Type 2 Diabetes, Medication-Induced Diabetes, and Monogenic Diabetes in Canadian Children

A prospective national surveillance study

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OBJECTIVE — To determine in Canadian children aged <18 years the *1*) incidence of type 2 diabetes, medication-induced diabetes, and monogenic diabetes; *2*) clinical features of type 2 diabetes; and *3*) coexisting morbidity associated with type 2 diabetes at diagnosis.

RESEARCH DESIGN AND METHODS — This Canadian prospective national surveillance study involved a network of pediatricians, pediatric endocrinologists, family physicians, and adult endocrinologists. Incidence rates were calculated using Canadian Census population data. Descriptive statistics were used to illustrate demographic and clinical features.

RESULTS — From a population of 7.3 million children, 345 cases of non–type 1 diabetes were reported. The observed minimum incidence rates of type 2, medication-induced, and monogenic diabetes were 1.54, 0.4, and 0.2 cases per 100,000 children aged <18 years per year, respectively. On average, children with type 2 diabetes were aged 13.7 years and 8% (19 of 227) presented before 10 years. Ethnic minorities were overrepresented, but 25% (57 of 227) of children with type 2 diabetes were Caucasian. Of children with type 2 diabetes, 95% (206 of 216) were obese and 37% (43 of 115) had at least one comorbidity at diagnosis.

CONCLUSIONS — This is the first prospective national surveillance study in Canada to report the incidence of type 2 diabetes in children and also the first in the world to report the incidence of medication-induced and monogenic diabetes. Rates of type 2 diabetes were higher than expected with important regional variation. These results support recommendations that screening for comorbidity should occur at diagnosis of type 2 diabetes.

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Until recently, childhood diabetes was predominantly due to autoimmune type 1 diabetes (1). The emergence of type 2 diabetes, medication-induced diabetes, and improved recognition of monogenic forms of diabetes has altered the pediatric diabetes landscape.

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The increase of type 2 diabetes in children parallels rising rates of childhood obesity. There are, however, insufficient population-based data documenting epidemiological trends. The only prospective national surveillance study from the U.K. estimated the incidence of type 2 diabetes to be 0.53 per 100,000 per year in children <17 years of age (2). A multicenter population-based study from the U.S. reported an incidence of 8.1 per 100,000 person-years and 11.8 per 100,000 person-years in children aged 10–14 and 15–19 years, respectively (3). Remaining data on childhood type 2 diabetes are not population-based and therefore are limited in their generalizability. The potential impact of childhood type 2 diabetes on workforce productivity and health care systems should not be underestimated. The development of diabetes-related micro- and macrovascular complications occurs in young adulthood (4,5). Thus, early cardiovascular disease related to obesity amplifies the morbidity associated with childhood type 2 diabetes (6).

There are limited epidemiological data available on other forms of non–type 1 diabetes. Greenspan et al. (7) reported that 7% of children were affected by medication-induced diabetes after renal transplant and 50% of these children were obese. Monogenic forms of diabetes account for ~1–5% of all cases of diabetes (8) with a minimum prevalence of 0.17 per 100,000 reported in children in the U.K. (9).

Data on pediatric type 2 diabetes in Canada, although limited to specific populations and geographic regions, indicate that the prevalence is increasing (10–13). There are no Canadian data on the incidence of medication-induced or monogenic diabetes in children. In this study, "children" refers to individuals aged <18 years and "non–type 1 diabetes" includes type 2 diabetes, medication-induced diabetes, and monogenic diabetes. We conducted a prospective, national surveillance study in Canadian children aged

Table 1—*Minimum incidence rates of type 2 diabetes, medication-induced diabetes, and monogenic diabetes in Canadian children aged <18 years*

Regions	Population estimate*	Incidence rates (per 100,000 children per year)			Total and participating family physicians, pediatricians, and adult endocrinologists					
					FP		Peds		AE	
		T2D	MID	MD	T†	P	T‡	P	T‡	P
Canada	7,358,935	1.54	0.4	0.2	31,127	98	2,835	2,567	368	49
Alberta	775,175	0.7	0.15	0.2	3,176	8	353	288	29	4
British Columbia	846,140	1.2	0.2	0.25	4,525	6	304	264	31	5
Manitoba	276,925	12.45	0.9	0.55	1,060	9	139	123	6	3
Ontario	2,720,310	1.7	0.6	0.2	10,656	50	1,131	988	147	17
Quebec	1,549,215	0.55	0.2	0.3	8,147	5	664	662	136	12
Atlantic Provinces§	1,136,545	0.7	0.2	0.05	2,521	10	188	188	16	8
Saskatchewan	233,900	0.4	0	0	948	10	53	51	3	0
Territories‖	31,235	0	0	0	94	0	3	3	0	0

*Data from 2006 Canadian Census—Statistics Canada. †Source: *Geographic Distribution of Physicians in Canada: Beyond How Many and Where*. Ottawa, ON, Canada, Canadian Institute for Health Information, 2005. ‡Source: Canadian Medical Directory. §Nova Scotia, New Brunswick, Prince Edward Island, Newfoundland, and Labrador. ‖Northwest Territories, Yukon, and Nunavut. AE, adult endocrinologists; FP, family physicians; MD, monogenic diabetes; MID, medication-induced diabetes; P, participating; Peds, pediatricians; T, total; T2D, type 2 diabetes.

<18 years to determine the *1*) incidence of non–type 1 diabetes, *2*) clinical features of type 2 diabetes at diagnosis, and *3*) comorbidity associated with type 2 diabetes at diagnosis.

RESEARCH DESIGN AND METHODS

— We established a national network of physicians who participated in the surveillance study. Surveillance was conducted in collaboration with the Canadian Pediatric Surveillance Program (CPSP) and the College of Family Physicians of Canada (CFPC)–National Research System (NaReS), both nationally recognized surveillance programs. The CPSP comprises >90% of practicing pediatricians in all regions of Canada and reported an average monthly response rate of 83% and a detailed questionnaire response rate of >90% in previous surveillance studies (14). NaReS, a network of the CFPC, comprises ~14,500 active members and, in surveillance initiatives for influenza, reported a response rate of 75.2% (15).

Physician recruitment

All Canadian pediatricians participated in surveillance (n = 2,560). Although rare pediatric conditions are seen only by pediatric practitioners, and most children with the diagnosis of diabetes are referred to pediatric physicians, some youth, particularly with type 2 diabetes, may be seen only by family physicians or adult endocrinologists. Therefore, innovative to this CPSP surveillance study was the recruitment of family practitioners and adult endocrinologists from across Canada. A targeted and enriched sample of family physicians and nurse practitioners was recruited into the study. A list of practitioners who self-identified through the CFPC as practicing pediatric, adolescent, Aboriginal, and rural or inner-city medicine in northern Canada or core urban areas was generated from a database housed at NaReS (n = 2,823). This database includes clinical practice information and demographics on ~16,000 practicing family physicians in Canada. The above identifiers were chosen to increase the likelihood of including physicians encountering a case of non–type 1 diabetes in a child. A letter was sent to these practitioners requesting participation and asking whether they had previously encountered a case of non–type 1 diabetes in a child. Feasibility allowed the involvement of 100 family physicians, and, therefore, those who agreed to participate *and* had previously seen a case of non–type 1 diabetes in a child in their practice were included. Adult endocrinologists from across Canada were identified using the Canadian Medical Association Directory (n = 335), and a convenience sample was generated by accepting all adult endocrinologists who agreed to participate. In total, 98 family physicians, 49 adult endocrinologists, and 2,567 pediatricians participated with geographic representation from across Canada (Table 1).

Surveillance methodology

Physicians were surveyed for 24 months between 1 April 2006 and 30 March 2008. All physicians received an introductory package that included a case definition (16). Physicians were asked to report new patient cases when there was uncertainty about the diagnosis and when an initial diagnosis of type 1 diabetes was revised to non–type 1 diabetes. A monthly reporting form was mailed out requiring a "yes" or "no" response to the identification of a new patient. A detailed questionnaire was subsequently sent to each physician who reported a new patient. This questionnaire requested information on clinical presentation, ethnicity, family history, laboratory investigations, treatment, and coexisting comorbidities (i.e., obesity, hypertension, dyslipidemia, polycystic ovary syndrome, nonalcoholic fatty liver disease, and nephropathy). Laboratory investigations were performed locally and were reported on the questionnaire. The availability of pancreatic antibody levels (i.e., GAD, islet cell, and insulin antibodies) varied across Canada, but, where possible, were reported and were included in the analysis. Duplicate reports were identified by region of residence, date of birth, sex, and date of diagnosis. This enabled duplicate cases to be removed.

Completed questionnaires were reviewed independently by three primary investigators (S.A., J.K.H., and H.J.D.) and a diagnosis of type 2 diabetes, medication-induced diabetes, monogenic diabetes, or other (i.e., indeterminate or type 1 diabetes) was assigned. In the event of disagreement, the questionnaire was forwarded to three pediatric endocrinology

coinvestigators (S.H., C.P., and E.A.C.S.) to independently assign a diagnosis. A consensus diagnosis was ascribed to the case. If no consensus was achieved, the case was labeled "indeterminate." All case patients met the criteria for diabetes as defined by the Canadian Diabetes Association (17). Criteria for the definition of each subgroup of non–type 1 diabetes were based on *1*) for type 2 diabetes, the presence of risk factors as outlined in the Canadian Diabetes Association 2003 clinical practice guidelines (17) and information on clinical presentation obtained from the detailed questionnaire (i.e., presence of obesity and/or absence of pancreatic autoimmunity on laboratory testing, and minimal or no insulin requirements); *2*) for medication-induced diabetes, a child receiving a known diabetogenic medication at the time of diagnosis (e.g., glucocorticoids, L-asparaginase, cyclosporine, tacrolimus, atypical antipsychotics, and anticonvulsants); and *3*) for monogenic diabetes, isolation of at least one of six different mutations (glucokinase, hepatic nuclear factor [HNF]-1α, HNF-4α, HNF-1β, insulin promoter factor-1, and neurogenic differentiation 1/β-cell E-box transactivator 2) or family history of diabetes affecting multiple generations in an autosomal dominant pattern and negative testing for markers of pancreatic autoimmunity.

Statistical methodology

An observed "minimum" incidence rate was calculated as the total number of new cases of non–type 1 diabetes per year per 100,000 children aged <18 years. Observed minimum incidence of the three unique categories of non–type 1 diabetes (type 2 diabetes, medication-induced diabetes, and monogenic diabetes) was calculated. This national surveillance study was designed to capture *all* new cases of physician-diagnosed type 2 diabetes, medication-induced diabetes, and monogenic diabetes in children aged <18 years living in Canada. The denominators used for Canadian incidence estimates and province-specific incidence estimates were derived from 2006 Canadian Census estimates (http://www.statcan.gc.ca), and it was assumed that the estimate remained the same over the 24-month study period. Denominators for population estimates of children belonging to specific ethnic groups (Caucasian, Aboriginal, African/Caribbean, and Asian) were obtained from the most recent Canadian Census that included these data (2001) assuming that this estimate closely approximated the ethnic distribution of Canadian children aged <18 years in 2006. Population estimates for children belonging to Hispanic, Middle Eastern, or mixed ethnicity ($n = 19$) were not available and therefore were not included. Descriptive statistics were used to illustrate demographics and clinical features of type 2, medication-induced, and monogenic diabetes.

Sensitivity analyses for non–type 1 and type 2 diabetes were conducted to account for the fact that an enriched subset of family physicians in Canada participated in this study. Adult endocrinologists reported only four cases over the 24-month period and therefore were excluded from the sensitivity analysis. The "maximum" incidence rate assumed that each nonparticipating family physician in Canada saw the same mean number of incident cases as those participating in this study. The selected enriched sample included physicians with a specific practice pattern who were located in regions known to contain a higher prevalence of children with non–type 1 diabetes; thus, the maximum incidence rate is probably an overestimate of the true incidence rate of non–type 1 and type 2 diabetes in Canadian children. The "conservative" incidence rate accounts for the enriched sample and so assumed that each nonparticipating family physician saw one-quarter of the incident cases of those participating.

Ethical considerations

Ethical approval was obtained from the University of Manitoba Health Research Ethics Board and The Hospital for Sick Children.

RESULTS — Over 24 months of surveillance, reporting rates remained consistent with overall response rates of 79% among pediatricians (including pediatric endocrinologists) and 96 and 85% among family physicians and adult endocrinologists, respectively. A total of 472 cases were reported, with an average of 14–16 cases per month over the surveillance period. Reporting physicians failed to return 40 (8%) questionnaires. Of the case reports, 21 (4%) were duplicates, and 66 (14%) case reports were excluded because they did not meet the case definition. Therefore, a total of 345 cases of non–type 1 diabetes were included for analysis; 227 cases of type 2 diabetes, 56 cases of medication-induced diabetes, and 31 cases of monogenic diabetes. The 31 remaining cases could not be classified and were labeled indeterminate. Ten cases of type 2 diabetes and 9 cases of monogenic diabetes were revised diagnoses after an initial diagnosis of type 1 diabetes. Pediatric endocrinologists reported 266 (77%) cases of non–type 1 diabetes; general pediatricians, family physicians, and adult endocrinologists reported 53 (15%), 22 (7%), and 4 (1%) cases of non–type 1 diabetes, respectively.

Incidence and demographics

The observed minimum incidence of non–type 1 diabetes in Canadian children was 2.34 cases per 100,000 children per year. Sensitivity analysis revealed a maximum incidence of 52.8 cases per 100,000 per year and a conservative incidence estimate of 15.0 cases per 100,000 children per year. Table 1 outlines the observed minimum incidence rates of type 2, medication-induced, and monogenic diabetes. Sensitivity analysis applied to type 2 diabetes alone revealed a maximum incidence of 40.5 cases per 100,000 children per year and a conservative incidence of 11.3 cases per 100,000 children per year. The observed minimum incidence of type 2 diabetes in female and male children aged <18 years was 2.0 and 1.3 cases per 100,000 per year, respectively. In children <10 and ≥10 years of age, the observed minimum incidence rates of type 2 diabetes were 0.27 and 3.1 per 100,000 per year, respectively. The observed minimum incidences of type 2 diabetes in Caucasian ($n = 5,236,199$), Aboriginal ($n = 215,831$), Asian ($n = 600,480$), and African/Caribbean ($n = 148,466$) children aged <18 years were 0.54, 23.2, 7.7, and 1.9 cases per 100,000 per year.

Clinical findings and investigations at diagnosis

Type 2 diabetes ($n = 227$). The mean ± SD age at diagnosis was 13.7 ± 2.5 years, and 58% (132 of 227) of patients were female. Twenty-five percent (57 of 227) were Caucasian, 44.1% (100 of 227) were Aboriginal, 10.1% (23 of 227) were African/Caribbean, and 10.1% (23 of 227) were Asian. The remaining patients were Hispanic (1.8% [4 of 227]), Middle Eastern (0.4% [1 of 227]), or of mixed ethnicity (6.2% [14 of 227]). Of children with newly diagnosed type 2 diabetes, 8% (19 of 227) were <10 years of age. Within ethnic groups, 11% (11 of 100) of Aboriginal, 8.8% (5 of 57) of Caucasian, 8.7% (2 of 23) of Asian, and 4.3%

Table 2—*Clinical features and comorbidity at diagnosis of type 2 diabetes*

Clinical feature	Proportion
Asymptomatic	78/225 (35)
Acanthosis nigricans	161/221 (73)
Obesity*	206/216 (95)
Ketosis	46/104 (44)
Diabetic ketoacidosis†	22/220 (10)
Comorbidity	
Polycystic ovarian syndrome	16/132 (12.1)
Dyslipidemia	78/174 (44.8)
Hypertension	58/205 (28.3)
Alanine transferase >90 IU/l or "fatty liver" on ultrasound	39/176 (22.2)
Micro-/macroalbuminuria	21/148 (14.2)

Data are *n* (%). $n = 227$. *BMI >95th percentile for age and sex. †pH <7.35.

(1 of 23) of African/Caribbean children presented before 10 years of age. A positive family history in either a first- or second-degree relative was reported in 91% (185 of 203) of children. Clinical features and comorbidity at diagnosis are shown in Table 2. The BMI at presentation was 32.1 ± 7.2 kg/m^2 with a BMI *Z* score of 2.08 ± 0.6. Of children with type 2 diabetes, 10% presented in diabetic ketoacidosis (DKA). There was no significant difference in the rate of DKA across ethnic groups ($P = 0.1$). Of children with type 2 diabetes, 37% (43 of 115) had at least one comorbidity and 13% (15 of 115) had three or more comorbidities at diagnosis. The A1C at diagnosis was 9.6 ± 3.0% (median 8.7%). Of children with type 2 diabetes who had pancreatic antibodies measured, 2.1% (2 of 97) had GAD antibodies, 0% (0 of 88) had islet cell antibodies, and 15.2% (12 of 79) had insulin antibodies. Patients were initially treated with lifestyle counseling alone (33% [69 of 211]), lifestyle counseling combined with insulin (27% [58 of 211]), lifestyle counseling combined with an oral agent (22% [46 of 211]), or lifestyle counseling, insulin, and an oral agent (16% [33 of 211]).

Medication-induced diabetes ($n = 56$). Children presented at a mean ± SD age of 13.3 ± 3.5 years; 55% (31 of 56) were Caucasian and 52% (24 of 46) were obese. Forty-one percent (22 of 54) were asymptomatic. Polyuria (51% [28 of 55]) and fatigue (39% [20 of 51]) were the most common symptoms. The average A1C at presentation was 6.6 ± 1.9% (median 5.9%). Glucocorticoid therapy was reported in 98% (55 of 56) of children; isolated glucocorticoid treatment was reported in 55% (31 of 56) and glucocorticoids in combination with tacrolimus, L-asparaginase, and cyclosporine in 21% (12 of 56), 16% (9 of 56), and 4% (2 of 56) of children, respectively. Fourteen percent (7 of 52) of children did not receive treatment for their diabetes. Lifestyle counseling alone (29% [15 of 52]), insulin therapy alone (29% [15 of 52]), and a combination of insulin and lifestyle counseling (29% [15 of 52]) were used at similar frequencies.

Monogenic diabetes ($n = 31$). Children presented at a mean ± SD age of 9.8 ± 6.5 years, and 71% (22 of 31) were Caucasian. The majority were asymptomatic (61% [19 of 31]). In those with symptoms, polyuria (29% [9 of 31]) and polydipsia (28% [8 of 29]) were most common. Acanthosis nigricans was reported in 7% (2 of 30) of children. The BMI *Z* score at diagnosis was 0.63 ± 0.12. Eleven percent (2 of 19) were overweight, and 16% (3 of 19) were obese at presentation. The mean A1C at presentation was 7.4 ± 2.4% (median 6.7%).

GAD antibodies ($n = 15$) and insulin antibodies ($n = 10$) were negative in all children; 14 patients were tested for islet cell antibodies, and 1 (7%) tested positive. Results of genetic testing were available in 16 patients; 7 had glucokinase mutations (including the child with positive islet cell antibodies), 2 had HNF-1α mutations, 1 had an insulin promoter factor-1 mutation, and 6 had confirmed neonatal diabetes (Kir6.2 mutations [$n = 3$], mutations involving chromosome 6 [$n = 2$], and syndromes associated with neonatal diabetes [$n = 1$]). Treatment was not initiated in 7% (2 of 29) of children. Of those treated, regimens included insulin alone (21% [6 of 29]), lifestyle counseling alone (55% [16 of 29]), a combination of insulin and lifestyle counseling (10% [3 of 29]), and insulin, an oral hypoglycemic agent, and lifestyle counseling (7% [2 of 29]). The majority of children (89% [24 of 27]) did not have comorbidity at diagnosis.

CONCLUSIONS — This is the second national surveillance study to report on the incidence of type 2 diabetes in children and the first to report the incidence and clinical features at presentation of type 2 diabetes and other forms of non–type 1 diabetes in Canadian children. Based on provincial database registries (13) and historical evidence (10,11), the incidence of type 2 diabetes in children in Canada seems to be increasing. Obesity seems to be the single most important risk factor for type 2 diabetes, a finding common to other studies (2,3). Interestingly, 8% of children with type 2 diabetes in our study were <10 years of age at presentation. In the U.S. SEARCH for Diabetes in Youth Study, only 3.6% of cases of type 2 diabetes occurred in children <10 years of age (3), indicating that this may be a finding unique to the Canadian population. Our results highlight the fact that pediatric type 2 diabetes is not exclusive to the adolescent age-group and can occur in younger children. Similar to other studies (18), treatment varied considerably, highlighting a need for clinical trials to identify optimal treatment strategies for pediatric type 2 diabetes.

The overall observed minimum incidence of type 2 diabetes in Canadian children is three times the rate reported in the U.K. (2) and approximately one-quarter of that of the U.S. for children >10 years of age (3). Although the observed minimum incidence of type 2 diabetes in Canadian Caucasian and Asian children is comparable to that reported by the U.K., the incidence in African/Caribbean children is twice that of the U.K. (2). Canadian and U.K. incidence estimates are easily comparable because of similar surveillance methodologies. The SEARCH study included 10 locations that were considered representative of the multiethnic distribution of the U.S. population. Differences in U.S. and Canadian estimates may relate to variations in ethnic distribution and screening practices or a sampling bias toward sites with higher proportions of ethnic groups at higher risk for type 2 diabetes in the SEARCH study. To our knowledge, ours is the first population-based study to report the national incidence of medication-induced and monogenic diabetes.

Canadian Aboriginal children <18 years of age have the highest incidence of type 2 diabetes and the majority of these children are from Manitoba, explaining the 20-fold higher incidence rate of type 2 diabetes in this province. This finding is comparable to the U.S., which reports an incidence of type 2 diabetes in American Navajo youth aged 10–14 years of 22.4 cases per 100,000 person-years and 39.34 cases per 100,000 person-years in those aged 15–19 years (19). Interestingly, type 2 diabetes in American Indian children <10 years of age is rare (19);

however, in Canadian Aboriginal children, 11 cases (11%) of type 2 diabetes occurred in children <10 years of age. This finding suggests that clinical practice guidelines on childhood type 2 diabetes may require revision for selected populations (20,21). Finally, although Aboriginal children are at the highest risk for type 2 diabetes, 50% of clinically diagnosed type 2 diabetes occurred in non-Aboriginal children.

The presence of hyperglycemia, ketosis, and pancreatic autoimmunity typically suggests a diagnosis of type 1 diabetes. In this study, 44% of children with type 2 diabetes presented with ketonuria, 10% presented in DKA, and a small percentage exhibited the presence of GAD and insulin antibodies. These findings are similar to those reported in the literature (2,22). The SEARCH study reported that 21.2% of children with clinically diagnosed type 2 diabetes were positive for GAD antibodies (3). There is debate as to whether these youth have been misclassified as having type 2 diabetes; however, clinically they present with "typical" features of type 2 diabetes including obesity and acanthosis nigricans. Furthermore, they respond quickly to insulin treatment and can wean off insulin for extended periods of time (23). Therefore, the presence of ketonuria and/or pancreatic autoimmunity does not preclude type 2 diabetes in the pediatric age-group. Additional studies are necessary to better understand the relationship of pancreatic autoimmunity to the etiology and natural history of diabetes in children.

This study was limited by factors common to other population-based surveillance studies. Our study generated a minimum incidence rate of pediatric non–type 1 diabetes for the following reasons: *1*) children with diabetes seen by nonparticipating physicians and nonresponders were not captured; 2) classification was not possible when case reports were incomplete; and 3) reporting physicians may not have recognized all children with cases of non–type 1 diabetes. The incidence of type 2 diabetes in Saskatchewan seems to be low. This may reflect the unique Aboriginal groups and other ethnic groups that live in that region of the country. The possibility of low reporting rates by pediatricians and family doctors in that province remains. The population estimate for Saskatchewan (233,900) represents only 3% of the total Canadian population of children <18 years of age, and, therefore, this underestimation probably had minimal impact on Canadian incidence rates. A previous surveillance study using the CPSP methodology reported cases from 7 of 13 provinces and territories, which represented 92% of the Canadian population (24). Second, our study depended on physician-based classification of diabetes followed by review and classification by clinician investigators. This methodology was similar to that used in the U.K. where, 1 year after their initial study, only one case of type 2 diabetes was reclassified (18). In the SEARCH study, differentiation of type 1 and type 2 diabetes was based on the diagnosis made by reporting physicians without review of clinical data by study investigators. Third, obesity-related morbidities such as hypertension and dyslipidemia were considered to be present if the reporting physician indicated as such; clinical or laboratory evidence was not requested. Last, testing for monogenic diabetes is not widely available in Canada. Patients with a typical family history and natural history of disease were classified as having monogenic diabetes even without confirmed genetic testing. Therefore, the calculated incidence of monogenic diabetes may be either an over- or underestimate of the true incidence.

Assessment of the completeness of ascertainment (i.e., capture-recapture method) using independent sources of information (i.e., prescription data and hospitalization) was not possible because many children with type 2 diabetes are not receiving medication and hospitalization is rare. It is likely that most new cases of non–type 1 diabetes in children were detected, as almost all Canadian children with uncommon conditions are referred to pediatric practitioners. In this study, 92% of cases were reported by pediatricians or pediatric endocrinologists, reflecting the model of care for pediatric chronic disease in Canada. In addition, a type 2 diabetes registry in Manitoba reports 35–45 new pediatric cases per year (25), a number that is consistent with our study results: a total of 69 new cases of type 2 diabetes were reported in Manitoba over 2 years. In the present study, >75% of children with type 2 diabetes were reported by a pediatric endocrinologist. Every region in Canada is served by a team specializing in the care, education, and support of children with diabetes. A particular strength of this study is that surveillance occurred over a 24-month period and reporting rates remained consistent over this time period.

A sensitivity analysis was conducted to account for the small enriched sample of family physicians who participated in this study. Pediatricians were excluded from the sensitivity analysis because participation rates were high, the sample was not enriched, and previous CPSP surveillance studies with similar participation rates did not require a sensitivity analysis (24). The maximum and conservative incidence rates were calculated to provide confidence intervals between which the true incidence of non–type 1 diabetes lies. Last, our response rates of 79–95% were acceptable for this type of surveillance study; however, cases could have been missed because of lack of reporting.

This prospective national surveillance study for non–type 1 diabetes provides information on the existing spectrum of non–type 1 diabetes in Canadian children. Until now, the majority of epidemiological data on pediatric type 2 diabetes originated from Manitoba where virtually all cases occur in Aboriginal youth. The results of this study provide a more accurate representation of type 2 diabetes in Canadian children and provide baseline incidence data based on Canada's unique ethnic, cultural, and geographic characteristics. As rates of type 2 diabetes increase, surveillance information is critical to inform health policy makers, track success of prevention and treatment strategies, and increase awareness among health care providers.

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