BMJ Open Diabetes Research & Care

Socioeconomic status and risk factors for complications in young people with type 1 or type 2 diabetes: a cross-sectional study

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To cite: Wijayaratna S, Lee A, Park HY, *et al*. Socioeconomic status and risk factors for complications in young people with type 1 or type 2 diabetes: a cross-sectional study. *BMJ Open Diab Res Care* 2021;**9**:e002485. doi:10.1136/bmjdrc-2021-002485

► Additional supplemental material is published online only. To view, please visit the journal online (http://dx.doi.org/10.1136/bmjdrc-2021-002485).

Received 16 July 2021
Accepted 26 November 2021

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ABSTRACT

Introduction Young people with type 2 diabetes (T2D) develop complications earlier than those with type 1 diabetes (T1D) of comparable duration, but it is unclear why. This apparent difference in phenotype could relate to relative inequality.

Research design and methods Cross-sectional study of young people referred to secondary diabetes services in Auckland, Aotearoa-New Zealand (NZ): 731 with T1D and 1350 with T2D currently aged ≤40 years, and diagnosed between 15 and 30 years. Outcome measures were risk factors for complications (glycemic control, urine albumin/creatinine ratio (ACR), cardiovascular disease (CVD) risk) in relation to a validated national index of deprivation (New Zealand Deprivation Index (NZDep)).

Results Young people with T2D were an average 3 years older than those with T1D but had a similar duration of diabetes. 71% of those with T2D were of Māori or Pasifika descent, compared with 24% with T1D (p<0.001). T1D cases were distributed evenly across NZDep categories. 78% of T2D cases were living in the lowest four NZDep categories (p<0.001). In both diabetes types, body mass index (BMI) increased progressively across the NZDep spectrum (p<0.002), as did mean glycated hemoglobin (HbA_{1c}) (p<0.001), the prevalence of macroalbuminuria (p≤0.01), and CVD risk (p<0.001). Adjusting for BMI, diabetes type, and duration and age, multiple logistic regression revealed deprivation was the strongest risk factor for poorly controlled diabetes (defined as HbA_{1c} >64 mmol/mol, >8%); OR 1.17, 95% CI 1.13 to 1.22, p<0.0001. Ordinal logistic regression showed each decile increase in NZDep increased the odds of a higher ACR by 11% (OR 1.11, 95% CI 1.06 to 1.16, p<0.001) following adjustment for BMI, blood pressure, diabetes type and duration, HbA_{1c}, and smoking status. Multiple linear regression indicated a 4% increase in CVD risk for every decile increase in NZDep, regardless of diabetes type.

Conclusions The apparent more aggressive phenotype of young-onset T2D is at least in part explicable by relative deprivation.

Significance of this study

What is already known about this subject?

- The number of young people diagnosed with type 2 diabetes is rapidly increasing.
- The prevalence of type 2 diabetes in younger people is associated with poorer socioeconomic status.
- Young-onset type 2 diabetes is recognised as having a more aggressive phenotype than type 1 diabetes, with higher complication rates, but the reasons why are not understood.

What are the new findings?

- Risk factors for diabetes complications (glycemic control, albuminuria and cardiovascular risk) were all associated with relative deprivation, irrespective of diabetes type.
- Socioeconomic deprivation is a stronger risk factor for poorly controlled diabetes than age, duration of diabetes, type of diabetes, or body mass index.
- The apparently more severe phenotype of young-onset type 2 diabetes is at least in part explicable by relative deprivation.

How might these results change the focus of research or clinical practice?

- Addressing socioeconomic disparities is essential in improving young people with type 2 diabetes.

INTRODUCTION

In recent decades, there have been significant increases in the prevalence of type 2 diabetes in many countries. As the prevalence has increased, there has also been a reduction in the average age at diagnosis, so that type 2 diabetes has become increasingly common in people in their teens, 20s, and 30s.[1–3] Type 2 diabetes in young people is of great concern; in what should be their most productive years those affected are at risk for the classical microvascular complications of diabetes (retinopathy, nephropathy, and neuropathy) and for cardiovascular disease (CVD) associated with obesity-related metabolic syndrome. In high-income countries, type 2 diabetes in young people disproportionately affects marginalized indigenous and minority communities, a phenomenon related to high

rates of obesity, that in turn is linked to socioeconomic inequality.[4–7] In the last 40 years, Aotearoa-New Zealand has experienced markedly widening inequality, particularly affecting the indigenous Māori population and people of South Pacific Island descent (Pasifika).[8]

Recent publications from the USA and Australia have emphasized the high cardiovascular risk profile of young people with type 2 diabetes compared with people with type 1 diabetes of comparable age and disease duration.[9 10] Long-term studies have confirmed increased cardiovascular morbidity and mortality.[6 10] Microvascular complications too may be more prevalent in young people with type 2 diabetes than in comparable populations with type 1 diabetes.[10] In particular, renal disease (manifested by increased albuminuria) is disproportionately prevalent in young people with type 2 diabetes.[2 6 10–12] Thus, type 2 diabetes in young people has been described as having a more severe, aggressive or lethal phenotype than type 1 diabetes.[6 10 13]

However, it is not clear whether the aggressive phenotype of young-onset type 2 diabetes really has a biological basis[2 13] or whether social determinants play a critical role. For example, there are data suggesting that socioeconomic status is associated with adherence to medication and thus to glycemic control.[14 15] We hypothesized that relative deprivation would be related not only to the prevalence of type 2 diabetes in young people, but also to risk factors for diabetes complications. If this hypothesis were true, then we should expect to see similar relationships between the deprivation measure and these same risk factors in people of comparable age with type 1 diabetes. We therefore explored the relationships between a national index of deprivation (the New Zealand Deprivation Index (NZDep)) and established risk factors for complications.

METHODS

People with type 1 or type 2 diabetes diagnosed between the ages of 15 and 30 years who had been referred to secondary diabetes services in the greater Auckland region between 2003 and 2015 were identified by searching hospital databases. Medical records were reviewed for people referred between 15 and 35 years of age, who were ≤40 years of age at the time of the study. The year of diagnosis was established based on clinical records and glycated hemoglobin (HbA_{1c}) levels from laboratory databases. We determined diabetes type from clinic letters.

Albuminuria was assessed on random urine specimens as the urine albumin/creatinine ratio ((ACR) g/mol). For people with type 2 diabetes, the highest ACR level from the year of diagnosis was noted. Otherwise, the laboratory data reported here was collected from 2015 to 2016: the highest ACR prior to treatment and the latest available non-fasting total cholesterol, HDL, and LDL levels were recorded. An individual's average HbA_{1c} from all values available was calculated. From the clinical records the most recent systolic blood pressure (BP) was recorded and from measures of height and weight, body mass index ((BMI) kg/m^2) was calculated. Ethnicity, dialysis dependence, and deaths were based on the National Minimum Dataset (coded data from public hospitals), as of the end of 2016. Socioeconomic deprivation was determined from the NZDep 2013 schedule based on residency status and geographic living area, obtained from Primary Health Organisations' enrolment demographic quarterly tables. Decile 1 represents the least deprived and decile 10 the most deprived area.

Nationwide dispensing of all antihypertensive medication over a 6-month period in 2016 was obtained from the Pharmaceutical Collection (a Ministry of Health database containing claim and payment information from pharmacists for subsidized dispensing) in order to complete the cardiovascular risk assessment calculation, as outlined below. Similarly, nationwide dispensing of all insulin types, statins, and available oral hypoglycemic agents were also obtained. Ophthalmology procedures (intravitreal injection, vitrectomy, photocoagulation), cataracts, lower-limb amputation, cerebrovascular disease, peripheral vascular disease, heart failure, and ischemic heart disease, as of the end of 2016, were identified using International Classification of Disease (ICD-10) coding and procedural codes (online supplemental appendix 1). Valvular heart disease, congenital conditions, cardiomyopathies and other conditions where diabetes was unlikely to be a major contributory factor were excluded.

Statistical analysis

Data analysis was conducted using GraphPad Prism (V.6.00.283) and SAS/STAT software, V.9.4 of the SAS System for Windows (SAS Institute, Cary, North Carolina, USA). Statistical tests were two-tailed and a significance level of 5% was maintained. To evaluate the relationships of NZDep with clinical and laboratory variables, it was analyzed in five groups: deciles: 1–2; 3–4; 5–6; 7–8 and 9–10.

χ^2 tests were used for comparison of categorical variables between groups. T-tests were used to compare means between two groups while analysis of variance was used for comparison of more than two groups. Non-parametric tests (Wilcoxon/Mann-Whitney and Kruskal-Wallis tests) were used for non-normally distributed continuous variables. Multiple logistic regression was used to analyze the binary variable of HbA_{1c} >64 mmol/mol (>8%), with NZDep, BMI, duration of diabetes, and age included as continuous explanatory variables and type of diabetes as a categorical explanatory variable (using type 1 diabetes as the reference level). Ordinal logistic regression was used to analyze the ordinal ACR variable (ACR <3.5 g/mol (normoalbuminuria), 3.5 ≤ACR <30 g/mol (microalbuminuria), ≥30 g/mol; (macroalbuminuria), with NZDep, BMI, duration of diabetes, age at diagnosis, systolic BP, HbA_{1c}, included as explanatory variables and type of diabetes (ref=type 1) and smoking status (reference=no smoking) as categorical explanatory variables. ORs and 95% CIs from logistic regression and ordinal logistic regression were presented. A CVD risk calculator developed from the New Zealand Diabetes Cohort Study was used to determine 5-year CVD risk.[16] Equation B, which derives a CVD risk score from the following variables was used: age at

Table 1 Characteristics, risk factors, and complications of people with type 1 and type 2 diabetes diagnosed between 15 and 30 years of age

	Type 1 diabetes	Type 2 diabetes	P value
No. of people	731 (35%)	1350 (65%)	–
No. of women	323 (44%)	778 (58%)	<0.001
Mean current age (years) (SD)	30 (6)	33 (6)	<0.001
Mean age at diagnosis (years) (SD)	21 (5)	24 (4)	<0.001
Median (range) duration of diabetes (years)	8 (1–26)	8 (1–28)	0.178
Mean body mass index (kg/m^2) (SD)	25.8 (5.1)	36.7 (9.1)	<0.001
No. (%) Māori or Pasifika	178 (24%)	942 (71%)	<0.001
Mean HbA_{1c} (mmol/mol) (SD); (HbA_{1c} %)	74 (22);(8.9%)	80 (24);(9.5%)	<0.001
HbA_{1c} ≥100 (mmol/mol) (≥11.3%)	79 (13%)	236 (22%)	<0.001
Mean systolic BP (mm Hg) (SD)	116 (13)	124 (16)	<0.001
Antihypertensive medication	93 (13%)	491 (36%)	<0.001
Mean total cholesterol (mmol/L) (SD)	4.8 (1.1)	5.0 (1.5)	0.007
HDL cholesterol (mmol/L) (SD)	1.5 (0.4)	1.1 (0.3)	<0.001
LDL cholesterol (mmol/L) (SD)	2.5 (0.8)	2.7 (0.9)	<0.001
Statin dispensing	72 (10%)	387 (29%)	<0.001
No. (%) that have ever smoked	222 (30%)	536 (40%)	<0.001
Median (range) 5-year CVD risk score (%)	4.9 (1.8–25.6)	8.1 (2.0–31.3)	<0.001
No. with ophthalmology procedures/cataracts	35 (5%)	66 (5%)	0.919
Albuminuria (n (%) of subjects)			
Urine albumin/creatinine ratio ≥3.5 g/mol	115 (22%)	580 (61%)	<0.001
Urine albumin/creatinine ratio ≥30 g/mol	27 (5%)	244 (26%)	<0.001
No. (%) with composite outcome	25 (3%)	107 (8%)	<0.001

Ethnicity unknown for 3 people with T1D and 25 people with T2D people. Data available in 2015–2016 period: BMI 668 (91%) T1D, 1211 (90%) T2D; BP 668 (91%) T1D, 1199 (89%) T2D; total cholesterol 705 (96%) T1D, 1325 (98%) T2D; LDL 496 (68%) T1D, 881 (65%) T2D (normal total cholesterol <5, HDL >1, LDL <3.4 mmol/L); smoking status 647 (89%) T1D, 1205 (89%) T2D (those with status unavailable attributed as non-smokers); HbA_{1c} 597 (82%) T1D, 1076 (80%) T2D; urine albumin/creatinine 530 (73%) T1D, 955 (71%) T2D. A full set of data for CVD risk calculation was available for 495 (68%) T1D, 840 (62%) T2D. Ophthalmology procedures: intravitreal injection, vitrectomy, photocoagulation. Composite outcome: dialysis/peripheral vascular disease/lower limb amputations/cerebrovascular disease/ischemic heart disease/heart failure/death.

BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; HbA_{1c}, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; T1D, type 1 diabetes; T2D, type 2 diabetes.

diagnosis, sex, HbA_{1c}, BP, smoking status, ethnicity, cholesterol ratio, albuminuria, duration of diabetes, and use of antihypertensive medication.[16] In the absence of a validated equation using available data for the type 1 diabetes population, the same risk calculator was used for both cohorts. Those with established CVD/heart failure were excluded from this calculation. Spearman's correlation coefficient was used to determine the association between CVD risk and NZDep, and the Wilcoxon two-sample test was used to compare CVD risk between type 1 and type 2 diabetes cohorts.

RESULTS

Differences between type 1 and type 2 diabetes

We identified 1350 people with young-onset type 2 diabetes and 731 people with type 1 diabetes (table 1). Māori (33%) and Pasifika (38%) were over-represented in the type 2 diabetes group compared with the general Auckland population (p<0.001); Māori represent 11% and Pasifika people represent 15% of Auckland's population.[17] The remainder of this group consisted of 11% NZ European, 10% South Asian, 7% East Asian and 1% other ethnicities. Young people with type 2 diabetes were on average 3 years older than those with type 1, but had been diagnosed later, so the median difference in (known) diabetes duration between the groups was the same. Glycemic control, defined by mean HbA_{1c}, was poorer in those with type 2 diabetes (table 1) (p<0.001). As expected, young people with type 2 diabetes had significantly higher BMI (p<0.001). They also had higher BP (p<0.001) and were more likely to have been smokers and to have higher cholesterol values and greater albuminuria (p<0.01). The median 5-year CVD risk score was higher (8.1% type 2 vs 4.9% type 1 diabetes, p<0.001).

Deprivation and prevalence of diabetes

The distribution of cases across NZDep categories differed markedly (p<0.001; table 2). Type 1 diabetes cases were

Table 2 Variation of risk factors and complications according to socioeconomic deprivation categories, in young people with T1D and T2D

		NZDep categories					
	Type	1–2	3–4	5–6	7–8	9–10	P value
Number of people (%)	T1D	127 (17.5%)	128 (17.5%)	154 (21%)	117 (16%)	201 (28%)	<0.001†
	T2D	60 (4.5%)	109 (8%)	126 (9.5%)	262 (19.5%)	789 (58.5%)	
Mean BMI kg/m^2 (SD)	T1D	24.4 (3.6)	25.2 (4.7)	25.8 (4.7)	26.9 (5.6)	26.4 (6.0)	0.0014*
	T2D	33.3 (7.9)	36.0 (8.3)	35.2 (8.0)	35.7 (8.2)	37.7 (9.6)	<0.001*
Mean HbA_{1c} mmol/mol (SD) (HbA_{1c} %)	T1D	66 (16)(8.2%)	70 (20)(8.6%)	71 (20)(8.6%)	74 (23)(8.9%)	82 (24)(9.7%)	<0.001*
	T2D	70 (23)(8.6%)	70 (24)(8.6%)	75 (26)(9.0%)	80 (22)(9.5%)	82 (23)(9.7%)	<0.001*
Per cent (n) people that ever smoked	T1D	26 (33/127)	25 (32/128)	29 (44/154)	31 (36/117)	38 (77/201)	0.059†
	T2D	40 (24/60)	26 (28/109)	37 (46/126)	40 (105/262)	42 (331/789)	0.025†
ACR ≥3.5 g/mol (%)	T1D	15 (14/96)	17 (16/92)	18 (21/115)	25 (18/73)	30 (46/154)	0.001†
	T2D	34 (13/38)	50 (35/70)	47 (42/89)	60 (113/188)	66 (377/570)	<0.001†
ACR ≥30 g/mol (%)	T1D	3 (3/96)	1 (1/92)	8 (9/115)	0 (0/73)	9 (14/154)	0.010†
	T2D	11 (4/38)	13 (9/69)	19 (17/89)	19 (35/188)	30 (170/570)	0.002†
CVD risk estimate (%)	T1D	4.3 (3.1, 5.8)	4.6 (3.4, 6.7)	4.9 (3.5, 7.1)	4.9 (3.7, 6.8)	5.5 (4.0, 7.8)	0.0002‡
	T2D	7.0 (5.1, 9.4)	6.6 (4.9, 9.)	7.1 (5.2, 10.1)	7.7 (5.3, 10.8)	8.9 (5.7, 12.6)	<0.001‡
Proportion (%) with (n) composite outcome	T1D	1 (2/127)	3 (4/128)	0.6 (1/154)	3 (3/117)	7 (15/201)	<0.001†
	T2D	5 (3/60)	11 (12/109)	5 (6/126)	6 (16/262)	9 (69/789)	0.215†

NZDep unavailable for four people in each group. Deciles 9–10: most deprived areas.
Composite outcome: dialysis/peripheral vascular disease/lower limb amputations/cerebrovascular disease/ischemic heart disease/heart failure/death.
*Analysis of variance.
†χ^2 test.
‡Kruskal-Wallis test.
ACR, urine albumin/creatinine ratio; BMI, body mass index; CVD, cardiovascular disease; HbA_{1c}, glycated hemoglobin; NZDep, New Zealand Deprivation Index; T1D, type 1 diabetes; T2D, type 2 diabetes.

distributed evenly, but 78% of the type 2 diabetes group were living in the lowest four categories and 59% in the two lowest. Ethnicity and NZDep were strongly related with 65% of Māori and 71% of Pasifika living in the lowest two decile areas; so only one of these two variables, NZDep, was used in multiple logistic regression analyses.

Body mass index

For both type 1 and type 2 diabetes, the mean BMI increased progressively with greater degrees of deprivation (p<0.002, table 2).

Glycemic control

For both type 1 and type 2 diabetes, there was a marked gradient across decile groupings (p<0.001; table 2) with those living in the most deprived areas having significantly poorer glycemic control, as defined by higher mean HbA_{1c} values. Multiple logistic regression adjusted for BMI, diabetes type, diabetes duration, and age indicated that each decile increase in NZDep increased the odds of poor glycemic control, defined as HbA_{1c} >64 mmol/mol (>8%) by 17% (OR 1.17, 95% CI 1.13 to 1.22). Type of diabetes (OR 1.32, 95% CI 0.99 to 1.76) and BMI (OR 1.00, 95% CI 0.99 to 1.02) did not show a significant relationship with the risk of poor glycemic control (table 3).

Sixty-three per cent (846) of the type 2 diabetes group had been dispensed diabetes medication in the preceding 6-month period; 54% (735) had been dispensed an oral hypoglycemic agent, and 30% (411) insulin. Of those with type 2 diabetes and an HbA_{1c} ≥100 mmol/mol (≥11.3%), 31% (74/236) had no diabetes medications dispensed in the 6-month period.

Albuminuria

In their year of diagnosis, 644 young people with type 2 diabetes had an ACR result available of whom 46% (295) already had microalbuminuria and 14% (91) macroalbuminuria. At a median of 8 years following diagnosis, 955 young people with type 2 diabetes had ACR results available and 61% (580) had microalbuminuria. At all levels of NZDep young people with type 1 diabetes were less likely to have microalbuminuria or macroalbuminuria than those with type 2 diabetes, but in both types of diabetes there was a marked gradient across the NZDep levels, with those in the most deprived areas having the highest prevalence of abnormal albuminuria. Ordinal logistic regression showed young people with type 2

Table 3 Modelling results for determinants of poor glycemic control and albuminuria (ACR)

Effect	OR	95% CI	P value*
Poor glycemic control (HbA_{1c}>64 mmol/mol)—logistic regression			
Type 2 vs type 1 diabetes	1.32	0.99 to 1.76	0.055
NZDep	1.17	1.13 to 1.22	<0.0001
Duration of diabetes	1.04	1.01 to 1.08	0.005
Body mass index	1.01	0.99 to 1.02	0.477
Age	0.97	0.94 to 0.99	0.007
Higher ACR value (ACR <3.5, 3.5≤ACR <30, ACR ≥30 g/mol)—ordinal logistic regression			
Type 2 vs type 1 diabetes	3.72	2.73 to 5.07	<0.001
NZDep	1.11	1.06 to 1.16	<0.001
Duration of diabetes	1.07	1.04 to 1.09	<0.001
Systolic blood pressure	1.03	1.02 to 1.04	<0.001
Body mass index	1.02	1.01 to 1.04	0.001
HbA_{1c}	1.02	1.02 to 1.03	<0.001
Age at diagnosis	0.99	0.96 to 1.02	0.383
Smoking yes versus no	1.12	0.89 to 1.41	0.337

*χ^2 test.
ACR, urine albumin/creatinine ratio; HbA_{1c}, glycated hemoglobin; NZDep, New Zealand Deprivation Index.

diabetes were at increased odds of having a higher ACR (OR 3.72, 95% 2.73 to 5.07) compared with young people with type 1 diabetes, when adjusted for differences in BMI, systolic BP, HbA_{1c}, smoking status, age at diagnosis, diabetes duration and NZDep. Each decile increase in NZDep increased the odds of having a higher ACR by 11% (OR 1.11, 95% CI 1.06 to 1.16). Each unit increase in BMI (OR 1.02, 95% CI 1.01 to 1.04), BP (OR 1.03, 95% CI 1.02 to 1.04), HbA_{1c} (OR 1.02, 95% CI 1.02 to 1.03), and duration of diabetes (OR 1.07, 95% CI 1.04 to 1.09) was associated with a higher ACR. Smoking (OR 1.12, 95% CI 0.89 to 1.41) and age at diagnosis (OR 0.99, 95% CI 0.96 to 1.02) did not show significant associations (table 3).

CVD risk factors and medication dispensing

CVD risk factors for young people with type 1 and type 2 diabetes and relevant medication use are included in table 1. The proportion prescribed statins or antihypertensive agents was significantly greater for the type 2 group (both p<0.001). Five-year CVD risk estimates showed a positive correlation with NZDep for both type 1 (r=0.21, p<0.001) and type 2 diabetes (r=0.17, p<0.001). Multiple linear regression indicated a 4% increase in CVD risk for every decile increase in NZDep, regardless of type of diabetes.

End organ complications and mortality

Severe end organ complications were analyzed as a composite outcome comprising: end-stage renal failure (on dialysis), peripheral vascular disease, lower limb amputations, cerebrovascular disease, ischemic heart disease, heart failure, or death. The composite outcome measure increased across deprivation categories in the type 1 diabetes group, but not in the type 2 diabetes cohort (table 2). Further detail on differences between the type 1 and type 2 diabetes groups for each complication in the composite outcome has been included in online supplemental appendix 2. Ten young people (1%) in the type 1 diabetes group and 25 (2%) in the type 2 diabetes group had died at a similar age (type 2 mean age 32 (6) years vs type 1 age 28 (7) years, p=0.132). The majority of deaths had been ascribed to diabetes and related causes such as infection and diseases of the circulatory system. Mortality did not show a significant relationship with NZDep.

DISCUSSION

Studies in many western countries including the UK, the USA and Australia have shown that young-onset type 2 diabetes is strongly associated with relative socioeconomic deprivation.[5 11–13 18–20] Young-onset type 2 diabetes has also been recognized to have a more severe or aggressive phenotype with regard to complications than type 1 diabetes of comparable duration.[9–13] In this study, we examined the relationships between a national index of deprivation and important risk factors for the development of major diabetes-related morbidities. The NZDep, which reflects dimensions including income, employment, communication, transport, support, qualifications, home ownership and living space, has been used widely in research and by the New Zealand Ministry of Health for resource allocation.[21–23] The risk factors we considered were: (1) glycemic control—the main modifiable risk factor for the classical microvascular complications of diabetes; (2) albuminuria and obesity—risk factors for kidney disease in diabetes and (3) a cardiovascular risk assessment, which included factors such as dyslipidemia, hypertension, glycemic control, smoking, and albuminuria. The striking observation was that all these risk assessments were significantly and progressively associated with increasing degrees of deprivation, and that these relationships held regardless of the type of diabetes.

In contrast, the distribution of diabetes cases across the NZDep categories was very different: in type 1 diabetes, cases were distributed equally across deprivation

categories, but in type 2 diabetes, as expected, there was a marked clustering toward the most deprived areas. Nearly 60% of cases were in the lowest two groupings, emphasising the point that type 2 diabetes in young people is largely a disease of poverty.[20] If we then factor in the greater risk burden in those living in the most deprived areas, then a potential explanation emerges for the 'more severe' phenotype of young-onset type 2 diabetes compared with young people with type 1 diabetes of comparable age. Factors that are likely to be important include food, housing, and job insecurity, disparities in access to healthcare, the costs of prescriptions and attending clinics, poor literacy, and mental health challenges—all of which limit the possibility of adhering to the necessary lifestyle changes.

As expected, we found that young people with type 2 diabetes were substantially more obese than those with type 1 diabetes, but we also found a marked gradient of BMI across the NZDep categories for both type 1 diabetes and type 2 diabetes. The environment of lower income neighbourhoods is commonly 'obesogenic' with, for example, higher densities of fast-food outlets and fewer exercise facilities, and sometimes issues of safety, that lead to higher levels of physical inactivity.[24–26] The prevalence of obesity has increased substantially in Aotearoa-New Zealand in recent decades paralleling rising inequality.[8 27]

Obesity is of course the major risk factor for type 2 diabetes, but it is also an independent cause of renal disease, most commonly through the development of focal segmental glomerulosclerosis.[28 29] This condition has similarities to classical diabetic nephropathy (bland urine sediment, increased albuminuria and progressive loss of renal function) and the two conditions can co-exist. In Aotearoa-New Zealand, more than half the people taken onto renal replacement treatment programmes have type 2 diabetes, with Māori having threefold to fourfold and Pasifika sixfold to sevenfold higher rates than Europeans.[30] Other studies comparing risk factors for complications in young people with type 1 or type 2 diabetes have noted that the difference in prevalence of microalbuminuria and macroalbuminuria greatly exceeds that of retinopathy.[2 6 10–12] We found that young people with type 2 diabetes had substantially higher levels of albuminuria than young people with type 1 diabetes, but in both groups, albuminuria increased progressively across the deprivation categories, in parallel to deprivation-related changes in BMI. Previous studies have noted that nearly a third of people with type 2 diabetes in our community had no diabetic retinopathy at the time macroalbuminuria was first detected,[31] suggesting obesity-related kidney disease is highly prevalent. Indeed, in the subjects reported here, 46% had microalbuminuria at the time type 2 diabetes was first recognized.

Preventing complications of diabetes depends on adherence to often complex drug regimens commonly comprising two or more antihyperglycemic agents (that may include insulin injections bringing added difficulties), and drugs for dyslipidemia and hypertension. Medication non-adherence is common among low-income patients with chronic conditions.[32] In young adults with type 2 diabetes, it has been shown that adherence to antihyperglycemic agents (as judged by prescriptions lodged with community pharmacies) was closely related to glycemic control and risk of hospitalization.[33] Medication dispensing was suboptimal among young people with type 2 diabetes in this study: only 63% had diabetes medications dispensed and nearly a third (31%) of those with severe hyperglycemia (HbA_{1c}≥100 mmol/mol (≥11.3%)) had not been dispensed any diabetes medication in a 6-month period.

A number of studies have noted that the mothers of young people with type 2 diabetes often have diabetes themselves, or had gestational diabetes. It has been suggested that intrauterine exposures are an important driver of young-onset type 2 diabetes, and that 'diabetes begets diabetes'.[2 13] This arguable proposition may obscure the more important point that 'poverty begets poverty',[34 35] and it is the latter that probably underlies the dramatic increases in young-onset type 2 diabetes seen in Aotearoa-New Zealand over the past few decades.[27 36 37] It is possible to view our data through the lens of 'ethnicity' as 71% of the young people with type 2 diabetes were of Māori or Pasifika descent but, as in many other societies, relative poverty in Aotearoa-New Zealand has a very unequal distribution.[8] Ethnicity itself does not offer a biologically plausible explanation for the findings, whereas the link to inequality does.[38] On the contrary, the fundamental causes of ethnic inequalities in health are social and economic inequalities.[39]

Limitations

There are limitations to our study. Measures such as NZDep cannot, in a single digit, incorporate all aspects of deprivation: some factors likely to impact on the risks of type 2 diabetes and its complications such as adverse childhood experiences are not captured.[40] Some data were missing, most notably for ACR and HbA_{1c} (see footnote to table 1). Only single BP and BMI measurements, the latest available, were included. The lack of data for retinopathy and neuropathy is also a limitation. We did not find clear relationships between NZDep and macrovascular complications or mortality (as opposed to risk factors), but this was likely due to the relatively short duration of diabetes and a low number of events in NZDep subgroup analyses. There was potential selection bias given that this study could not include young people with well-controlled diabetes managed in primary care, or those not referred to our regional diabetes services. However, we believe these numbers are likely to be low. Most young people with type 1 diabetes and the great majority of those with young-onset type 2 diabetes would be referred to specialist diabetes services, because retinal screening is almost exclusively undertaken in the public sector. Of course undiagnosed community cases of type 2 diabetes could not be included, and a challenge in comparing type 1 and type 2 diabetes is the assessment

of diabetes duration, which is often less certain in the latter. Constantino *et al* using the prevalence of retinopathy in their cohorts with type 1 diabetes or type 2 diabetes (diagnosed age 15–30 years) found that delay in diagnosis was unlikely to be an explanation for the differences in outcome measures.[6] Finally, the cardiovascular risk calculator we used has not been independently validated for use in people with type 1 diabetes or for this younger demographic, so while the calculated CVD risk may not truly indicate absolute CVD risk, the increase in risk across deprivation categories is a striking finding.

In summary, our data suggest that the apparently more severe or aggressive phenotype of young-onset type 2 diabetes is at least in part explicable by relative deprivation, the effects of which are evident irrespective of the type of diabetes. This does not exclude the possibility that biological factors that we did not assess (eg, accelerated β-cell failure or impaired insulin action) are also important,[41] but does emphasize that socioeconomic factors must be considered in understanding the biology and complications of young-onset type 2 diabetes.

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Funding A+Trust Project Grant (Number 6889) and Summer Studentship. All authors have completed the Unified Competing Interest form (available on request from the corresponding author) and declare: no support from any organization for the submitted work; no financial relationships with any organizations that might have an interest in the submitted work in the previous 3 years, no other relationships or activities that could appear to have influenced the submitted work. SW affirms with BMJ's transparency policy.

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval Consent for the study was obtained from the Health & Disability Ethics Committee of New Zealand. A waiver for the need for individual consent was granted (reference number: 15/CEN/94). This study did not involve human participants.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request. All data relevant to the study are included in the article or uploaded as supplementary information.

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