



First national audit of the outcomes of care in young people with diabetes in New Zealand: high prevalence of nephropathy in Māori and Pacific Islanders

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Abstract

Background Diabetes is an important cause of morbidity and mortality amongst young people. Despite improvements in technology, maintenance of good glycaemic control is hard to achieve.

Methods In July 2003, 12 paediatric and adult hospital-based diabetes services across New Zealand were invited to take part in an audit of the process and outcomes of care. By March 2004, 9 centres had submitted data on 1282 (1117 with Type 1 diabetes, 105 with Type 2) children and young people born after 1 January 1978.

Results There were significant centre differences in terms of glycaemic control, rates of microvascular complications and complication screening. The group mean HbA1c was $9.1 \pm 0.3\%$. Amongst 789 people aged 16–25 years, the prevalence of retinopathy was 12.8% (range 0–26%); nephropathy was 17.1% (range 7–28%). Of those with a duration of diabetes >10 years, 25% had retinopathy and 27% nephropathy. Over the age of 12, microalbuminuria was more common amongst Māori and Pacific Islanders (43.8%) compared to Europeans (17%) or Others (17.8%). This was independent of the type of diabetes.

Conclusions This is the largest study of young people with diabetes undertaken in New Zealand. The results confirm the difficulty of achieving good glycaemic control in children and young adults. Microvascular complications were common, particularly in those of long duration, and cardiovascular risk factors were present in many young adults. The difference in average HbA1c% between centres was highly significant and independent of other factors. Type 2 diabetes mellitus in young people was associated with early onset nephropathy and dyslipidaemia (almost from diagnosis), thus suggesting the need for earlier diagnosis.

New Zealand (NZ) has a population of 4 million people with approximately 20% identifying themselves as Māori, and 5% as Pacific Islanders. In 2000, there were an estimated 115,000 people with known diabetes (NZ Ministry of Health data)—predominantly Type 2 diabetes mellitus (T2DM).

Under the age of 26 years, most people with diabetes have Type 1 diabetes mellitus (T1DM)—although with the rising tide of obesity, more teenagers and young adults are found to have T2DM. The estimated number of people with diabetes under age 26 in NZ is uncertain, but Christchurch data indicate an increase in T1DM over the last 30 years.^{1,2}

Children diagnosed with diabetes have greater morbidity and mortality at all ages compared to their non-diabetic counterparts.³⁻⁵ Moreover, compared with adult-onset diabetes patients, the risk of developing renal or retinal complications is greater if diabetes is diagnosed under 15 years.⁶

It is over 10 years since the Diabetes Control and Complications Study reported the beneficial effects of tight glycaemic control in adults and adolescents.⁷ The care of young people with diabetes is challenging, and recent studies from Europe and Japan have illustrated the difficulties of achieving and maintaining good glycaemic control. The studies also highlight the high prevalence of complications and the wide range of glycaemic control between centres (unrelated to patient selection or choice of insulin regimen).⁸⁻¹³ Nevertheless, some centres consistently have mean HbA1cs as good if not better than the intensive arm of DCCT, without the increased risk of hypoglycaemia.¹⁴⁻¹⁶

Little is known about the prevalence or progression of Type 2 diabetes in children and young adults in New Zealand, but with the increase in obesity, the picture is likely to mirror that of the rest of the World.¹⁷

Methods

Study design—This audit had approval from the local ethics committee of each participating district health board (DHB).

The all-NZ young person's diabetes audit was begun in July 2003 when 14 centres (adult and paediatric diabetes services in each) covering 2.3 million of the NZ population were invited to participate in an audit of the process and outcomes of care of young people with diabetes up to the age of 26. By April 2004, 1 centre in South Island and 8 in the North (8 adult and 6 paediatric) had submitted data. Data from the Waikato has been published in detail elsewhere.¹⁸

If they had attended a diabetes centre at least once in the previous 3 years, any person with diabetes born after 1 January 1978 was eligible for inclusion in the study. Up to 45 data items (including date of birth, duration of diabetes, last weight, height and BMI, lowest HbA1c during first year after diagnosis, last HbA1c (and mean of last 3), presence of microvascular complications, and details of treatment regimen were collected from either paper health records or electronic diabetes registers.

Pathology laboratory databases were searched for missing test results. The hospital number was used to eliminate or combine duplicates where individuals had attended more than one centre over the last 3 years.

HbA1c was measured by a variety of methods but all were Diabetes Control & Complications Trial (DCCT)-aligned. Microalbuminuria was defined as an early morning urine with an albumin:creatinine ratio of >2.5 in males (>3.5 females) on more than one occasion. Where available the number of abnormal urines was recorded.

Statistics—Statistical analysis was performed using SPSS for Windows (version 12, SPSS Inc., Chicago, IL) software. Univariate ANOVA was used to compare centre differences (using HbA1c as the dependent variable), with age and duration of diabetes as covariates.

Chi-squared was used to compare ethnic differences and type of diabetes. Pearson correlation coefficient was used to explore the relationship between early glycaemic control and recent HbA1c. The significance value was set to 5%

Results

There were 1282 (1251 after duplicates removed) people with diabetes under the age of 26 as of 1 July 2003. The number, gender, ethnicity, and type of diabetes by centre can be seen in Table 1. Of Europeans with diabetes, 90.4% had T1DM compared to 66% of non-Europeans ($p < 0.0001$).

Table 1. Characteristics of young people with diabetes (0 to 25 years) by centre

Diabetes centre	1	2	3	4	5	6	7	8	9	All
Total patient numbers	164	252	150	71	37	109	62	45	392	1282
DHB pop	284000	326000	434000	177000	165000	135000	100000	115000	477000	2177000
Prevalence/1000	0.7	0.8	0.4	0.4	0.2	0.6	0.6	0.4	0.8	0.6
Gender (no. Female)	90F	135F	74F	40F	17F	38F	27F	26F	194F	641F
T1DM (%)	93.3	94.4	90.0	90.1	91.9	94.5	80.6	95.6	84.9	90.4
T2DM (%)	6.1	5.2	9.3	4.2	8.1	5.5	17.7	4.4	12.6	8.7
European (%)	83.5	84.1	94	81.7	94.6	82.6	69.4	80.0	70.2	80.1
Māori (%)	1.8	10.7	1.3	14.1	2.7	7.3	21	13.3	5.9	7.3
Pacific Islander (%)	4.3	2.0	0.7	1.4	2.7	1.8	0.0	2.2	11.7	5.0
Other (%)	10.4	3.2	4.0	2.8	0.0	8.3	9.7	4.4	12.2	7.6
Age (SE)	16 (0.4)	17.7 (0.3)	19.1 (0.3)	17.8 (0.5)	20 (0.4)	15.5 (0.6)	22.9 (0.6)	14.3 (0.9)	17.8 (0.2)	17.9 (0.5)
Duration (SE)	6.6 (0.4)	7.3 (0.3)	7.9 (0.4)	5.2 (0.5)	7.6 (0.8)	7.3 (0.9)	7.6 (0.6)	5.9 (0.7)	6.8 (0.3)	6.9 (0.6)

DHB pop is the estimated population of the district health board (DHB) serving the centre.

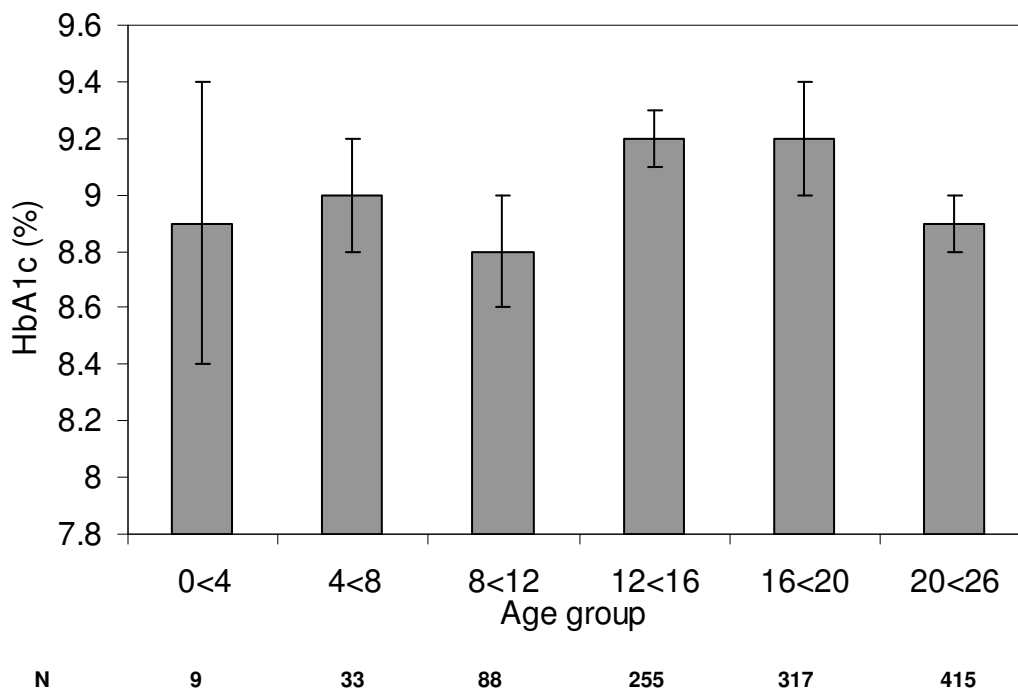
Prevalence/1000 is the prevalence of diabetes (in young people 25 years and younger) per 1000 population.

T1DM (%) is percentage of total with Type 1 diabetes.

T2DM (%) is percentage of total with Type 2 diabetes.

The mean HbA1c was $9.1 \pm 0.1\%$. After correcting for gender and ethnicity, there were significant differences in mean HbA1c between the 2 centres, with the highest values ($9.6 \pm 0.2\%$) and lowest ($8.5 \pm 0.12\%$) values ($p=0.000$). There were significant differences in HbA1c between age groups ($p=0.034$) (Figure 1).

Figure 1: Glycaemic control by age group (T1DM). Using ANOVA there are significant differences in HbA1c between age groups ($p=0.034$)



When type of diabetes was included as a variable, age group remained significant ($p=0.06$). For T1DM alone, age group ($p=0.017$) and centre ($p<0.0001$) were highly significant.

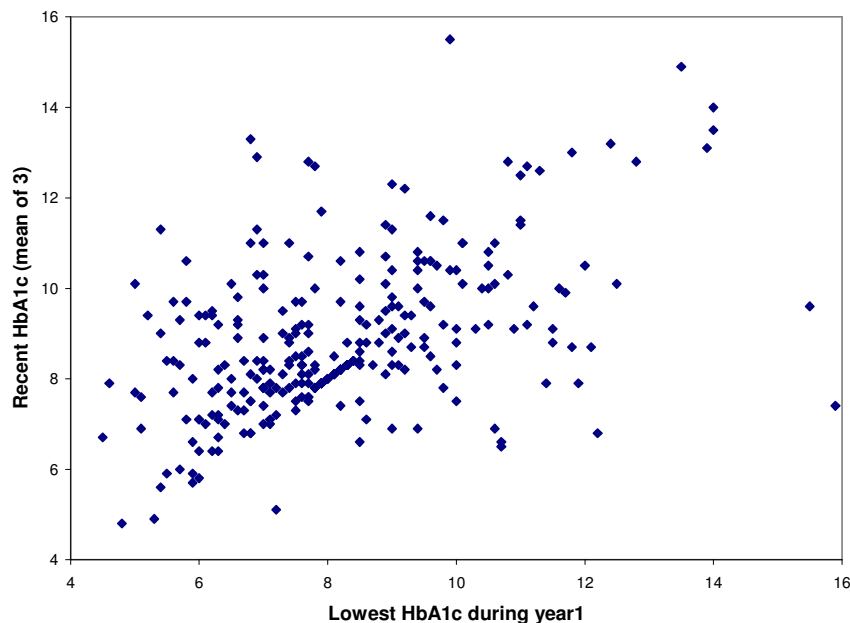
There were no ethnic differences in glycaemic control either during the first year after diagnosis, or the latest or the mean, HbA1c. An HbA1c measured during the first year after diagnosis was available in 274 (27%) of the subjects. The mean of the lowest recorded HbA1c during that year was $8.2 \pm 0.1\%$.

Of those who had a recorded HbA1c during this period, 12.5% were in the normal range. There was a positive correlation between the lowest HbA1c during the first year after diagnosis and future glycaemic control (Figure 2).

There were 662 subjects with Type 1 diabetes between the ages of 16–25 years. Over 50% were on multiple injection therapy (4 or more injections per day); 23 patients were on pumps (predominantly from 1 centre). There was no correlation between number of injections and glycaemic control. The prevalence of microalbuminuria and

retinopathy varied considerably from centre to centre, and increased with increasing duration of diabetes (Figure 3 and Figure 4).

Figure 2: Relationship between lowest HbA1c during first year after diagnosis and latest HbA1c (mean of last 3) in a sub-sample of 274 who had HbA1c results available from the first year after diagnosis. Pearsons correlation is significant ($p < 0.01$) (2-tailed)

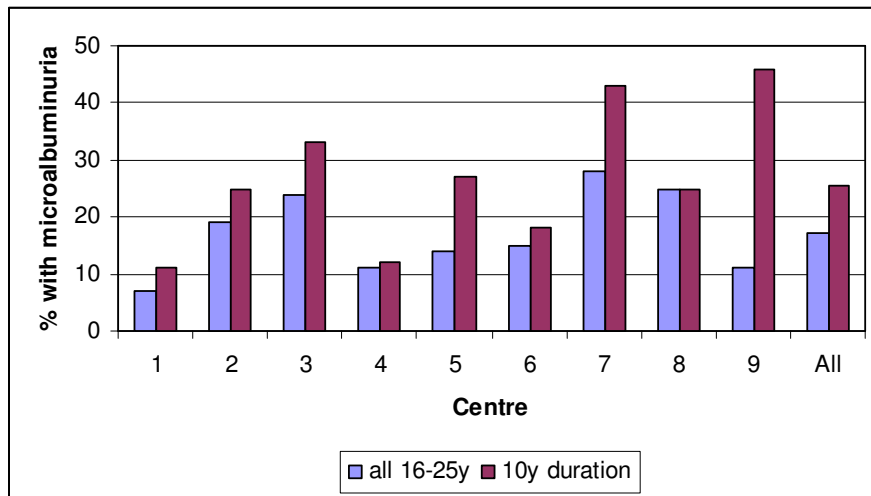


There were 105 subjects with T2DM; 63% were non-European. The mean age was 20 ± 0.4 y with duration of 3 ± 0.3 y. All were overweight (mean \pm SE BMI 35 ± 0.8). The majority were managed with diet alone, 19% insulin treated and 8% on Metformin or Acarbose with or without insulin.

The mean HbA1c was $8.5 \pm 0.2\%$; 20% were hypertensive (blood pressure [BP] $> 130/80$ mmHg), 72% had microalbuminuria (of whom 19% were treated with ACE-inhibitors), 4% had background retinopathy, and 4% had sight-threatening retinopathy.

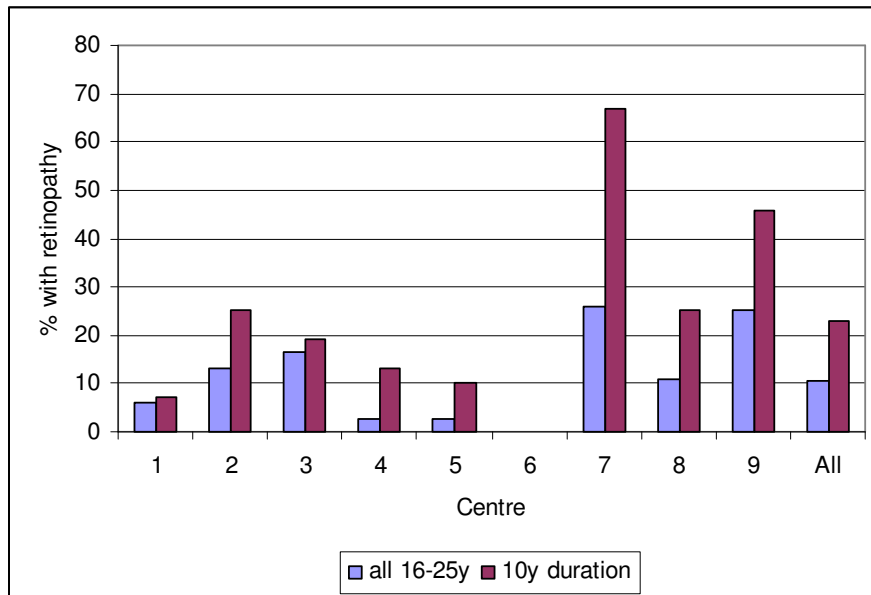
The mean total cholesterol was 5.5 ± 0.1 mmol/L, HDL cholesterol 1.2 ± 0.05 mmol/L, triglycerides 3.5 ± 0.5 mmol/L. Amongst those with T2DM, hyperlipidaemia was common with 61.5% having a total cholesterol > 5.0 mmol/L, 36% an HDL < 1 mmol/L, and 52.6% with triglycerides > 2.0 mmol/L. Only 3% were on lipid-lowering drugs.

Figure 3. Prevalence of microalbuminuria in 16–25 year olds with T1DM (left bars), including those with diabetes for 10 years (y) (right bars)



Over the age of 12 years, microalbuminuria were more common amongst Māori and Pacific Islanders (43.8%) compared to Europeans (17%) or Others (17.8%). This was independent of the type of diabetes.

Figure 4. Prevalence of retinopathy in 16–25 year olds with T1DM and effect of duration of diabetes (Centre 6 reported no retinopathy)



Discussion

This is the first study to look at the outcomes of care of children and young adults with diabetes across NZ and involved 8 adult and 6 paediatric diabetes services in 9 centres. The results demonstrate a disappointing picture of poor glycaemic control and moderately high rates of microvascular complications, as seen in other studies. Despite widespread use of multiple injection therapy, at all ages, few people achieved satisfactory control (only 22% had a recent HbA1c < 8%).

Although glycaemic control in each centre was poor, it is similar to other published studies in Europe of unselected young people with diabetes.⁸⁻¹² Little data on young people with diabetes in NZ is available. In 2002, the Christchurch group reported a mean HbA1c for females aged between 13 and 20 years was 10.2% and 9.5% for males.¹⁹

The type of insulin regimen (including use of pumps) did not appear to have much impact on glycaemic control. Our finding of a relationship between the HbA1c during the first year after diagnosis and future glycaemic control is consistent with data from Australia which suggested that poor control in childhood led to poor control in adolescence and beyond.²⁰

Other studies have suggested that poor early control is associated with a four-fold increase in the subsequent prevalence of nephropathy.²¹ An intriguing observation of the DCCT collaborators was that tight control initiated a year after diagnosis was associated with preservation of islet cell function for a greater period than the group randomised to conventional (poor) control.²²

There have been a few small studies (but no long-term randomised studies) looking at the impact (on beta cell function) of intensive normalisation of glycaemic control from diagnosis, with conflicting results.^{23,24}

Recent twin studies may offer an alternative explanation for the association between early and long term glycaemic control, which suggest that 62% of the population variance in HbA1c levels is genetically determined and independent of the genes influencing fasting blood glucose.²⁵

This is unlikely to be the explanation, since both in our study and in a UK study²⁶ there were marked differences between centres in the number of children with a normal HbA1c during the first year after diagnosis. This implies differences in both expectation and training of the person with diabetes rather than differences in genetics.

Access to health is not always equitable and socioeconomic factors may explain some of the differences between centres, though in the Scottish study, age, sex, insulin regimen, BMI, season, social circumstances, and family history were all associated with glycaemic control but not with deprivation score based on post code.⁸

The DCCT trial also suggested a period of improved control during adolescence is associated with long-term improvements in risk of complications—although HbA1c became similar in the intensive management and control groups soon after the end of the DCCT, the benefits of intensive management on microvascular complications persisted.²⁷ This suggests adolescence is a critical period for future risk of complications.

The success of the DCCT, and the difficulties in obtaining similarly improved control outside the clinical trial setting (together with the association we have observed between metabolic control soon after diagnosis and future metabolic control) suggest that more intensive effort in diabetes education, support, and motivation (as occurred in the intensive arm of the DCCT along with intensive insulin management) may be particularly important during the first year after diagnosis as well as during adolescence.

The prevalence of retinopathy amongst those screened was similar to published series,^{9,10,28,29} but the difference in screening methods between centres (from direct ophthalmoscopy without pupillary dilatation to retinal photography with mydriasis) makes comparisons between centres difficult.

Microalbuminuria rates showed less variation which makes the finding of no retinopathy in one centre (where screening was with direct ophthalmoscopy) suspect, and emphasises the need for standardised screening methods across the country.

Some centres did not begin retinal screening until 16 whereas the International Society for Paediatric and Adolescent diabetes (ISPAD) guidelines are to start screening for microvascular complications from either 5 years after diagnosis or age 11 (whichever is earlier) with pre-pubertal diagnosis or from 2 years from diagnosis with pubertal onset.³⁰ The extremely high prevalence of microalbuminuria in those with T2DM, and the known high incidence of renal failure and increased mortality in Māori and Pacific Islanders with the metabolic syndrome,³¹⁻³³ make early diagnosis and intervention essential. This ethnic predisposition to nephropathy was apparent in both T1DM and T2DM.

Although the cause of nephropathy was not confirmed by renal biopsy in our study, adult studies in obese patients with T2DM suggest that persistent microalbuminuria is associated with either diabetic nephropathy, or obesity related focal and segmental sclerosis.

Use of ACE inhibitors is reasonable in those with confirmed nephropathy, although in part this depends on the criteria for diagnosis (in one centre, of 13 young people with 3 or more abnormal results, 11 were on ACE inhibitors. None of those with just 2 abnormal results were treated with ACE-inhibitors).

A recent publication found that up to 60% of people with T1DM have spontaneous resolution unrelated to ACE inhibitor use.³⁴ This finding, and concerns about using ACE inhibitors in young women of child bearing age, may be the reasons why they are not frequently used.

Sub-optimal lipid profiles were very common especially in those with type 2 diabetes, yet only 3% were receiving any lipid lowering therapy. Cardiovascular Risk charts underestimate risk and are inappropriate for this age group.³⁵ Only the American Diabetes Association has published specific guidelines for young people with T1DM.³⁶

With the knowledge that most will die prematurely from a vascular accident, and that vascular disease is even more common in T2DM (especially in Māori and Pacific Islanders), earlier use of statins may be appropriate. As with use of ACE inhibitors, however, consideration has to be given to the risk to the developing foetus in the event of conception occurring whilst taking them.

Nearly 36% of those with T1DM over 16 yrs, and 100% of those with T2DM, are overweight. This reflects obesity in the community as in 1997 approximately 25% of 15 to 18 year olds and one in three 19 to 24 year olds were overweight or obese.³⁷ In addition, weight gain (especially in girls) on intensive insulin therapy can sometimes be spectacular,³⁸ and likely to be a disincentive to better glycaemic control.

A limitation of our study is that the exact number of people under 26 years of age with diabetes in NZ is unknown. However, a careful prevalence study from Christchurch, New Zealand³⁹ estimates that there are approximately 2540 with T1DM in this age group in the country.

Using their estimates of prevalence for the participating centres we appear to have identified 92% of predicted. Of those young people 'lost to follow-up', the published literature suggests they have worse control and greater risk of complications.⁴⁰

Another limitation is that not all centres in New Zealand participated in the study. Of concern, some were unable to gather the data. On the basis of studies from countries with similar socioeconomic circumstances as New Zealand, it is unlikely that the addition of data from these remaining centres would have altered our conclusions, but it is vital that all centres (caring for children and young adults with diabetes) audit their services and we urge them to do so.

In summary, this multicentre study of nearly 50% of the children and young people with diabetes in NZ has revealed large numbers with poor glycaemic control and a disturbing prevalence of early microvascular disease, despite introduction of intensive insulin therapy. Nevertheless, there are highly significant differences between centres independent of other factors suggesting opportunities for improvement.

The factors influencing success or otherwise in achieving good glycaemic control need to be investigated further. There needs to be greater adherence to management guidelines in screening for complications. T2DM in young people is becoming a major problem and is associated with early onset nephropathy and dyslipidaemia (almost from diagnosis), thus suggesting the need for earlier diagnosis, which is likely only going to be achieved by targeted screening of high risk children and young adults.

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Acknowledgements: We thank Professor Michael Greenacre (Department of Economics, University Pompeu Fabra, Barcelona, Spain) for statistical help and advice as well as Robyn Toomath for the original idea for this study.

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