2 Diabetes in Youth. Diabetes Metab Syndr Obes. 2021;14:215-25.
123. Gow ML, Baur LA, Johnson NA, Cowell CT, Garnett SP. Reversal of type 2 diabetes in youth who adhere to a very-low-energy diet: a pilot study. Diabetologia. 2017;60(3):406-15.
124. Lister NB, Jebeile H, Truby H, et al. Fast track to health - Intermittent energy restriction in adolescents with obesity. A randomised controlled trial study protocol. Obes Res Clin Pract. 2020;14(1):80-90.
125. House ET, Gow ML, Lister NB, et al. Pediatric weight management, dietary restraint, dieting, and eating disorder risk: a systematic review. Nutr Rev. 2021;79(10):1114-33.

126. Diabetes Australia. Position Statement: Low Carbohydrate Eating for People with Diabetes. Diabetes Australia August 2018.
127. Dashti HM, Mathew TC, Al-Zaid NS. Efficacy of Low-Carbohydrate Ketogenic Diet in the Treatment of Type 2 Diabetes. Med Princ Pract. 2021;30(3):223-35.
128. Brouwer A, van Raalte DH, Rutters F, et al. Sleep and HbA(1c) in Patients With Type 2 Diabetes: Which Sleep Characteristics Matter Most? Diabetes Care. 2020;43(1):235-43.
129. Mokhlesi B, Temple KA, Tjaden AH, et al. The association of sleep disturbances with glycemia and obesity in youth at risk for or with recently diagnosed type 2 diabetes. Pediatr Diabetes. 2019;20(8):1056-63.
130. Haszard JJ, Jackson R, Morrison S, et al. Losing sleep influences dietary intake in children: a longitudinal compositional analysis of a randomised crossover trial. Int J Behav Nutr Phys Act. 2024;21(1):61.
131. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's updated sleep duration recommendations: final report. Sleep Health. 2015;1(4):233-43.
132. Perez-Chada D, Bioch SA, Schonfeld D, Gozal D, Perez-Lloret S, Sleep in Adolescents Collaborative study g. Screen use, sleep duration, daytime somnolence, and academic failure in school-aged adolescents. PLoS One. 2023;18(2):e0281379.
133. Galland BC, de Wilde T, Taylor RW, Smith C. Sleep and pre-bedtime activities in New Zealand adolescents: differences by ethnicity. Sleep Health. 2020;6(1):23-31.
134. Galland BC, Gray AR, Penno J, Smith C, Lobb C, Taylor RW. Gender differences in sleep hygiene practices and sleep quality in New Zealand adolescents aged 15 to 17 years. Sleep Health. 2017;3(2):77-83.
135. Pastor A, O'Brien CL, Teng J, et al. Experiences of young adults with type 1 diabetes while using alcohol and recreational drugs: An interpretative phenomenological analysis (IPA) of semi-structured interviews. Diabetes Res Clin Pract. 2018;141:47-55.
136. Kinney GL, Akturk HK, Taylor DD, Foster NC, Shah VN. Cannabis Use Is Associated With Increased Risk for Diabetic Ketoacidosis in Adults With Type 1 Diabetes: Findings From the T1D Exchange Clinic Registry. Diabetes Care. 2020;43(1):247-9.
137. American Diabetes Association Professional Practice C. 14. Children and Adolescents: Standards of Care in Diabetes-2024. Diabetes Care. 2024;47(Suppl 1):S258-S81.
138. Herbst A, Kapellen T, Schober E, et al. Impact of regular physical activity on blood glucose control and cardiovascular risk factors in adolescents with type 2 diabetes mellitus – a multicenter study of 578 patients from 225 centres. Pediatric Diabetes. 2015;16(3):204-10.
139. Department of Health. Physical activity and exercise guidelines for children and young people (5-17 years). Canberra: Department of Health; 2021.
140. Haghjoo P, Siri G, Soleimani E, Farhangi MA, Alesaeidi S. Screen time increases overweight and obesity risk among adolescents: a systematic review and dose-response meta-analysis. BMC Primary Care. 2022;23(1):161.
141. Brosnan B, Haszard JJ, Meredith-Jones KA, Wickham SR, Galland BC, Taylor RW. Screen Use at Bedtime and Sleep Duration and Quality Among Youths. JAMA Pediatr. 2024;178(11):1147-54.
142. Australian Institute of Health and Welfare. Australia’s youth: Mental illness. Canberra; 2021.
143. Fleming T, Crengle S, Peiris-John R, et al. Priority actions for improving population youth mental health: An equity framework for Aotearoa New Zealand. Mental Health & Prevention. 2024;34:200340.
144. Dabelea D, Sauder KA, Jensen ET, et al. Twenty years of pediatric diabetes surveillance: what do we know and why it matters. Ann N Y Acad Sci. 2021;1495(1):99-120.
145. Copeland KC, Zeitler P, Geffner M, et al. Characteristics of adolescents and youth with recent-onset type 2 diabetes: the TODAY cohort at baseline. J Clin Endocrinol Metab. 2011;96(1):159-67.
146. Anderson BJ, Edelstein S, Abramson NW, et al. Depressive symptoms and quality of life in adolescents with type 2 diabetes: baseline data from the TODAY study. Diabetes Care. 2011;34(10):2205-7.
147. Silverstein J, Cheng P, Ruedy KJ, et al. Depressive Symptoms in Youth With Type 1 or Type 2 Diabetes: Results of the Pediatric Diabetes Consortium Screening Assessment of Depression in Diabetes Study. Diabetes Care. 2015;38(12):2341-3.
148. Hood KK, Beavers DP, Yi-Frazier J, et al. Psychosocial burden and glycemic control during the first 6 years of diabetes: results from the SEARCH for Diabetes in Youth study. J Adolesc Health. 2014;55(4):498-504.
149. Trief PM, Wen H, Burke B, et al. Psychosocial Factors and Glycemic Control in Young Adults With Youth-Onset Type 2 Diabetes. JAMA Netw Open. 2024;7(4):e245620.
150. Dart AB, Wicklow B, Blydt-Hansen TD, et al. A Holistic Approach to Risk for Early Kidney Injury in Indigenous Youth With Type 2 Diabetes: A Proof of Concept Paper From the iCARE Cohort. Can J Kidney Health Dis. 2019;6:2054358119838836.

151. Australian Government. National Aboriginal and Torres Strait Islander Health Plan 2013-2023. Canberra; 2013.
152. Durie M. Mental health and Maori development. Aust N Z J Psychiatry. 1999;33(1):5-12.
153. Wilson D, Moloney E, Parr JM, Aspinall C, Slark J. Creating an Indigenous Māori-centred model of relational health: A literature review of Māori models of health. J Clin Nurs. 2021;30(23-24):3539-55.
154. Pitama S RP, Cram F, Gillies M, Huria T, Dallas-Katoa W. Meihana Model: A Clinical Assessment Framework. New Zealand Journal of Psychology. 2007;36(3):8.
155. Capstick S, Norris P, Sopoaga F, Tobata W. Relationships between health and culture in Polynesia - a review. Soc Sci Med. 2009;68(7):1341-8.
156. McCubbin L MH, Zhang W, Kehl L, Strom I. Relational Well-being: An Indigenous Perspective and Measure. Family Relations. 2013;62(April 2013):12.
157. Spry E, Seear K, Harkin B, et al. Aboriginal young people's experiences of type 2 diabetes diagnosis, management and support: A qualitative study in the Kimberley region of Western Australia. Health Promot J Austr. 2024.
158. Zheng K, Abraham C, Bruzzese JM, Smaldone A. Longitudinal Relationships Between Depression and Chronic Illness in Adolescents: An Integrative Review. J Pediatr Health Care. 2020;34(4):333-45.
159. Levitt Katz LE, Swami S, Abraham M, et al. Neuropsychiatric disorders at the presentation of type 2 diabetes mellitus in children. Pediatr Diabetes. 2005;6(2):84-9.
160. Roberts AJ, Bao H, Qu P, et al. Mental health comorbidities in adolescents and young adults with type 2 diabetes. J Pediatr Nurs. 2021;61:280-3.
161. Wicklow BA, Dart AB, Mckee J, et al. Experiences of First Nations adolescents living with type 2 diabetes: a focus group study. CMAJ : Canadian Medical Association Journal. 2021;193:E403 - E9.
162. Stapleton C, Watkins E, Hare MJL, Timms F, Wood AJ, Titmuss A. The prevalence of diabetes distress and its association with glycaemia in young people living with insulin-requiring-diabetes in a regional centre in Australia. J Paediatr Child Health. 2022;58(12):2273-9.
163. Speight J, Holmes-Truscott E, Garza M, et al. Bringing an end to diabetes stigma and discrimination: an international consensus statement on evidence and recommendations. Lancet Diabetes Endocrinol. 2024;12(1):61-82.
164. Carman R, Towell-Barnard A, Shah M, et al. "Sometimes Our Mob Don't Really Take It Serious Until It's Serious": The Experiences of Western Australian Aboriginal Adolescents Living With Type 2 Diabetes, Their Parents, and Their Family Members. Can J Diabetes. 2023;47(5):420-7.
165. Venditti EM, Tan K, Chang N, et al. Barriers and strategies for oral medication adherence among children and adolescents with Type 2 diabetes. Diabetes Res Clin Pract. 2018;139:24-31.
166. Nip ASY, Reboussin BA, Dabelea D, et al. Disordered Eating Behaviors in Youth and Young Adults With Type 1 or Type 2 Diabetes Receiving Insulin Therapy: The SEARCH for Diabetes in Youth Study. Diabetes Care. 2019;42(5):859-66.
167. Pinhas-Hamiel O, Levy-Shraga Y. Eating disorders in adolescents with type 2 and type 1 diabetes. Curr Diab Rep. 2013;13(2):289-97.
168. Duncanson E, Le Leu RK, Shanahan L, et al. The prevalence and evidence-based management of needle fear in adults with chronic disease: A scoping review. PLOS ONE. 2021;16(6):e0253048.
169. Walders-Abramson N, Venditti EM, Ievers-Landis CE, et al. Relationships among stressful life events and physiological markers, treatment adherence, and psychosocial functioning among youth with type 2 diabetes. J Pediatr. 2014;165(3):504-8 e1.
170. Simon SL, Higgins J, Melanson E, Wright KP, Jr., Nadeau KJ. A Model of Adolescent Sleep Health and Risk for Type 2 Diabetes. Curr Diab Rep. 2021;21(2):4.
171. Guerrero Fernandez de Alba I, Gimeno-Miguel A, Poblador-Plou B, et al. Association between mental health comorbidity and health outcomes in type 2 diabetes mellitus patients. Sci Rep. 2020;10(1):19583.
172. Mateo K, Greenberg B, Valenzuela J. Disordered Eating Behaviors and Eating Disorders in Youth With Type 2 Diabetes: A Systematic Review. Diabetes Spectrum. 2024.
173. Hayes JF, Fitzsimmons-Craft EE, Karam AM, Jakubiak J, Brown ML, Wilfley DE. Disordered Eating Attitudes and Behaviors in Youth with Overweight and Obesity: Implications for Treatment. Curr Obes Rep. 2018;7(3):235-46.
174. de Wit M, Gajewska KA, Goethals ER, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Psychological care of children, adolescents and young adults with diabetes. Pediatr Diabetes. 2022;23(8):1373-89.

175. St George SM, Pulgaron ER, Ferranti D, et al. A Qualitative Study of Cognitive, Behavioral, and Psychosocial Challenges Associated With Pediatric Type 2 Diabetes in Ethnic Minority Parents and Adolescents. Diabetes Educ. 2017;43(2):180-9.
176. Smith GL, McGuinness TM. Adolescent Psychosocial Assessment: The HEEADSSS. J Psychosoc Nurs Ment Health Serv. 2017;55(5):24-7.
177. Weissberg-Benchell J, Antisdel-Lomaglio J. Diabetes-specific emotional distress among adolescents: feasibility, reliability, and validity of the problem areas in diabetes-teen version. Pediatr Diabetes. 2011;12(4 Pt 1):341-4.
178. Young-Hyman D, de Groot M, Hill-Briggs F, Gonzalez JS, Hood K, Peyrot M. Psychosocial Care for People With Diabetes: A Position Statement of the American Diabetes Association. Diabetes Care. 2016;39(12):2126-40.
179. Polonsky WH, Fisher L, Earles J, et al. Assessing psychosocial distress in diabetes: development of the diabetes distress scale. Diabetes Care. 2005;28(3):626-31.
180. Topp CW, Ostergaard SD, Sondergaard S, Bech P. The WHO-5 Well-Being Index: a systematic review of the literature. Psychother Psychosom. 2015;84(3):167-76.
181. Kessler RC AG, Colpe LJ, Hiripi E, Mroczek DK, Normand SLT, Walters EE, Zaslavsky AM. Kessler Psychological Distress Scale (K6, K10). 2002.
182. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606-13.
183. Kroenke K, Spitzer RL, Williams JB. The Patient Health Questionnaire-2: validity of a two-item depression screener. Med Care. 2003;41(11):1284-92.
184. Johnson JG, Harris ES, Spitzer RL, Williams JB. The patient health questionnaire for adolescents: validation of an instrument for the assessment of mental disorders among adolescent primary care patients. J Adolesc Health. 2002;30(3):196-204.
185. Getting it Right Collaborative G. Getting it Right: validating a culturally specific screening tool for depression (aPHQ-9) in Aboriginal and Torres Strait Islander Australians. Med J Aust. 2019;211(1):24-30.
186. Skinner T, Brown A, Teixeira-Pinto A, et al. Sensitivity and specificity of Aboriginal-developed items to supplement the adapted PHQ-9 screening measure for depression: results from the Getting it Right study. Med J Aust. 2024;221(5):258-63.
187. Harriss LR, Kyle M, Connolly K, et al. Screening for depression in young Indigenous people: building on a unique community initiative. Aust J Prim Health. 2018.
188. Hill LS, Reid F, Morgan JF, Lacey JH. SCOFF, the development of an eating disorder screening questionnaire. International Journal of Eating Disorders. 2010;43(4):344-51.
189. Zuijdwijk CS, Pardy SA, Dowden JJ, Dominic AM, Bridger T, Newhook LA. The mSCOFF for Screening Disordered Eating in Pediatric Type 1 Diabetes. Diabetes Care. 2014;37(2):e26-e7.
190. Nagel T, Hinton R, Griffin C. Yarning about Indigenous mental health: Translation of a recovery paradigm to practice. Advances in Mental Health. 2012;10(3):216-23.
191. Goodman R. The Strengths and Difficulties Questionnaire: A Research Note. Journal of Child Psychology and Psychiatry. 1997;38(5):581-6.
192. Axelsson P, Kukutai T, Kippen R. The field of Indigenous health and the role of colonisation and history. Journal of Population Research. 2016;33(1):1-7.
193. Curtis E, Jones R, Tipene-Leach D, et al. Why cultural safety rather than cultural competency is required to achieve health equity: a literature review and recommended definition. Int J Equity Health. 2019;18(1):174.
194. Fields T, Foster W, Biles BJ, Yashadhana A. Redefining the gap in Aboriginal health: from deficit to cultural connection. Lancet Reg Health West Pac. 2024;52:101176.
195. McGavock J, Durksen A, Wicklow B, et al. Determinants of Readiness for Adopting Healthy Lifestyle Behaviors Among Indigenous Adolescents with Type 2 Diabetes in Manitoba, Canada: A Cross-Sectional Study. Obesity (Silver Spring). 2018;26(5):910-5.
196. Bennetter KE, Clench-Aas J, Raanaas RK. Sense of mastery as mediator buffering psychological distress among people with diabetes. J Diabetes Complications. 2016;30(5):839-44.
197. Pivarunas B, Kelly NR, Pickworth CK, et al. Mindfulness and eating behavior in adolescent girls at risk for type 2 diabetes. Int J Eat Disord. 2015;48(6):563-9.
198. Gulley LD, Shomaker LB, Kelly NR, et al. Examining cognitive-behavioral therapy change mechanisms for decreasing depression, weight, and insulin resistance in adolescent girls at risk for type 2 diabetes. J Psychosom Res. 2022;157:110781.

199. Shomaker LB, Pivarunas B, Annameier SK, et al. One-Year Follow-Up of a Randomized Controlled Trial Piloting a Mindfulness-Based Group Intervention for Adolescent Insulin Resistance. Front Psychol. 2019;10:1040.
200. Allen D, Scarinci N, Hickson L. The Nature of Patient- and Family-Centred Care for Young Adults Living with Chronic Disease and their Family Members: A Systematic Review. Int J Integr Care. 2018;18(2):14.
201. Mokhtari Z, Mokhtari S, Afrasiabifar A, Hosseini N. The Effect of Family-Centered Intervention on Key Indicators of Diabetes Management and Control in Patients with Type-2 Diabetes. Int J Prev Med. 2023;14:54.
202. Laffel L, Chang N, Grey M, et al. Metformin monotherapy in youth with recent onset type 2 diabetes: experience from the prerandomization run-in phase of the TODAY study. Pediatr Diabetes. 2012;13(5):369-75.
203. Lascar N, Brown J, Pattison H, Barnett AH, Bailey CJ, Bellary S. Type 2 diabetes in adolescents and young adults. Lancet Diabetes Endocrinol. 2018;6(1):69-80.
204. Hosey C, Halpin K, Yan Y. Considering metformin as a second-line treatment for children and adolescents with prediabetes. Journal of Pediatric Endocrinology and Metabolism. 2022;35.
205. Canadian Diabetes Association Clinical Practice Guidelines Expert C, Panagiotopoulos C, Riddell MC, Sellers EA. Type 2 diabetes in children and adolescents. Can J Diabetes. 2013;37 Suppl 1:S163-7.
206. Group TS. Safety and tolerability of the treatment of youth-onset type 2 diabetes: the TODAY experience. Diabetes Care. 2013;36(6):1765-71.
207. Zeitler P, Arslanian S, Fu J, et al. ISPAD Clinical Practice Consensus Guidelines 2018: Type 2 diabetes mellitus in youth. Pediatr Diabetes. 2018;19 Suppl 27:28-46.
208. Wheeler MD, Barrientos-Perez M, Lo FS, et al. A 26-week, randomized trial of insulin detemir versus NPH insulin in children and adolescents with type 2 diabetes (iDEAt2). Eur J Pediatr. 2018;177(10):1497-503.
209. Reznik Y, Cohen O, Aronson R, et al. Insulin pump treatment compared with multiple daily injections for treatment of type 2 diabetes (OpT2mise): a randomised open-label controlled trial. Lancet. 2014;384(9950):1265-72.
210. Badaru A, Klingensmith GJ, Dabelea D, et al. Correlates of treatment patterns among youth with type 2 diabetes. Diabetes Care. 2014;37(1):64-72.
211. Group TS, Zeitler P, Hirst K, et al. A clinical trial to maintain glycemic control in youth with type 2 diabetes. N Engl J Med. 2012;366(24):2247-56.
212. Consortium R. Impact of Insulin and Metformin Versus Metformin Alone on beta-Cell Function in Youth With Impaired Glucose Tolerance or Recently Diagnosed Type 2 Diabetes. Diabetes Care. 2018;41(8):1717-25.
213. Tamborlane WV, Barrientos-Perez M, Fainberg U, et al. Liraglutide in Children and Adolescents with Type 2 Diabetes. N Engl J Med. 2019;381(7):637-46.
214. Monami M, Nreu B, Scatena A, et al. Safety issues with glucagon-like peptide-1 receptor agonists (pancreatitis, pancreatic cancer and cholelithiasis): Data from randomized controlled trials. Diabetes Obes Metab. 2017;19(9):1233-41.
215. Marshall S, Ryan E, Rivera J, Reynolds L, Atti S. GLP-1 Receptor Agonist Exposures Are Increasingly Common and Generally Associated with Mild Symptoms: A Single Poison Center Experience. J Med Toxicol. 2024;20(3):278-85.
216. Stauder U, Enginee D, Elton H, Penfornis A, Edelman S. Comparative Assessment of Lixisenatide, Exenatide, and Liraglutide Pen Devices: A Pilot User-Based Study. J Diabetes Sci Technol. 2014;8(1):123-31.
217. Kahn HS, Divers J, Fino NF, et al. Alternative waist-to-height ratios associated with risk biomarkers in youth with diabetes: comparative models in the SEARCH for Diabetes in Youth Study. International journal of obesity (2005). 2019;43(10):1940-50.
218. Tung JY-L, Kwan EY-W, But BW-M, et al. Incidence and clinical characteristics of pediatric-onset type 2 diabetes in Hong Kong: The Hong Kong childhood diabetes registry 2008 to 2017. Pediatric diabetes. 2022;23(5):556-61.
219. Copeland KC, Zeitler P, Geffner M, et al. Characteristics of adolescents and youth with recent-onset type 2 diabetes: the TODAY cohort at baseline. J Clin Endocrinol Metab. 2011;96(1):159-67.
220. Abdelhameed F, Giuffrida A, Thorp B, Moorthy MK, Gevers EF. Exploring the Surge in Paediatric Type 2 Diabetes in an Inner-City London Centre-A Decade-Long Analysis of Incidence, Outcomes, and Transition. Children (Basel, Switzerland). 2024;11(2).
221. Jensen ET, Rigdon J, Rezaei KA, et al. Prevalence, Progression, and Modifiable Risk Factors for Diabetic Retinopathy in Youth and Young Adults With Youth-Onset Type 1 and Type 2 Diabetes: The SEARCH for Diabetes in Youth Study. Diabetes Care. 2023;46(6):1252-60.
222. Development and Progression of Diabetic Retinopathy in Adolescents and Young Adults With Type 2 Diabetes: Results From the TODAY Study. Diabetes Care. 2021;45(5):1049-55.
223. Long-term Outcomes Among Young Adults With Type 2 Diabetes Based on Durability of Glycemic Control: Results From the TODAY Cohort Study. Diabetes Care. 2022;45(11):2689-97.

224. Cioana M, Deng J, Hou M, et al. Prevalence of Hypertension and Albuminuria in Pediatric Type 2 Diabetes: A Systematic Review and Meta-analysis. JAMA network open. 2021;4(4):e216069.
225. Fan Y, Lau ESH, Wu H, et al. Incidence of long-term diabetes complications and mortality in youth-onset type 2 diabetes: A systematic review. Diabetes Res Clin Pract. 2022;191:110030.
226. Sun J, Wang Y, Zhang X, Zhu S, He H. Prevalence of peripheral neuropathy in patients with diabetes: A systematic review and meta-analysis. Prim Care Diabetes. 2020;14(5):435-44.
227. Varley BJ, Gow ML, Cho YH, et al. Higher frequency of cardiovascular autonomic neuropathy in youth with type 2 compared to type 1 diabetes: Role of cardiometabolic risk factors. Pediatr Diabetes. 2022;23(7):1073-9.
228. Risk Factors for Diabetic Peripheral Neuropathy in Adolescents and Young Adults With Type 2 Diabetes: Results From the TODAY Study. Diabetes Care. 2021;45(5):1065-72.
229. Jaiswal M, Divers J, Dabelea D, et al. Prevalence of and Risk Factors for Diabetic Peripheral Neuropathy in Youth With Type 1 and Type 2 Diabetes: SEARCH for Diabetes in Youth Study. Diabetes Care. 2017;40(9):1226-32.
230. Jaiswal M, Divers J, Urbina EM, et al. Cardiovascular autonomic neuropathy in adolescents and young adults with type 1 and type 2 diabetes: The SEARCH for Diabetes in Youth Cohort Study. Pediatr Diabetes. 2018;19(4):680-9.
231. Shah AS, El Ghormli L, Vajravelu ME, et al. Heart Rate Variability and Cardiac Autonomic Dysfunction: Prevalence, Risk Factors, and Relationship to Arterial Stiffness in the Treatment Options for Type 2 Diabetes in Adolescents and Youth (TODAY) Study. Diabetes Care. 2019;42(11):2143-50.
232. Urbina EM, Isom S, Dabelea D, et al. Association of Elevated Arterial Stiffness With Cardiac Target Organ Damage and Cardiac Autonomic Neuropathy in Young Adults With Diabetes: The SEARCH for Diabetes in Youth Study. Diabetes Care. 2023;46(4):786-93.
233. Chowdhury M, Nevitt S, Eleftheriadou A, et al. Cardiac autonomic neuropathy and risk of cardiovascular disease and mortality in type 1 and type 2 diabetes: a meta-analysis. BMJ Open Diabetes Res Care. 2021;9(2).
234. Cioana M, Deng J, Nadarajah A, et al. The Prevalence of Obesity Among Children With Type 2 Diabetes: A Systematic Review and Meta-analysis. JAMA Netw Open. 2022;5(12):e2247186.
235. Weghuber D, Barrett T, Barrientos-Pérez M, et al. Once-Weekly Semaglutide in Adolescents with Obesity. N Engl J Med. 2022;387(24):2245-57.
236. Olbers T, Beamish AJ, Gronowitz E, et al. Laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity (AMOS): a prospective, 5-year, Swedish nationwide study. Lancet Diabetes Endocrinol. 2017;5(3):174-83.
237. Inge TH, Laffel LM, Jenkins TM, et al. Comparison of Surgical and Medical Therapy for Type 2 Diabetes in Severely Obese Adolescents. JAMA Pediatr. 2018;172(5):452-60.
238. Beamish AJ, Ryan Harper E, Järvholm K, Janson A, Olbers T. Long-term Outcomes Following Adolescent Metabolic and Bariatric Surgery. J Clin Endocrinol Metab. 2023;108(9):2184-92.
239. Peña AS, Curran JA, Fuery M, et al. Screening, assessment and management of type 2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines. Med J Aust. 2020;213(1):30-43.
240. Teede HJ, Khomami MB, Morman R, et al. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. The Lancet. 2026;407(10545):2329-39.
241. Cioana M, Deng J, Nadarajah A, et al. Prevalence of Polycystic Ovary Syndrome in Patients With Pediatric Type 2 Diabetes: A Systematic Review and Meta-analysis. JAMA Netw Open. 2022;5(2):e2147454.
242. Naz MSG, Tehrani FR, Majd HA, et al. The prevalence of polycystic ovary syndrome in adolescents: A systematic review and meta-analysis. Int J Reprod Biomed. 2019;17(8):533-42.
243. Long C, Feng H, Duan W, et al. Prevalence of polycystic ovary syndrome in patients with type 2 diabetes: A systematic review and meta-analysis. Front Endocrinol (Lausanne). 2022;13:980405.
244. Peña AS, Witchel SF, Boivin J, et al. International evidence-based recommendations for polycystic ovary syndrome in adolescents. BMC Med. 2025;23(1):151.
245. Peña AS, Witchel SF, Hoeger KM, et al. Adolescent polycystic ovary syndrome according to the international evidence-based guideline. BMC Med. 2020;18(1):72.
246. Teede HJ, Tay CT, Laven JJE, et al. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Eur J Endocrinol. 2023;189(2):G43-g64.
247. Merino PM, Codner E. Contraception for Adolescents and Young Women with Type 2 Diabetes-Specific Considerations. Curr Diab Rep. 2022;22(2):77-84.
248. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. Ann Hepatol. 2024;29(1):101133.

249. Liu J, Mu C, Li K, Luo H, Liu Y, Li Z. Estimating Global Prevalence of Metabolic Dysfunction-Associated Fatty Liver Disease in Overweight or Obese Children and Adolescents: Systematic Review and Meta-Analysis. Int J Public Health. 2021;66:1604371.
250. Nobili V, Mantovani A, Cianfarani S, et al. Prevalence of prediabetes and diabetes in children and adolescents with biopsy-proven non-alcoholic fatty liver disease. J Hepatol. 2019;71(4):802-10.
251. Koutny F, Weghuber D, Bollow E, et al. Prevalence of prediabetes and type 2 diabetes in children with obesity and increased transaminases in European German-speaking countries. Analysis of the APV initiative. Pediatr Obes. 2020;15(4):e12601.
252. Bardugo A, Bendor CD, Zucker I, et al. Adolescent Nonalcoholic Fatty Liver Disease and Type 2 Diabetes in Young Adulthood. J Clin Endocrinol Metab. 2021;106(1):e34-e44.
253. Isaacs SD, Farrelly FV, Brennan PN. Role of anti-diabetic medications in the management of MASLD. Frontline Gastroenterology. 2025;16(3):239.
254. Gabbs MHJ, Dart AB, Woo MR, Pinto T, Wicklow BA. Poor Sleep, Increased Stress and Metabolic Comorbidity in Adolescents and Youth With Type 2 Diabetes. Canadian journal of diabetes. 2022;46(2):142-9.
255. Wu J-Y, Lin Y-M, Hsu W-H, et al. Effects of semaglutide on obstructive sleep apnea risk and outcomes in patients with obesity. Annals of the American Thoracic Society. 2026;23(4):584-96.
256. Levitt Katz LE, Bacha F, Gidding SS, et al. Lipid Profiles, Inflammatory Markers, and Insulin Therapy in Youth with Type 2 Diabetes. J Pediatr. 2018;196:208-16 e2.
257. Brady RP, Shah AS, Jensen ET, et al. Glycemic control is associated with dyslipidemia over time in youth with type 2 diabetes: The SEARCH for diabetes in youth study. Pediatr Diabetes. 2021;22(7):951-9.
258. Peters A, Laffel L, American Diabetes Association Transitions Working G. Diabetes care for emerging adults: recommendations for transition from pediatric to adult diabetes care systems: a position statement of the American Diabetes Association, with representation by the American College of Osteopathic Family Physicians, the American Academy of Pediatrics, the American Association of Clinical Endocrinologists, the American Osteopathic Association, the Centers for Disease Control and Prevention, Children with Diabetes, The Endocrine Society, the International Society for Pediatric and Adolescent Diabetes, Juvenile Diabetes Research Foundation International, the National Diabetes Education Program, and the Pediatric Endocrine Society (formerly Lawson Wilkins Pediatric Endocrine Society). Diabetes Care. 2011;34(11):2477-85.
259. Agarwal S, Raymond JK, Isom S, et al. Transfer from paediatric to adult care for young adults with Type 2 diabetes: the SEARCH for Diabetes in Youth Study. Diabet Med. 2018;35(4):504-12.
260. Pundyk KJ, Sellers EAC, Kroeker K, Wicklow BA. Transition of Youth With Type 2 Diabetes: Predictors of Health-Care Utilization After Transition to Adult Care From Population-Based Administrative Data. Can J Diabetes. 2021;45(5):451-7.
261. Zurynski Y, Carrigan A, Meulenbroeks I, et al. Transition models of care for type 1 diabetes: a systematic review. BMC Health Serv Res. 2023;23(1):779.
262. Gerhardsson P, Schwandt A, Witsch M, et al. The SWEET Project 10-Year Benchmarking in 19 Countries Worldwide Is Associated with Improved HbA1c and Increased Use of Diabetes Technology in Youth with Type 1 Diabetes. Diabetes Technology & Therapeutics. 2021;23(7):491-9.
263. Burry E, Ivers N, Mahmud FH, Shulman R. Interventions using pediatric diabetes registry data for quality improvement: A systematic review. Pediatric Diabetes. 2018;19(7):1249-56.
264. Ayub A, Ng C, Portales-Casamar E, Metzger D, Amed S. Toward Building a Provincial Diabetes Registry of Children and Youth Living With Diabetes in British Columbia, Canada. Canadian Journal of Diabetes. 2022;46(4):346-52.e1.
265. Witsch M, Kosteria I, Kordonouri O, et al. Possibilities and challenges of a large international benchmarking in pediatric diabetology—The SWEET experience. Pediatr Diabetes. 2016;17(S23):7-15.
266. Speight J, Skinner, TC, Dunning, T, Black, T, Kilov, G, Lee, C, Scinbilia, R, Johnson, G. Our language matters: Improving communication with and about people with diabetes. A position statement by Diabetes Australia. Diabetes Res Clin Pract. 2021;173:108655.
267. O'Sullivan JB, Mahan CM. Criteria for the Oral Glucose Test in Pregnancy. Diabetes. 1964;13:278-85.
268. World Health Organization. Diagnostic Criteria and Classification of Hyperglycaemia First Detected in Pregnancy. Geneva: World Health Organization; 2013.
269. Sweeting A, Hare MJL, de Jersey SJ, et al. Australasian Diabetes in Pregnancy Society (ADIPS) 2025 consensus recommendations for the screening, diagnosis and classification of gestational diabetes. Medical Journal of Australia. 2025;223(3):161-7.

270. International Association of Diabetes and Pregnancy Study Groups Consensus Panel. International Association of Diabetes and Pregnancy Study Groups Recommendations on the Diagnosis and Classification of Hyperglycemia in Pregnancy. Diabetes Care. 2010;33(3):676-82.
271. HAPO Study Cooperative Research Group, Metzger BE, Lowe LP, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008;358:1991-2002.
272. Bacha F, Cheng P, Gal RL, et al. Initial Presentation of Type 2 Diabetes in Adolescents Predicts Durability of Successful Treatment with Metformin Monotherapy: Insights from the Pediatric Diabetes Consortium T2D Registry. Horm Res Paediatr. 2018;89(1):47-55.
273. Jones KL, Arslanian S, Peterokova VA, Park JS, Tomlinson MJ. Effect of metformin in pediatric patients with type 2 diabetes: a randomized controlled trial. Diabetes Care. 2002;25(1):89-94.
274. Gottschalk M, Danne T, Vlajnic A, Cara JF. Glimepiride versus metformin as monotherapy in pediatric patients with type 2 diabetes: a randomized, single-blind comparative study. Diabetes Care. 2007;30(4):790-4.
275. Arslanian S, Pyle L, Washington G, et al. Effects of metfromin, metformin plus rosiglitazone, and metfromin plus lifestyle on insulin sensitivity and B-cell funtion in TODAY. Diabetes Care. 2013;36(6):1749-57.
276. TODAY Study Group. Treatment Effects on Measures of Body Composition in the TODAY Clinical Trial. Diabetes Care. 2013;36(6):1742-8.
277. Gandica R, Zeitler P. Update on Youth-Onset Type 2 Diabetes: Lessons Learned from the Treatment Options for Type 2 Diabetes in Adolescents and Youth Clinical Trial. Adv Pediatr. 2016;63(1):195-209.
278. Narasimhan S, Weinstock RS. Youth-onset type 2 diabetes mellitus: lessons learned from the TODAY study. Mayo Clin Proc. 2014;89(6):806-16.
279. Meyers AG, Hudson J, Cravalho CKL, et al. Metformin treatment and gastrointestinal symptoms in youth: Findings from a large tertiary care referral center. Pediatr Diabetes. 2021;22(2):182-91.
280. Cravalho CKL, Meyers AG, Mabundo LS, et al. Metformin improves blood glucose by increasing incretins independent of changes in gluconeogenesis in youth with type 2 diabetes. Diabetologia. 2020;63(10):2194-204.
281. X. Gao PhD MCP, G.A. Burghen MD, P. Velasquez-Mieyer MD, K.T. Moore BA, S.R. Donahue MD, R.A. Reeves MD, K.C. Turner PhD. Pharmacokinetics of Metformin in Pediatric Type 2 Diabetic and Healthy Adult Subjects. Clinical Pharmacology & Therapeutics. 2003;PII-65.
282. Sanchez-Infantes D, Diaz M, Lopez-Bermejo A, Marcos MV, de Zegher F, Ibanez L. Pharmacokinetics of metformin in girls aged 9 years. Clin Pharmacokinet. 2011;50(11):735-8.
283. Hurley-Kim K, Vu CH, Dao NM, et al. Effect of Metformin Use on Vitamin B12 Deficiency Over Time (EMBER): A Real-World Evidence Database Study. Endocr Pract. 2023;29(11):862-7.
284. Sellers EA, Dean HJ. Short-term insulin therapy in adolescents with type 2 diabetes mellitus. J Pediatr Endocrinol Metab. 2004;17(11):1561-4.
285. Shahid M, Shaibi GQ, Baines H, Garcia-Filion P, Gonzalez-Garcia Z, Olson M. Risk of hypoglycemia in youth with type 2 diabetes on insulin. J Pediatr Endocrinol Metab. 2018;31(6):625-30.
286. Shaefer CF, Reid TS, Dailey G, et al. Weight change in patients with type 2 diabetes starting basal insulin therapy: correlates and impact on outcomes. Postgrad Med. 2014;126(6):93-105.
287. Wood JR, Silverstein J. Incretins and amylin in pediatric diabetes: new tools for management of diabetes in youth. Curr Opin Pediatr. 2013;25(4):502-8.
288. Kelly AS, Metzig AM, Rudser KD, et al. Exenatide as a weight-loss therapy in extreme pediatric obesity: a randomized, controlled pilot study. Obesity (Silver Spring). 2012;20(2):364-70.
289. Tamborlane WV, Bishai R, Geller D, et al. Once-Weekly Exenatide in Youth With Type 2 Diabetes. Diabetes Care. 2022;45(8):1833-40.
290. Petri KC, Jacobsen LV, Klein DJ. Comparable liraglutide pharmacokinetics in pediatric and adult populations with type 2 diabetes: a population pharmacokinetic analysis. Clin Pharmacokinet. 2015;54(6):663-70.
291. Arslanian SA, Hannon T, Zeitler P, et al. Once-Weekly Dulaglutide for the Treatment of Youths with Type 2 Diabetes. N Engl J Med. 2022;387(5):433-43.
292. Barrientos-Perez M, Hsia DS, Sloan L, et al. A study on pharmacokinetics, pharmacodynamics and safety of lixisenatide in children and adolescents with type 2 diabetes. Pediatr Diabetes. 2022;23(6):641-8.
293. Malloy J, Capparelli E, Gottschalk M, Guan X, Kothare P, Fineman M. Pharmacology and tolerability of a single dose of exenatide in adolescent patients with type 2 diabetes mellitus being treated with metformin: a randomized, placebo-controlled, single-blind, dose-escalation, crossover study. Clin Ther. 2009;31(4):806-15.
294. Klein DJ, Battelino T, Chatterjee DJ, et al. Liraglutide's safety, tolerability, pharmacokinetics, and pharmacodynamics in pediatric type 2 diabetes: a randomized, double-blind, placebo-controlled trial. Diabetes Technol Ther. 2014;16(10):679-87.

295. Bensignor MO, Bomberg EM, Bramante CT, et al. Effect of liraglutide treatment on body mass index and weight parameters in children and adolescents with type 2 diabetes: Post hoc analysis of the ellipse trial. Pediatr Obes. 2021;16(8):e12778.
296. Chadda KR, Cheng TS, Ong KK. GLP-1 agonists for obesity and type 2 diabetes in children: Systematic review and meta-analysis. Obes Rev. 2021;22(6):e13177.
297. Kelly AS, Auerbach P, Barrientos-Perez M, et al. A Randomized, Controlled Trial of Liraglutide for Adolescents with Obesity. N Engl J Med. 2020;382(22):2117-28.
298. Hannon TS, Chao LC, Barrientos-Pérez M, et al. Efficacy and safety of tirzepatide in children and adolescents with type 2 diabetes (SURPASS-PEDS): a randomised, double-blind, placebo-controlled, phase 3 trial. The Lancet. 2025;406(10511):1484-96.
299. Laffel LMB, Tamborlane WV, Yver A, et al. Pharmacokinetic and pharmacodynamic profile of the sodium-glucose co-transporter-2 inhibitor empagliflozin in young people with Type 2 diabetes: a randomized trial. Diabet Med. 2018;35(8):1096-104.
300. Laffel LM, Danne T, Klingensmith GJ, et al. Efficacy and safety of the SGLT2 inhibitor empagliflozin versus placebo and the DPP-4 inhibitor linagliptin versus placebo in young people with type 2 diabetes (DINAMO): a multicentre, randomised, double-blind, parallel group, phase 3 trial. Lancet Diabetes Endocrinol. 2023;11(3):169-81.
301. Tirucherai GS, LaCreta F, Ismat FA, Tang W, Boulton DW. Pharmacokinetics and pharmacodynamics of dapagliflozin in children and adolescents with type 2 diabetes mellitus. Diabetes Obes Metab. 2016;18(7):678-84.
302. Parkinson J, Tang W, Johansson CC, Boulton DW, Hamren B. Comparison of the exposure-response relationship of dapagliflozin in adult and paediatric patients with type 2 diabetes mellitus. Diabetes Obes Metab. 2016;18(7):685-92.
303. Shehadeh N, Barrett T, Galassetti P, et al. Dapagliflozin or Saxagliptin in Pediatric Type 2 Diabetes. NEJM Evid. 2023;2(12):EVIDoa2300210.
304. Tamborlane WV, Laffel LM, Shehadeh N, et al. Efficacy and safety of dapagliflozin in children and young adults with type 2 diabetes: a prospective, multicentre, randomised, parallel group, phase 3 study. Lancet Diabetes Endocrinol. 2022;10(5):341-50.
305. Tamborlane WV, Polidori D, Argenti D, Di Prospero NA. Pharmacokinetics and pharmacodynamics of canagliflozin in pediatric patients with type 2 diabetes. Pediatr Diabetes. 2018;19(4):649-55.
306. Shamchuk A, Doulla M, Jetha M. Possible association between diabetic ketoacidosis and use of sodium-glucose co-transporter 2 inhibitor in a 17-year-old youth with type 2 diabetes. CMAJ. 2021;193(35):E1385-E8.
307. Fraser IP, Neufeld ND, Fox LA, et al. A randomized clinical trial to evaluate the single-dose pharmacokinetics, pharmacodynamics, and safety of sitagliptin in pediatric patients with type 2 diabetes. Pediatr Diabetes. 2018.
308. Jalaludin MY, Deeb A, Zeitler P, et al. Efficacy and safety of the addition of sitagliptin to treatment of youth with type 2 diabetes and inadequate glycemic control on metformin without or with insulin. Pediatr Diabetes. 2022;23(2):183-93.
309. Shankar RR, Zeitler P, Deeb A, et al. A randomized clinical trial of the efficacy and safety of sitagliptin as initial oral therapy in youth with type 2 diabetes. Pediatr Diabetes. 2022;23(2):173-82.
310. Dudkowski C, Tsai M, Liu J, Zhao Z, Schmidt E, Xie J. The pharmacokinetics and pharmacodynamics of alogliptin in children, adolescents, and adults with type 2 diabetes mellitus. Eur J Clin Pharmacol. 2017;73(3):279-88.
311. Peng XV, Klingensmith G, Hsia DS, et al. A Randomized Phase 3 Study Evaluating the Efficacy and Safety of Alogliptin in Pediatric Participants with Type 2 Diabetes Mellitus. Diabetes Ther. 2025.
312. Tamborlane WV, Laffel LM, Weill J, et al. Randomized, double-blind, placebo-controlled dose-finding study of the dipeptidyl peptidase-4 inhibitor linagliptin in pediatric patients with type 2 diabetes. Pediatr Diabetes. 2018;19(4):640-8.
313. Christensen ML, Meibohm B, Capparelli EV, Velasquez-Mieyer P, Burghen GA, Tamborlane WV. Single- and multiple-dose pharmacokinetics of pioglitazone in adolescents with type 2 diabetes. J Clin Pharmacol. 2005;45(10):1137-44.
314. Christensen ML, Franklin BE, Momper JD, Reed MD. Pediatric drug development programs for type 2 diabetes: A review. J Clin Pharmacol. 2015;55(7):731-8.
315. Nissen SE, Wolski K. Rosiglitazone revisited: an updated meta-analysis of risk for myocardial infarction and cardiovascular mortality. Arch Intern Med. 2010;170(14):1191-201.
316. Vaidyanathan J, Choe S, Sahajwalla CG. Type 2 diabetes in pediatrics and adults: thoughts from a clinical pharmacology perspective. J Pharm Sci. 2012;101(5):1659-71.

317. Donahue SR, Turner KC, Patel S. Pharmacokinetics and pharmacodynamics of glyburide/metformin tablets (Glucovance) versus equivalent doses of glyburide and metformin in patients with type 2 diabetes. Clin Pharmacokinet. 2002;41(15):1301-9.
318. Zhu X, Zhou G, Gu X, et al. Comparing bariatric surgery and medical therapy for obese adolescents with type 2 diabetes. Asian J Surg. 2023;46(10):4337-43.
319. Dobbs TJ, Cree MG, Bailey AJ, et al. Glycemia, Insulin Sensitivity, and Secretion Improve 3 Months Post-sleeve Gastrectomy in Youth With Type 2 Diabetes. J Endocr Soc. 2025;9(3):bvaf020.
320. Alqahtani AR, Elahmedi M, Abdurabu HY, Alqahtani S. Ten-Year Outcomes of Children and Adolescents Who Underwent Sleeve Gastrectomy: Weight Loss, Comorbidity Resolution, Adverse Events, and Growth Velocity. J Am Coll Surg. 2021;233(6):657-64.
321. Ryder JR, Xu P, Nadeau KJ, et al. Effect of surgical versus medical therapy on estimated cardiovascular event risk among adolescents with type 2 diabetes and severe obesity. Surg Obes Relat Dis. 2021;17(1):23-33.
322. Zitsman JL, DiGiorgi MF, Zhang AZ, et al. Adolescent Gastric Banding: a 5-Year Longitudinal Study. Obes Surg. 2020;30(3):828-36.
323. Silberhumer GR, Miller K, Pump A, et al. Long-term results after laparoscopic adjustable gastric banding in adolescent patients: follow-up of the Austrian experience. Surg Endosc. 2011;25(9):2993-9.