Type 2 Diabetes, Medication-Induced Diabetes, and Monogenic Diabetes in Canadian Children: A Prospective National Surveillance Study

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**Objectives:** To determine in Canadian children <18 years the: 1) incidence of type 2 diabetes, medication-induced diabetes and monogenic diabetes, 2) clinical features of type 2 diabetes and 3) co-existing 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:** 345 cases of non-type 1 diabetes were reported from a population of 7.3 million children. The observed minimum incidence rates of type 2, medication-induced, and monogenic diabetes were 1.54, 0.4, and 0.2 cases/100,000 children <18 years of age/year, respectively. On average, children with type 2 diabetes were 13.7 years and 8% (19/227) presented before 10 years. Ethnic minorities were over-represented, but 25% (57/227) of children with type 2 diabetes were Caucasian. 95% (206/216) of children with type 2 diabetes were obese and 37% (43/115) had at least one co-morbidity 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 co-morbidity should occur at diagnosis of type 2 diabetes.
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. 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 United Kingdom (UK) estimated the incidence of type 2 diabetes to be 0.53/100,000/year in children <17 years of age (2). A multi-center population-based study from the United States reported an incidence of 8.1/100,000 person-years and 11.8/100,000 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 reported 7% of children affected by medication-induced diabetes after renal transplant and 50% of these children were obese (7). Monogenic forms of diabetes account for approximately 1-5% of all cases of diabetes (8) with a minimum prevalence of 0.17/100,000 reported in children in the UK (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 persons <18 years of age 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 <18 years to determine the: 1) incidence of non-type 1 diabetes, 2) clinical features of type 2 diabetes at diagnosis, and 3) co-morbidity 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 Paediatric Surveillance Program (CPSP) and the College of Family Physicians of Canada – National Research System (CFPC-NaReS), both nationally recognized surveillance programs. The CPSP is comprised of greater than 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 greater than 90% in previous surveillance studies (14). NaReS, a network of the College of Family Physicians of Canada, is comprised of approximately 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=2560). Although rare pediatric conditions are seen only by pediatric practitioners, and most children diagnosed with 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 College of Family Physicians as practicing pediatric, adolescent, Aboriginal, and rural or inner city medicine in northern Canada or core urban areas was generated from a database housed NaReS (N=2823). This database includes clinical practice information and demographics on approximately 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. A letter was sent to these practitioners requesting participation and asking whether they had previously encountered a case of pediatric non-type 1 diabetes. Feasibility allowed the involvement of 100 family physicians and therefore, those who agreed to participate and had previously seen a case of pediatric non-type 1 diabetes 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 2560 pediatricians participated with geographic representation from across Canada (Table 1).

**Surveillance Methodology:** Physicians were surveyed for 24 months between April 1st, 2006 and March 30th, 2008. All physicians received an introductory package that included a case definition (16). Physicians were asked to report new patient cases where 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 case. A detailed questionnaire was subsequently sent to each physician who reported a new case. This questionnaire requested information on clinical presentation, ethnicity, family history, laboratory investigations, treatment, and co-existing co-morbidities (i.e. obesity, hypertension, dyslipidemia, polycystic ovarian syndrome, non-alcoholic fatty liver disease, and nephropathy). Laboratory investigations were performed locally and were reported on the questionnaire. The availability of pancreatic antibody levels (i.e. glutamic acid decarboxylase, islet cell, and insulin antibodies) varied across Canada, but where possible, were reported and included in the analysis. Duplicate reports were identified by region of residence, date of birth, gender and date of diagnosis. This enabled duplicate cases to be removed. Completed questionnaires were reviewed independently by three primary investigators (S.A., J.H., H.D.) and a diagnosis of type 2 diabetes, medication-induced diabetes, monogenic diabetes, or other (i.e. indeterminate, type 1 diabetes) was assigned. In the event of disagreement, the questionnaire was forwarded to three pediatric endocrinology co-investigators (S.H., C.P., E.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 cases met criteria for diabetes as defined by the Canadian Diabetes Association (CDA) (17). Criteria for the definition of each subgroup of non-type 1 diabetes was based on: 1) for type 2 diabetes; presence of risk factors as outlined in the CDA 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
Canadian Surveillance of type 2 diabetes; a child receiving a known diabetogenic medication at the time of diagnosis (e.g. glucocorticoids, L-asparaginase, cyclosporine, tacrolimus, atypical antipsychotic, anticonvulsant); 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 (IPF)-1, 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/year/100,000 children <18 years of age. Observed minimum incidence of the 3 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 <18 years of age living in Canada. The denominators used for Canadian incidence estimates and province specific incidence estimates were derived from 2006 Canadian Census estimates (www.statcan.ca) and it was assumed 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, Asian) were obtained from the most recent Canadian Census that included this data (2001) assuming that this estimate closely approximated the ethnic distribution of Canadian children <18 years of age 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 4 cases over the 24-month period therefore, were excluded from the sensitivity analysis. The “maximum” incidence rate assumed that each non-participating 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; as such the maximum incidence rate is likely 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 non-participating 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/month over the surveillance period. Reporting physicians failed to return 40 (8%) questionnaires. Twenty-one (4%) case reports 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
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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/100,000 children/year. Sensitivity analysis revealed a maximum incidence of 52.8 cases/100,000/year and a conservative incidence estimate of 15.0 cases/100,000 children/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/100,000 children/year and a conservative incidence of 11.3 cases/100,000 children/year. The observed minimum incidence of type 2 diabetes in females and males <18 years of age was 2.0 cases/100,000/year and 1.3 cases/100,000/year respectively. In children <10 and ≥10 years of age, the observed minimum incidence rates of type 2 diabetes were 0.27/100,000/year and 3.1/100,000/year respectively. In children <18 years of age was 2.0 cases/100,000/year and 1.3 cases/100,000/year respectively. In children <10 and ≥10 years of age, the observed minimum incidence rates of type 2 diabetes were 0.27/100,000/year and 3.1/100,000/year respectively. The observed minimum incidence 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 <18 years of age were 0.54, 23.2, 7.7, and 1.9 cases/100,000/year.

**Clinical Findings and Investigations at Diagnosis:** Type 2 diabetes (n=227) The mean age at diagnosis was 13.7 ± 2.5 years and 58% (132/227) of cases were female. Twenty-five percent (57/227) were Caucasian, 44.1% (100/227) Aboriginal, 10.1% (23/227) African/Caribbean, and 10.1% (23/227) Asian. The remaining cases were Hispanic (1.8% [4/227]), Middle Eastern (0.4% [1/227]) or of mixed ethnicity (6.2% [14/227]). Eight percent (19/227) of children with newly diagnosed type 2 diabetes were less than 10 years of age. Within ethnic groups, 11% (11/100) of Aboriginal, 8.8% (5/57) of Caucasian, 8.7% (2/23) of Asian, and 4.3% (1/23) 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% (185/203) of cases. Clinical features and co-morbidity at diagnosis are shown in Table 2. The mean body mass index (BMI) at presentation was 32.1 ± 7.2 kg/m^2 with a mean BMI z-score of 2.08 ± 0.6. Ten percent of children with type 2 diabetes presented in diabetic ketoacidosis (DKA). There was no significant difference in the rate of DKA across ethnic groups (p=0.1). Thirty-seven percent (43/115) of cases had at least one co-morbidity and 13% (15/115) had three or more co-morbidities at diagnosis. The mean A1c at diagnosis was 9.6 ± 3.0% (median 8.7%). Of children with type 2 diabetes who had pancreatic antibodies measured, 2.1% (2/97) had glutamic acid decarboxylase (GAD) antibodies, 0% (0/88) had islet cell antibodies, and 15.2% (12/79) had insulin antibodies.

Patients were initially treated with lifestyle counseling alone (33% [69/211]), lifestyle counseling combined with insulin (27% [58/211]), lifestyle counseling combined with an oral agent (22% [46/211]), or lifestyle counseling, insulin and an oral agent (16% [33/211]).

**Medication-induced diabetes (n=56):** Children presented at a mean age of 13.3 ± 3.5 years, 55% (31/56) were Caucasian and 52% (24/46) were obese. Forty-one percent (22/54) were asymptomatic. Polyuria (51%
and fatigue (39% [20/51]) were the most common symptoms. The average A1c at presentation was 6.6 ± 1.9% (median 5.9%). Glucocorticoid therapy was reported in 98% (55/56) of cases; isolated glucocorticoid treatment in 55% (31/56) and glucocorticoids in combination with tacrolimus, L-asparaginase, and cyclosporine in 21% (12/56), 16% (9/56) and 4% (2/56) of cases respectively. Fourteen percent (7/52) of cases did not receive treatment for their diabetes. Lifestyle counseling alone (29% [15/52]), insulin therapy alone (29% [15/52]), and a combination of insulin and lifestyle counseling (29% [15/52]) were used at similar frequencies.

**Monogenic Diabetes (n=31):** Children presented at a mean age of 9.8 ± 6.5 years and 71% (22/31) were Caucasian. The majority were asymptomatic (61% [19/31]). In those with symptoms, polyuria (29% [9/31]) and polydypsia (28% [8/29]) were most common. Acanthosis nigricans was reported in 7% (2/30) of cases. The mean BMI z-score at diagnosis was 0.63 ± 0.12. Eleven percent (2/19) were overweight and 16% (3/19) 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 cases; 14 patients were tested for islet cell antibodies and 1 (7%) tested positive. Results of genetic testing was available in 16 patients; 7 had glucokinase mutations (including the case with positive islet cell antibodies), 2 HNF 1-α mutations, 1 with an IPF-1 mutation, and 6 cases with 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% (2/29) of cases. Of those treated, regimens included insulin alone (21% [6/29]), lifestyle counseling alone (55% [16/29]), a combination of insulin and lifestyle counseling (10% [3/29]) and insulin, an oral hypoglycemic agent and lifestyle counseling (7% [2/29]). The majority of cases (89% [24/27]) did not have co-morbidity 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 appears to be increasing. Obesity appears 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 US SEARCH for Diabetes in Youth Study, only 3.6% of cases of type 2 diabetes occurred in children <10 years of age (3) indicating this may be a finding unique to the Canadian population. Our results highlight 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 UK (2) and approximately a quarter of that of the US for children older than 10 years of age (3). While the observed minimum incidence of type 2 diabetes in Canadian Caucasian and Asian children is comparable to that reported by the UK, the incidence in African/Caribbean children is twice that of the UK (2). Canadian and UK incidence estimates are easily comparable because of similar surveillance methodologies. The SEARCH study included 10 locations that were considered
representative of the multi-ethnic distribution of the US population. Differences in US and Canadian estimates may relate to variation in ethnic distribution and screening practices or a sampling bias towards 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 is comparable to the US which reports an incidence of type 2 diabetes in American Navajo youth aged 10-14 years of 22.4 cases/100,000 person-years and 39.34 cases/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 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 suggest a diagnosis of type 1 diabetes. In this study, 44% of children with type 2 diabetes presented with ketonuria, 10% 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 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: (i) cases seen by non-participating physicians and non-responders were not captured, (ii) classification was not possible when case reports were incomplete, and (iii) reporting physicians may not have recognized all cases of non-type 1 diabetes. The incidence of type 2 diabetes in SK appears 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 SK (233,900) represents only 3% of the total Canadian population of children <18 years of age and therefore, this underestimation likely had minimal impact on Canadian incidence rates. A previous surveillance study using the CPSP methodology reported cases from 7/13 provinces and Territories, which represented 92% of the Canadian population (24). Secondly, 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 UK where, one year after their initial study, only 1 case of type 2 diabetes was re-classified (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. Thirdly, obesity-related morbidity such as hypertension and dyslipidemia were considered to be present if the reporting physician indicated as such; clinical or laboratory evidence was not requested. Lastly, testing for monogenic diabetes is not widely available in Canada. Cases 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, hospitalization) was not possible because many children with type 2 diabetes are not on medication and hospitalization is rare. It is likely that most new cases of non-type 1 diabetes were detected as almost all Canadian children with type 2 diabetes are not on medication and hospitalization is rare. It is likely that most new cases of non-type 1 diabetes 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. Also, a type 2 diabetes registry in Manitoba reports 35-45 new pediatric cases/year (25), a number that is consistent with our study results where a total of 69 new cases of type 2 diabetes were reported in Manitoba over 2 years. In the present study over 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. Lastly, our response rates of 79-95% were acceptable for this type of surveillance study however cases could have been missed due to 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 amongst health care providers.

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This article is based on information gathered through the Canadian Paediatric Surveillance Program. The views, opinions and/or conclusions expressed by the author(s) are their own and do not necessarily reflect the views, opinions and/or conclusions of either the Canadian Paediatric Society, the Public Health Agency of Canada or the Canadian Paediatric Surveillance Program.
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8. Ledermann HM. Maturity-onset diabetes of the young (MODY) at least ten times more common in Europe than previously assumed? Diabetologia 1995;38(12):1482.
### Table 1: Minimum incidence rates of type 2 diabetes, medication-induced diabetes and monogenic diabetes in Canadian children <18 years of age

<table>
<thead>
<tr>
<th>Regions</th>
<th>Population Estimate</th>
<th>T2DM</th>
<th>MID</th>
<th>Monogenic DM</th>
<th>TOTAL (T) AND PARTICIPATING (P) FAMILY PHYSICIANS (FP), PEDIATRICIANS (PEDS) AND ADULT ENDOCRINOLOGISTS (AE)</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>FP** T</td>
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<td>CANADA</td>
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<td>1.54</td>
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<td>31,235</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>94</td>
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*2006 Canadian Census – Statistics Canada
†T2DM – type 2 diabetes
‡MID – medication induced diabetes
†Nova Scotia, New Brunswick, Prince Edward Island, Newfoundland & Labrador
¶Northwest Territories, Yukon, Nunavut

*Source – *Geographic Distribution of Physicians in Canada: Beyond How Many and Where.* Canadian Institute for Health Information (CIHI). 2005
*Source – Canadian Medical Directory
Table 2: Clinical features and co-morbidity at diagnosis of type 2 diabetes (n=227)

<table>
<thead>
<tr>
<th>Clinical Feature</th>
<th>Proportion - %</th>
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<tbody>
<tr>
<td>Asymptomatic</td>
<td>35</td>
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<tr>
<td>Acanthosis Nigricans</td>
<td>73</td>
<td>(161/221)</td>
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<tr>
<td>Obesity*</td>
<td>95</td>
<td>(206/216)</td>
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<tr>
<td>Ketosis</td>
<td>44</td>
<td>(46/104)</td>
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<tr>
<td>Diabetic ketoacidosis†</td>
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<td>(22/220)</td>
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<table>
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<th>Co-morbidity</th>
<th>Proportion - %</th>
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<td>Polycystic ovarian syndrome</td>
<td>12.1</td>
<td>(16/132)</td>
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<tr>
<td>Dyslipidemia</td>
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<td>(78/174)</td>
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<tr>
<td>Hypertension</td>
<td>28.3</td>
<td>(58/205)</td>
</tr>
<tr>
<td>Alanine transferase &gt;90 IU/L or</td>
<td>22.2</td>
<td>(39/176)</td>
</tr>
<tr>
<td>“fatty liver” on ultrasound</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Micro/macroalbuminuria</td>
<td>14.2</td>
<td>(21/148)</td>
</tr>
</tbody>
</table>

*Body mass index >95th %tile for age and gender
†pH<7.35