**Supplementary Material**

***Country-specific prevalence and incidence of youth-onset type 2 diabetes – a narrative literature review***

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**Table S1. Overview of the search terms applied in the literature search of PubMed**

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| --- | --- | --- | --- | --- | --- |
| Terms | Major | Title | Title and abstract | MeSH | Whole article |
| Pediatric | "Child"; “Child Health”; "Pediatrics"; "Infant"; "Adolescent"; or “Adolescent Health” | -- | “Child”; “Children”; “Childhood”; “Pediatric”; “Paediatric”; “Paediatrics”; “Pediatrics”; “Infant”; “Infants”; “Adolescent”; “Adolescents”; “Teen”; “Teens”; “Adolescence”; “Teenager”; “Teenagers”; “Youth”; or “Youths” | -- | -- |
| T2D | “Diabetes Mellitus, Type 2” | “Diabetes-Mellitus”; "Diabetes"; “Diabetics” AND (“Type 2” OR “Type-2” OR “Type II” OR “Type-II”); “Non-insulin-dependent-diabetes”; “Noninsulin-dependent-diabetes”; “Noninsulin-dependent diabetes”; “Noninsulin dependent diabetes”; “NIDDM”; “Type-II-diabetes”; “Type-2-diabetes”; “T2DM”; or “T2D” | -- | -- | -- |
| Epidemiology | “Epidemiology” | -- | “Epidemiology”; “Epidemiologies”; “Epidemiologic”; “Morbidity”; “Occurrence”; “Prevalence”; “Incidence”; “Endemics”; “Endemic”; “Age at Diagnosis”; “Age Diagnosed”; “Method of Diagnosis”; or “Method to diagnose” | “Geography”; “Geography, Medical”; or “Geographic Locations”  | “Disease Rate”; “Disease Rates” ; “Geography”; “Geographic Variation”; “Regional Difference”; “Regional Differences”; “Regional Analysis”; or “Regional Analyses”  |

MeSH, medical subject headings; NIDDM, non-insulin dependent-diabetes mellitus; T2D, type 2 diabetes; T2DM, type 2 diabetes mellitus.

**Table S2. Prevalence and incidence of pediatric T2D**

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| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Trial name/ reference** | **Study type (period)** | **Country/ location** | **Number of participants/ population size**  | **Age range** | **T2D diagnosis criteria used** | **Age at T2D diagnosis** | **Prevalence rate (measured in cases per population, unless otherwise stated)** | **Incidence rate****(measured in cases per population, unless otherwise stated)** |
| ***North America*** |
| Amed et al. 2010 [2] | Prospective national surveillance study (2006–2008) | Canada | 7,358,935 (227 cases of T2D) | 0–18 y | Canadian Diabetes Association | Mean ± SD: 13.7 ± 2.5 y | NR | 1.54/100,000 children per year |
| Wahi et al. 2009 [3] | Community participatory action research (prior to 2009; specific date not provided) | Canada (Hartley Bay, British Columbia) | 30 | 6–18 y | ADA (2007) guidelines | 10.4 y (age of the one child in the study when diagnosed with T2D) | 1 child had T2D (5 had IFG, 1 had IFG+IGT) (the one child with T2D had no outward symptoms of diabetes) | NR |
| Jabar et al. 2018 [4] | Cross-sectional study of offspring exposed *in utero* to T2D (of First Nation heritage) (study initiated in 2008 and is ongoing) | Canada (Manitoba) | 260 (7–9 y, N=88; 14–16 y, N=27) | 7–9 y and 14–16 y | Diabetes Canada criteria, negative antibody testing, and associated clinical findings | Mean: 11.4 y | 11.9% of the total cohort developed T2D (23.3% of those aged ≥10 y; 44.9% of those aged ≥16 y) | NR |
| Leon et al. 2018 [5] | Cross-sectional retrospective chart review of patients seen in a tertiary care pediatric weight management clinic (May 2012–May 2014) | Canada (Alberta) | 199 | 2–17 y | FPG (≥7.0 mmol/L) (≥126 mg/dL), OGTT (≥11.1 mmol/L) (≥200 mg/dL), or random blood sugar (≥11.1 mmol/L)(≥200 mg/dL); or prior T2D diagnosis | NR | Comorbidity prevalence of T2D: 1.5% (3 cases)(pre-diabetes, 5.5%; 11 cases) | NR |
| Lipman et al. 2013 [6] | Population-based survey (survey completed in 2005; data from 1998–2004) | USA (Philadelphia, Pennsylvania: 510 schools) | 252,896(492 children identified with diabetes) | 4–18 y | Physician diagnosis | Mean ± SD: 11.9 ± 0.5 y | 0.38/1,000 school-aged children | NR |
| Nsiah-Kumi et al. 2013 [7] | Cross-sectional, prospective study (years not stated) | USA (Nebraska) | 3,000 (focused on Native Americans) | 5–18 y | ADA guidelines (using IFG or IGT only) | NR | Of overall diabetes: 1.0% | NR |
| SEARCH study; Bell et al. 2009 [8] | Multicenter, observational study (2001–2005) | USA (6 states: California, Ohio, Colorado, South Carolina, Washington,Hawaii) | 7,284 (with diabetes), this analysis focused on non-Hispanic White youths | <20 y | Physician diagnosis | NR | 0.18/1,000 (in 2001 and in those 10–19 y of age) | 3.7/100,000 (2002–2005; in those 10–19 y of age) |
| SEARCH study; Mayer-Davies et al. 2009 [9] | Multicenter, observational study (2001–2005) | USA (6 states: California, Ohio, Colorado, South Carolina, Washington, Hawaii) | 1,440 (with diabetes), this analysis focused on African American youths | <20 y | Physician diagnosis | Mean: 11.7 y (in those 10–14 y) and 15.1 in those ≥15 y) | 1.06/1,000 (in 2001 and in those 10–19 y of age).There were 16 cases in those <10 y of age between 2002 and 2006 | 19/100,000 (2002–2005; in those 10–19 y of age) |
| SEARCH study; Liu et al. 2009 [10]  | Multicenter, observational study (2001–2005) | USA (6 states: California, Ohio, Colorado, South Carolina, Washington, Hawaii) | 245 (with diabetes), this analysis focused on Asian and Pacific Islander youths | <20 y | Physician diagnosis | NR | 0.52/1,000 (in those 10–19 y of age) | 12.1/100,000 (2002–2005; in those 10–19 y of age) |
| SEARCH study; Dabelea et al. 2009 [11] | Multicenter, observational study (2001–2005) | USA (6 states: California, Ohio, Colorado, South Carolina, Washington, Hawaii for the SEARCH study, this analysis focused on data from Colorado) | 158 (with diabetes), this analysis focused on Asian and Pacific Islander youths | <20 y | ICD-9 code (250.0-250.9) followed by validation using medical records | NR | 1.45/1,000 | 27.74/100,000 |
| SEARCH studyLiese et al. 2010[12] | Observational study (2002–2003) | USA(South Carolina, Colorado, Ohio region and Washington region | 1,968,411 (total population aged 10–19 y) (313 with T2D) | 10–19 y | Physician diagnosis | NR | NR | Rates reported for each region, age group and ethnicity |
| SEARCH studyMayer-Davies et al. 2017 [13] | Multicenter, observational (2002–2012) | USA (five states: California, Ohio, Colorado, South Carolina, Washington) | 2,846 | 10–19 y | Physician diagnosis  | 10–19 y | NR | Unadjusted incidence rates: 2002–2003: 9.0/100,000 youths/year; 2010–2012: 12.5/100,000 youths/year.Relative annual increase (after adjustment for age, sex, race or ethnicity)=4.8% |
| SEARCH studyDabelea et al. 2014 [14] | Multicenter observational study (2001 and 2009) | USA (five states: California, Ohio, Colorado, South Carolina, Washington and American Indian reservations in Arizona and New Mexico) | 1.7 million in 2001 and 1.8 million in 2009 | 10–19 y | Physician diagnosis | 10–19 y | 2001= 0.34/1,000, 2009= 0.46/1,000;30.5% increase in T2D between 2001 and 2009 | NR |
| Chen et al. 2019 [15] | Data derived from the Truven Health MarketScan Multi-State Medicaid Database (2002–2016) | USA | 73,593 with diabetes (40,109 with T2D) | <18 y | ICD-9/-10-Clinical Modification codes | 89.7% were 12–18 y | Annual prevalence of T2D increased from 0.70/1,000 (2002) to 2.76/1,000 (2011), but then dropped to 2.12/1,000 (2016)Prevalence was higher in girls and Black children | NR |
| SEARCH studyPowell et al. 2019 [16] | Multicenter, observational study (active surveillance of Indian Health Service databases) (incidence: 2002–2013; prevalence: 2001, 2009) | USA (Navajo Nation) | 40,000–50,000(356 with T2D; incident cases since 2002, N=211; prevalent cases: 2001, N=75; 2009, N=70) | 10–19 y | NR | NR | 146.6/100,000 (95% CI: 116.8; 184.0) in 2001; 141.5/100,000 (95% CI: 112.0; 178.8) in 2009 (p=0.65 from 2001 to 2009) | 26.3/100,000 (95% CI: 20.1; 34.3) in 2002–2005; 34.8/100,000 (95% CI: 27.5; 44.1) in 2006–2009; 51.7/100,000 (95% CI: 42.1; 63.5) in 2010–2013 (overall p<0.001) |
| Pollock et al. 2018 [17] | Longitudinal, early life-course data from the Bogalusa Heart Study (1973–2010) | USA | 7,725 | At baseline: 6.6–11.6 yAt follow-up: 13.6–29.9 | Currently taking oral antidiabetic medication, current diagnosis of T2D, and/or FPG >126 mg/dL | NR | NR | Cumulative incidence of 2.3% (176 cases of incident T2D)Males, 2.1%; females, 2.4% (p=0.39) |
| ***Central and Latin America*** |
| Washington et al. 2013 [18] | Retrospective, medical record review (2001–2010) | US Virgin Islands | 37,093 (91 diagnosed) | 0–19 y | ICD-9 codes within medical records | Mean ± SD: 14.24 ± 3.61 y for Non-Hispanic Black, 13.49 ± 4.6 y for other | NR | 9.6/100,000 (95% CI: 6.8; 13.5) |
| Batson et al. 2013 [19] | Cross-sectional study (Jan–June 2009) | West Indies (Trinidad: 415 schools)  | 67,000 screened, population size =250,00 (7 identified with T2D) | 5–17 y | Glycosuria test for initial screening, followed by OGTT. ADA criteria used | NR | T2D: 10.4/100,000 | NR |
| Telo et al. 2019 [20] | Cross-sectional school-based multicenter study (in cities with more than 100,000 inhabitants) (February 2013–November 2014) | Brazil | 37,854 | 12–17 y | ADA criteria (T2D: self-report, fasting glucose ≥126 mg/dL, or HbA1c ≥6.5%;Prediabetes: fasting glucose 100–125 mg/dL or HbA1c 5.7–6.4%) | NR | T2D: 3.3% (95% CI: 2.9; 3.7)Prediabetes: 22.0% (95% CI: 20.6; 23.4)  | NR |
| Hernández-Montoya et al. 2019 [21] | Analytical, ecologic study of trends over time(2003–2013) | Mexico | Female early adolescents, 5,557,805–5,534,420;female late adolescents, 5,242,068–5,547,538;male early adolescents, 5,587,610–5,746,298;male late adolescents, 5,129,833–5,623,359 | 10–19 y (early adolescence, 10–14 y; late adolescence, 15–19 y) | Physician diagnosis (consistent with WHO criteria) | NR | NR | Early adolescence: stable over time and similar in both sexes (annual standardized cumulative incidence ranged from 0.13–0.31 cases/100,000 females and 0.13–0.26 cases/100,000 males) Late adolescence: linear increase and higher in females (annual standardized cumulative incidence ranged from 0.56–1.01 cases/100,000 females and 0.36–0.56 cases/100,000 males) |
| ***Europe*** |
| Shield et al. 2009[22] | Prospective study (2004–2005) | UK | 76 cases of T2D  | 0–16 y | Raised fasting insulin(0.132 pmol/L (20 mU/ml) or equivalent) or fasting C-peptide concentrations (0.600 pmol/L) and/or the absenceof autoimmune antibodies found in T1D with no insulin requirement 1 year after diagnosis  | NR | NR | 0.6/100,000 per year |
| Abbasi et al. 2017 [23] | Cohort with nested case controlusing data from UK Clinical Practice Research Datalink)(1994–2013) | UK (375 general practices) | 369,362 | 2–15 y | Prescription of oral glucose-lowering medications only, or diagnosis of diabetes mellitus or HbA1c ≥6.5% (48 mmol/mol) but no insulin prescription | NR | NR | 1994–1998: 6.4/100,000; 2009–2013: 33.2/100,000  |
| Shepherd et al. 2016 [24] | Systematic study of patients at 6 pediatric diabetes clinics (years of study not stated) | UK(six clinics) | 808 had diabetes and completed the study | <20 y | In addition to diagnosis, UCPCR was tested along with GAD/IA2 antibodies | Median age at diabetes diagnosis (type not specified)= 8 y | NR | T2D was diagnosed in 3.3% |
| Khanolkar et al. 2016 [25] | Cross-sectional study of a national diabetes register (2012–2013) | England and Wales, UK (178 centers) | 307 patients with T2D; population: 10,579,132 | <16 y | Physician diagnosis | Mean: 13 y (across all ethnicities) | 2.9/100,000 (95% CI: 2.6; 3.2).  | NR |
| Candler et al. 2018 [26] | Prospective monthly surveillance of >3400 consultant pediatricians (April 2015–April 2016) | UK and Republic of Ireland | 106 newly confirmed T2D cases (104 from the UK, 2 from the Republic of Ireland) | <17 y | ADA (2000) guidelines | Median (range): 14.3 (7.9–16.9) y | NR | Comparing 2005 with 2015 data, UK incidence of 0.72/100,000 (95% CI: 0.58; 0.88)(White children: 0.44/100,000 [95% CI: 0.31; 0.60]; Asian children: 2.92/ 100,000 [95% CI: 2.00; 4.12]; Black children: 1.67/ 100,000 [95% CI: 0.77; 3.18])Increased incidence from 2005 among girls and children of South-Asian ethnicity |
| Schober et al. 2009 [27] | Prospective, population-based incidence study(1999–2007) | Austria | 1,378,239 (1999 census),31 cases with T2D | 0–15 y | ADA criteria | NR | NR | Incidence for each year was<0.6/100,000(1.7% of all cases of diabetes diagnosed) |
| Schober et al. 2009 [28] | Observational study (2008) | Germany and Austria (262 centers) | 40,567 with diabetes(562 having T2D) | 0–20 y (but diagnosed >18 y) | ADA and ISPAD | Median: 13.6 y | 1.4% of the pediatric diabetes population | NR |
| Neu et al. 2009 [29] | Population-based study (2004–2005) | Germany (Baden-Württemberg) | 2,400,000 (1987 census) (56 with T2D) | 0–20 y | Physician diagnosis (with genetic testing to differentiate between MODY and T2D) | NR | 2.30/100,000 | NR |
| Neu et al. 2017 [30] | Cross-sectional survey study (2016 vs 2005/2005) | Germany (Baden-Württemberg)  | 50 in 2016 vs 56 in 2004/2005with T2D; 2.4 million population | <20 y | Physician diagnosis | NR | 2016= 2.42/100,000 (95% CI: 1.75; 3.09)2004/2005= 2.30/100,000 (95% CI: 1.70; 2.90) | NR |
| Cambuli et al. 2009 [31] | Observational study(2007–2008) | Italy (Cagliari) | 736 children were screened, of which 535 were overweight/ obese and included in the study | 2–20 y | ADA criteria, with antibody titers used to differentiate between T1D and T2D | NR | 0.18% (1 of the 535 children included) | NR |
| Brufani et al. 2010 [32] | Observational study (1997–2007) | Italy(central region) | 510 children with overweight and obesity | 3–18 y | ADA criteria (FPG) | NR | 0.4% of the children included in the study | NR |
| Aguayo et al. 2013 [33] | Prospective, observational study(2005–2009) | Spain/Basque region | 136 (inclusion criteria: obese, Caucasian) | 6.2–14.5 y | OGTT results assessed using the ADA guidelines; and HOMA used for IR (where resistance to insulin = HOMA ≥3.8) | Not applicable | 0 | NR |
| O’Dea et al. 2017 [34] | Cross-sectional survey (2015) | Republic of Ireland (19 centers) | 12 with T2D; 1,036,817 population size | <16 y | Diagnosis validated using ISPAD 2014 criteria | NR | 1.2/100,000 (95% CI: 0.6; 2) | NR |
| Oester et al. 2016 [35]  | Cross-sectional study of a national diabetes register(2014) | Denmark | 7 with T2D; population size not stated | 0–16 y | Physician diagnoses, which were then validated using ADA/ ISPAD guidelines | Mean: 11.3 y | 0.6/100,000 | NR |
| Haliloğlu et al. 2018 [36] | Single-center, observational, retrospective record review (January 1999–December 2016) | Turkey (Istanbul) | 835 | 0–18 y | ISPAD Consensus 2014 | Mean ±SD: 8.8 ± 4.4 y | T2D: 5.7% (N=48)T1D: 84% (N=701)Clinical MODY: 5.3% (N=44)Other diabetes: 5% (N=42)T2D was more common in girls and older children | NR |
| ***Middle East and North Africa*** |
| Moadab et al. 2010 [37] | Cross-sectional, population-based study (year not stated, likely to be 2007) | Iran (Isfahan) | 7,554 (672 children with overweight and obesity selected) | 6–19 y | ADA criteria | 18 y | 0.1% (1 of the 672 children who were screened) | NR |
| Al-Rubeaan et al. 2015 [38] | Population-based study(2007–2009) | Saudi Arabia (13 administrative regions) | 25,523 | ≤18 y | ADA criteria used to classify patients with diabetes. Those without known diabetes were classified based on FPG assessment |  | Overall prevalence of diabetes (T1D + T2D) = 10.84% | Newly identified cases of diabetes (T1D + T2D) = 4.27% |
| Al-Agha et al. 2012 [39] | Retrospective, cross-sectional study (2006–2010) | Saudi Arabia (Jeddah: 1 clinic)  | 387 | 2–18 y | Serum insulin for initial screening. Physician diagnosis | 13.1±2.02 years for T2D | Hyperinsulinism: 44.7%;T2D: 9.04% | NR |
| Moussa et al. 2008 [40] | Medical records maintained by schools(2000–2002) | Kuwait (182 schools) | 128,918 | 6–18 y (mean age: 14.2, SD: 3.0 y) | WHO and ADA guidelines | NR | 34.9 per 100,000 (45 children identified) | NR |
| Osman et al. 2013 [41] | Retrospective, descriptive hospital-based study(2006–2009) | Sudan (one clinic) | 958 | Not stated | Factors involved in diagnosis included: symptoms at onset, obesity, acanthosis nigricans, other features of metabolic syndrome, family history of T2D and availability of abnormal insulin, or C-peptide levels as well as treatment administered | 11–18 y (92.1%) and <10 y (7.9%) | NR | 38 of the 958 patients (4%) had T2D |
| Alyafei et al. 2018 [42] | Prospective cohort study (January 2012–December 2016) | Qatar | 45 with T2D (440 with T1D) | 0.5–14 y | NR | Age at presentation: 24.1% 5–9.99 y; 76.9% 10–14 y | NR | 1.16/100,000 in 2012 increasing to 2.72/100,000 in 2016.Incidence rate of T2D equal to 2.9/100,000 per year, and an annual increased incidence of 3.12% |
| ***Asia*** |
| Dedov et al. 2017 [43] | Analysis of database registry up to 31 Dec 2016 | Russian Federation (79 regions) | 4,001,860 (total population, including adults) | Children and adolescents (specific age ranges not provided) | NR | NR | 5.34 per 100,000 population (children); 6.82 per 100,000 population (adolescents) | 1.52 per 100,000 population (children); 0.92 per 100,000 population (adolescents) |
| Jin et al. 2011[44] | (2000–2011) | NR (China\*) | 503 cases of diabetes (31 cases of T2D) | NR | NR | NR | 0.18/1,000 (2006-2011) | NR |
| Wu et al. 2017 [45] | Population-based registry (2007–2013)  | China (Zhejiang) | 392 | 5–19 y | WHO criteria for diabetes with differential diagnosis based on: frequent ketoacidosis or ongoing insulin therapy or antibodies or C-peptide levels | NR | NR | Mean annual age-standardized incidence =1.96/100,000 person yearsStandardized incidence rate increased from 0.72 in 2007 to 3.64 in 2013 |
| Chi et al. 2018 [46] | Data from the China Nutrition and Health Survey and China Nutrition and Health Surveillance (2002–2012) | China | 243,479 in 2002 and 183,137 in 2012 | 7–17 y | FPG and 2 h-OGTT levels | NR | In 2002: diabetes, 0.24%; IFG, 0.33% (both were higher in urban vs rural areas)In 2012: diabetes, 0.52%; IFG, 1.95% (both were higher in rural vs urban areas) | NR |
| Chen et al. 2016 [47] | Nationwide longitudinal study using the National Health Insurance Research Database in individuals with ASD(diagnosed with ASD between 2002–2009, study continued to 2011) | Taiwan | 6,122 patients with ASD and 24,488 age- and sex-matched controls | 10–29 y (adolescents 10–17 y, young adults 18–29 y) | Diagnosis of T2D (ICD-9-CM code 250.x0 and 250.x2, x=0-9) provided by healthcare professionals based on laboratory exams | ASD cohort= 22.22 y; control cohort= 23.06 y | NR | Incidence of T2D for the ASD and control cohorts = 2.63/1,000 person years vs 0.63/1,000 person years.ASD was associated with a higher risk of developing T2D (HR 2.71 for adolescents and 5.31 for young adults) |
| Tung et al. 2018 [48]  | Retrospective and prospective childhood diabetes registry (2008–2017) | Hong Kong | 889 (with diabetes: 498 with T1D; 391 with T2D) | <18 y | ISPAD | Mean ± SD: 14.7 ± 2.1 y | NR | 1.9/100,000 (increased by 8.4% annually 2008–2017) |
| Panamonta et al. 2010 [49] | Cross-sectional, prospective study (2006) | Thailand (Kohn Kaen)  | 2,156 school children (4 withT2D) | 10–15 y | WHO | Range: 10–12 y | 2.2% of the 594 children who were overweight or obese | NR |
| Khemaprasit et al. 2018 [50] | Cross-sectional study of a pediatric diabetes registry at a single center (March 2016–July 2018) | Thailand (Bangkok) | 295 (with diabetes: 229 with T1D; 45 with T2D) | <18 y | NR | Mean ± SD: 12.20 ± 2.45 y | 15.3% of the 295 children enrolled | NR |
| Mungklarat et al. 2018 [51] | Retrospective review of overweight and obese children at a single center (January 2002–December 2016) | Thailand (Bangkok) | 204 | 6–18 y | NR | NR | 1% (n=2) had T2D (0.5% IFG; 17.6% IGT; 1.5% both IFG and IGT) | NR |
| MEDI study; Unnikrishnan et al. 2008 [52] | Cross-sectional study of tertiary-care hospitals (period not stated) | India (seven institutions) | 603 | <20 y  | ADA 2004 guidelines | Mean ± SD: 16.2 ± 2.9 y | 6% (36 patients; this is not a national prevalence, but just in this study) | NR |
| Zabeen et al. 2016 [53] | Observational cross-study (2011–2015) | Bangladesh (one center)  | 77 with T2D; 939, population size at the clinic | ≤18 y | Presence of obesity or overweight; positive family history; presence of acanthosis nigricans; other features of metabolic syndrome, for example,polycystic ovary syndrome and dyslipidemia; availability of fasting insulin level | Of those with T2D, 14% were 9–10 y,60% were 11–14 y, 26% were 15–17 y | 8% of the 939 patients registered at the clinic | NR |
| Fuziah et al. 2008 [54] | Diabetes in Children and Adolescents Registry (April 2006 – June 2007) | Malaysia | 240 in total | ≤20 y (mean: 12.51 y) | Clinical diagnoses, including: hyperosmolar symptoms, obesity, and acanthosis nigricans | For all types of diabetes: mean ± SD, 8.31 ± 4.13 | (240 patients in total, 41 with T2D) | NR |
| Urakami et al. 2018 [55] | Urine glucose screening program at schools in the Tokyo metropolitan area (1975–2015) | Japan (Tokyo) | 11,652,205 (301 with T2D; 64 primary school students and 237 junior high school students) | 6–15 y | Clinical manifestations (PG, HbA1c levels [WHO criteria] and OGTT levels) | NR | NR | 2.58/100,000/year in all students0.80/100,000/year in primary school students and 6.41/100,000/year in junior high school students (p<0.0001) |
| ***Australia and Pacific*** |
| Haynes et al. 2016 [56] | Retrospective, population-based cohort; used the Western Australian Children’s Diabetes Database (1990–2012) | Australia (Western Australia) | (Overall population not stated, but taken from the Australian Bureau of Statistics; 135 cases of T2D identified) | <17 y | ADA guidelines current at time of the publication, based on both clinical and laboratory findings | Mean ± SD: 13.3 ± 2.0 y | NR | 1.3 per 100,000 person years |
| Jefferies et al. 2012 [57] | Retrospective analysis population-based treatment referral cohort (1995–2007) | New Zealand(Auckland)  | 300,000 (795 with diabetes and 52 with T2D) | 0–15 y | Physician diagnosis (antibody titers were considered) | Mean ± SD: 12.9 ± 1.8 y | NR | 1.3/100,000 (95% CI: 1.0; 1.8) |
| Sjardin et al. 2018 [58] | Retrospective analysis population-based treatment referral cohort (1995–2015) | New Zealand (Auckland: single center) | 318,000 (104 with T2D) | <15 y | Physician diagnosis (antibody titers were considered) | Mean ± SD: 12.9 ± 1.9 y | NR | 1.5/100,000/year (95% CI: 1.2; 1.9)Higher rates in Pacific island (5.9/100,000) and Maori (4.1/ 100,000) populations |
| Ogle et al. 2016 [59] | Cross-sectional study, using the IDF LFAC data (2001–2012) | Fiji (three pediatric centers) | 42 with diabetes / population size interpolated from 1996 data, where school-going population: 274,377 and from 2007 data, when: 243,121 | 0–15 y | Diabetes diagnosed on the basis of history and clinical features (C-peptide and antibody tests were not available in Fiji at this time) | Mean ± SD: 12.2 ± 2.7 y | 2001: 0.38/100,0002012:2.4/100,000 | 0.43/100,000 |

\*Country not specifically stated in the abstract but assumed, based on author affiliation.

ADA, American Diabetes Association; ASD, Autism Spectrum Disorder; CI, confidence interval; DD, double diabetes; FPG, fasting plasma glucose; GAD, glutamic acid decarboxylase; HbA1c, glycated hemoglobin; HOMA, homeostatic model assessment; IA2, insulinoma antigen 2; ICD, international classification of diseases; IDF, International Diabetes Federation; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; IR, insulin resistance; ISPAD, International Society for Pediatric and Adolescent Diabetes; LFAC, Life for a Child Program; MODY, maturity onset diabetes of the young; NR, not reported; OGTT, oral glucose tolerance test; PG, plasma glucose; SD, standard deviation; T1D, type 1 diabetes; T2D, type 2 diabetes; UCPCR, urinary C-peptide creatinine ratio; WHO, World Health Organization; y, years.

**Table S3.** **National and international guidelines for the diagnosis of pediatric T2D**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Country (reference)** | **Government/ association, year** | **Based on ADA/WHO/ISPAD** | **BG levels (FPG and OGTT)** | **Antibody measurement** | **HbA1c** | **Other specifics mentioned, focusing on screening** |
| ***Country-specific*** |
| Austria[60] | Austrian Diabetes Society (Österreichische Diabetes Gesellschaft), 2019 | Not mentioned  | FPG ≥126 mg/dL (7.0 mmol/L) (fasting for 8 h)orOGTT ≥200 mg/dL (11.1 mmol/L) (glucose administered by body weight) | Antibody titers may be used to differentiate between T1D and T2D | >6.5% (>48 mmol/mol), but see caveat in next cell | Screening is recommended for children/adolescents at risk of developing T2D.Diagnostic criteria for adults and children are the same, although it remains unclear whether same HbA1c cut-off should be used for diagnosis in children/ adolescents as the studies that formed the basis of recommending HbA1c for diagnosis included only adult populations. Differential diagnosis between T1D and T2D can be difficult, as symptoms have a high degree of variability |
| Canada [61] | Diabetes Canada/Canadian Diabetes Association, 2018 | Not stated as such | FPG is recommended (but fasting may be challenging for children); OGTT may also be used, but its reproducibility is poor | All children with diabetes should be screened for autoantibodies to ensure T1D and T2D are correctly identified | ≥6.0% (≥42 mmol/mol) | Children at high risk for T2D should be screened every 2 years |
| Egypt‡ | NR‡ | ISPAD | (see ISPAD below) | NR | (see ISPAD below) | No national, government-funded screening of high-risk children undertaken (some private insurance and academic screening occurs) |
| India [62] | NR† | ADA and WHO | FPG ≥7.0 mmol (126 mg/dL);OGTT ≥11.1 mmol (200 mg/dL) | Not mentioned | ≥6.5% (≥48 mmol/mol) | OGTT is usually not required, but useful in diagnosing T2D, monogenic diabetes or cystic fibrosis-related diabetes |
| Israel [63] | Israeli Association of Endocrinology, Israel Association of Pediatric Endocrinology,Israel Association of Pediatrics,Family Medicine Association,Israeli Association of Internal Medicine,Israel Diabetes Association,National Diabetes Council, 2016 | Not mentioned | No screening directions provided | Antibody test is recommended to distinguish between T1D and T2D | No screening directions provided | T2D is often accompanied by obesity, insulin resistance and insulin deficiency |
| Malaysia [64] | Ministry of Health, Malaysia, 2015 | Adapted from ISPAD 2014 | Blood glucose in symptomatic individuals:Random: ≥11.1 mmol/L (200 mg/dL) orfasting: ≥7.0 mmol/L (126 mg/dL): have T2D; if values lower than these cut-off points, then use OGTT. If OGTT ≥11.1 mmol/L (200 mg/dL) at 2 h, then T2D.If asymptomatic but at risk, need record of two abnormal glucose tests | Use presence of autoantibodies to differentiate between T1D and T2D | HbA1c not appropriate for use in those <18 y of age for diagnosis. Treatment goal HbA1c <6.5% (<48 mmol/mol). At diagnosis: if HbA1c <9% (<75 mmol/mol) – and otherwise asymptomatic, including no acidosis – treatment includes metformin and lifestyle intervention; if HbA1c >9% (>75 mmol/mol) and no acidosis, basal insulin should be added to metformin and lifestyle intervention; if HbA1c >9% (>75 mmol/mol) with acidosis, treat like T1D until acidosis resolves and continue with basal insulin, metformin and lifestyle intervention | Screen patients if symptomatic or overweight and have two of the following risk factors:-Maternal history of diabetes;-Family history of T2D (first- or second degree relative);-Signs of insulin resistance.Screen every 2 years.Fasting insulin and C-peptide may also be used (but with caution due to overlap in first 2 years of diagnosis) |
| Mexico [65] | Ministry of Health, Mexico, 2015 | Not mentioned | FPG >126 mg/dL (7.0 mmol/L) with symptoms confirms T2D; random/casual BG ≥200 mg/dL (11.1 mmol/L) with no symptoms requires another abnormal BG test to confirm T2D | Not mentioned | OGTT is recommended when HbA1c is≥6.5% (≥48 mmol/mol) for thin children;≥5.7% (≥39 mmol/mol) for overweight/obese children | Screening can be used. HbA1c alone is not confirmatory of T2D; further tests are needed |
| Russia [66] | Ministry of Health, Russian Federation, 2017  | ADA | Not mentioned in main criteria, but FPG and OGTT can help to determine IR, with the immunoreactive insulin levels | Used to differentiate between T2D and T1D | Not used | Children’s weight and fasting C-peptide/insulin levels are also used |
| UK [67] | NICE, 2015 (update published in 2017, but still carries the 2015 date) | Yes, WHO 2006 (assume T1D and confirm using the WHO 2006 report guidelines, unless there are strong indications of another type of diabetes) | Not mentioned | Do not measure C-peptide or other disease-specific autoantibody titers at initial presentation. Only measure these if there is trouble distinguishing between types of diabetes, as the tests have better discriminative value the longer the time between initial presentation and test completion | Not mentioned | T2D risk factors:-Strong family history of T2D;-Obese at presentation;-Black or Asian family origin;-No insulin requirement (or require >0.5 units/kg body weight/day, after partial remission phase);-Show evidence of IR.(screening not mentioned in this guideline) |
| USA [68, 69] | American Diabetes Association, 2019 | NR | NR | Consider antibody testing to exclude T1D diagnosis | ≥6.5% (48 mmol/mol). The test should be performed using a NGSP-certified method and standardized to the DCCT assay. In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing | Test for T2D after the onset of puberty or after 10 y of age, whichever occurs earlier, if:-Overweight (for age) AND one or more of the following;-Maternal history of diabetes;-Family history of T2D (first- or second-degree relative);-Race/ethnicity (Native American, African American, Asian American, Latino, Pacific Islander);-Signs of insulin resistance |
| *Global* |
| ISPAD [70, 71] | ISPAD, 2018 | ADA 2018 guidelines | FPG: ≥126 mg/dL (≥7.0 mmol/L); 2-h post-load OGTT: ≥200 mg/dL (≥11.1 mmol/L) | May be used to differentiate between T1D and T2D | >6.5% (>48 mmol/mol), but the usefulness of HbA1c is unclear in some situations) | Classic symptoms of diabetes (including hyperglycemic crisis of FPG: ≥200 mg/dL (≥11.1 mmol/L)) (confirm diabetes first, then determine which type, using: increased fasting C-peptide level may be indicative of T2D (but there may also be some overlap between T2D and T1D in this parameter); presence of acanthosis nigricans can be indicative of T2D.(general screening of obese youth outside research settings not likely to be cost-effective) |
| WHO [72] | WHO, 2006 | The last guidelines published by the WHO on definition and diagnosis of diabetes mellitus did not specify guidelines for pediatric T2D. In the WHO global report of 2016 [73], it was acknowledged that sophisticated laboratory tests are needed to differentiate between T1D and T2D, so global data cannot be divided into T1D and T2D. Any publication based on WHO diagnostic guidelines, therefore, have used criteria devised for adults rather than children.In 2019, the WHO released a new document on the classification of diabetes, including the use of age at diagnosis as a guide to subtyping diabetes [74]. They suggested that at age 10 to <25 years, clinical features favoring a diagnosis of T2D rather than T1D include: being overweight or obese, age above 10 years, strong family history of T2D, acanthosis nigricans, undetectable islet autoantibodies, and elevated or normal C-peptide |

†Information from non-institutional reference provided by Paturi Vishnupriya Rao. ‡Information provided as a personal communication by Mona Hafez. ADA, American Diabetes Association; BG, blood glucose; DCCT, Diabetes control and complications trial; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; OGTT, oral glucose tolerance test; NGSP, national glycohemoglobin standardization program; NICE, National Institute for Health and Care Excellence; IR, insulin resistance; ISPAD, International Society for Pediatric and Adolescent Diabetes; NR, not reported; T1D, type 1 diabetes; T2D, type 2 diabetes; WHO, World Health Organization; y, years.

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**Figure S1. Flow chart of literature search results to identify youth-onset T2D epidemiology studies**

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\*Asia Pacific Paediatric Endocrine Society and Latin American Pediatric Endocrinology Society congresses. T2D, type 2 diabetes. Based on guidelines from Moher et al., 2009 [1].