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Title

Type 2 diabetes in children and adolescents across Australia and New Zealand: a 6 year audit from The Australasian Diabetes Data Network (ADDN)

Running title

Pediatric type 2 diabetes ADDN audit

Authors

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Where indicated, "data was sourced from the National Diabetes Services Scheme (NDSS) – an initiative of the Australian Government administered with the assistance of Diabetes Australia. Through the NDSS, Diabetes Australia provides diabetes self-management products and support services to over one million Australians with diabetes."

Author contributions

Study designed by KL, PB, MC, KD, AM
Data supplied by the ADDN study group
Data analysed by KL and PB
Manuscript prepared by KL, PB, MC, KD, AM

Conflict of interest disclosure

Conflict of interest declaration for author AM: Honorarium paid for participation in Medtronic National Advisory Board meeting 15 November 2019, to discuss in

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Abstract

Objectives: To assess the clinical and demographic characteristics of children and adolescents across Australia and New Zealand with type 2 diabetes.

Methods: We performed a descriptive audit of data prospectively reported to the Australasian Diabetes Data Network (ADDN) registry. Data were collected from six tertiary paediatric diabetes centres across Australia (New South Wales, Queensland, South Australia, Western Australia and Victoria) and New Zealand (Auckland). Children and adolescents diagnosed with type 2 diabetes aged ≤18 years with data reported to ADDN between 2012-2017 were included. Age, sex, ethnicity, HbA1c, blood pressure, BMI, waist circumference and lipid profile at first visit were assessed.

Results: There were 269 cases of type 2 diabetes in youth reported to ADDN between 2012 and 2017. The most common ethnicities were Indigenous Australian in 56/243 (23%) and New Zealand (NZ) Maori or Pacifica in 47 (19%). Median age at diagnosis was 13.7 years and 94% of participants were overweight or obese. Indigenous Australian and Maori/Pacifica children were younger at diagnosis compared with non-Indigenous children: median 13.3 years (Indigenous Australian); 13.1 years (Maori/Pacifica); 14.1 years (non-Indigenous), p=0.005. HbA1c was higher in Indigenous Australian (9.4%) and Maori/Pacifica youth (7.8%) compared with non-Indigenous (6.7%) p<0.001. BMI-SDS was higher in Maori/Pacifica youth (2.3) compared with Indigenous Australian (2.1) and non-Indigenous (2.2) p=0.011.

Conclusions: Indigenous Australian and Maori/Pacifica youth in ADDN were younger and had worse glycaemic control at diagnosis of type 2 diabetes. Our findings underscore the need to consider targeted and earlier screening in these “high risk” populations.

Key words

Diabetes mellitus, type 2

Child

Adolescent

Indigenous peoples

Introduction

Type 2 diabetes diagnosed during childhood and adolescence is associated with a higher risk of complications and premature death compared with a diagnosis in adulthood, despite similar disease duration.[1] Furthermore, type 2 diabetes in youth is associated with a poorer prognosis and high prevalence of comorbidities compared with type 1 diabetes.[2,3]

Global trends suggest the incidence of type 2 diabetes in children and adolescents is rising, particularly within the Indigenous and ethnic minority populations of industrialised countries such as the United States, Canada and New Zealand.[4-9] State-based data from Australia suggested a plateau in recent years.[10] There are limited data available on the phenotype of this high-risk group in Australasia. The Australasian Diabetes Data Network (ADDN) provides an opportunity to describe the demographic and clinical characteristics of youth at diagnosis of type 2 diabetes in Australia and New Zealand.

The ADDN database involves prospective collection of de-identified patient data from the electronic records of participating centres. Data are collected in a longitudinal, visit-based approach, using a common data dictionary. Every 6 months data are transferred from participating ADDN centres to a central ADDN registry.[11]

Methods

This was a descriptive audit of type 2 diabetes incident cases in youth aged ≤18 years reported to the ADDN registry between January 2012 and December 2017. A diagnosis of type 2 diabetes was made by the treating clinician. Pediatric endocrinologists in Australia and New Zealand follow the International Society for Pediatric and Adolescent Diabetes (ISPAD) guidelines for diagnosis of type 2 diabetes.[12] Antibodies are routinely performed at diagnosis of diabetes in children and adolescents and genetic testing for monogenic diabetes is readily accessible in the public health system and routinely performed as clinically indicated. Data were obtained from six tertiary paediatric diabetes centres that had prospectively enrolled participants with type 2 diabetes across Australia (New South Wales, Victoria, Queensland, South Australia, Western Australia) and New Zealand (Auckland) from 2012 onwards. Data extracted from ADDN included demographic characteristics (age,

sex, ethnicity, date of diagnosis), clinical phenotype (body mass index (BMI), waist circumference and blood pressure) and biochemical measures to assess glycemic control (HbA1c) and lipid profile (LDL cholesterol and triglycerides). We analysed data on the participant's first visit recorded in ADDN.

Ethnicity was defined according the Australian Bureau of Statistics *Australian Standard Classification of Cultural and Ethnic Groups 2016* and Stats NZ *Ethnicity.*[13,14] For this analysis, youth were categorised as Indigenous Australian, New Zealand Maori/Pacifica, or non-Indigenous.

Overweight and obesity were defined according to the International Obesity Task Force (IOTF) age and sex-specific criteria.[15] Waist circumference percentiles were based curves produced from the US National Health and Nutrition Survey data for ages 5-19 years.[16] Hypertension was defined according to the 2017 American Academy of Pediatrics Clinical Practice Guideline as a systolic and/or diastolic blood pressure >95th percentile for participants aged <13 years and >130/80mmHg for participants aged ≥ 13 years.[17] Hypercholesterolaemia was defined as LDL cholesterol >3.36 mmol/L (131 mg/dL) and hypertriglyceridemia >1.7 mmol/L (150 mg/dL) based on the recent Australasian Paediatric Endocrine Group type 2 diabetes consensus guidelines.[18]

Descriptive statistics are reported as mean ± standard deviation (SD) for parametric data and median [interquartile range, IQR] for non-parametric data. Clinical characteristics were compared between sexes and across the three main categories of ethnicity. Between-group comparisons were performed using Mann Whitney U tests for non-parametric continuous variables and Chi square tests for categorical data. Comparisons between multiple groups were performed using Kruskal-Wallis H-tests. Analyses were performed using SPSS version 25, IBM, New York.

Ethics approval was granted by the Hunter New England Human Research Ethics Committee (HREC) (for all New South Wales sites: NSW HREC reference - HREC/08/HNE/379), Children's Health Services Queensland HREC (for all Queensland sites: HREC reference - HREC/09/QRCH/68; amendment - HREC/09/QRCH/68/ AM04), The Women's and Children's Health Network HREC (for

all South Australian sites: HREC reference - REC1048/06/2019), The Royal Children's Hospital, Melbourne HREC (HREC reference - HREC 37124; amendment - 37124D), Perth Children's Hospital HREC (for all Western Australian sites: reference, 2013051EP) and Starship Children's Hospital Northern A Health and Disability Ethics Committee (HDEC Ethics reference - NTX/12/EXP/076/AM04). Informed consent for database inclusion was obtained from parents and, when required, assent was obtained from participants aged 10-14 years.

Results

Over the six-year study period, there were 269 cases of type 2 diabetes in youth (42% male) reported across the six tertiary paediatric centres in Australasia. The number of cases reported by individual centres ranged from 4 to 88. Clinical and demographic characteristics are reported in Table 1. Ethnicity was classified as Maori/Pacifica in 47/243 (19%) and Indigenous Australian in 56 (23%) of participants. Indigenous Australians accounted for 30% of participants from Australian centres. Median age at diagnosis was 13.7 years (range 6 to 18) and median duration since diabetes diagnosis was 0.2 years at the first clinic visit.

Overweight or obesity was present in 94% of participants. The median age-adjusted waist circumference percentile was 97 [IQR 93-99] and median waist/height ratio was 0.61 [0.55-0.66]. Hypertension was present in 35% of participants at first visit. Median HbA1c at first visit was 7.3 [IQR 6.0-9.2]%, IFCC: 56 [42-77] mmol/mol. Elevated LDL cholesterol was present in 29% of participants and 43% had raised triglycerides, however fasting status was not documented in half of those with hypertriglyceridemia.

Compared with males, females were younger at diagnosis (13.1 vs 14.3 years, p=0.002) and had a higher proportion of elevated LDL cholesterol (41% vs 13% p=0.012). There were no significant gender differences in HbA1c or prevalence of other comorbidities (obesity or hypertension).

Participants with a normal BMI at presentation (15/260, 6%) were less likely to be hypertensive (0%) than overweight/obese participants (60%) p=0.012. However, they presented with a higher median HbA1c of 8.8 [7.4-11.1]%, IFFC 72 [58-102] mmol/mol

vs 7.2 [6.0-9.1]%, IFFC 55 [42-76] mmol/mol in the overweight/obese group (p=0.015). There was no difference in age at diagnosis or hypercholesterolemia.

Indigenous Australian and Maori/Pacifica youth were significantly younger at diagnosis compared with non-Indigenous youth (13.3 and 13.1 years vs 14.1 years respectively) p=0.005. Thirteen percent of Indigenous youth were aged <10 years at diagnosis, compared with 9% of Maori/Pacifica and 4% of non-Indigenous (p=0.031) (Figure 1). HbA1c was significantly higher in Indigenous Australian compared with Maori/Pacifica and non-Indigenous youth (9.4%; 79 mmol/mol vs 7.8%; 62 mmol/mol vs 6.7%; 50 mmol/mol respectively p<0.001). BMI-SDS was significantly higher in Maori/Pacifica compared with Indigenous Australian and non-Indigenous youth (2.3 vs 2.1 vs 2.2. p=0.011). There was no statistically significant difference in rates of hypertension and hypercholesterolaemia between the ethnicities (Table 2). There were insufficient data for meaningful waist circumference comparisons.

Discussion

We present the first and largest profile of youth with type 2 diabetes across paediatric centres in Australia and New Zealand from the ADDN registry. Our data suggest Indigenous Australian and Maori/Pacifica youth are at higher risk of developing type 2 diabetes and metabolic syndrome at a younger age. The majority of participants were overweight or obese, and around a third were hypertensive and/or had hypercholesterolaemia.

Higher rates of type 2 diabetes have been reported in Indigenous populations in Canada and the United States.[6,19] Previous Australian studies have reported a 6 to 20-fold higher incidence of type 2 diabetes in Indigenous Australian youth.[7,20] Although this was not a prevalence study, our data demonstrated that 30% of youth with type 2 diabetes from the Australian ADDN centres were Indigenous Australian. Based on population estimates by the Australian Bureau of Statistics in 2017, Indigenous Australian youth made up 4.9% of 6 to 18-year-olds in states represented by ADDN centres.[21,22] While we were unable to calculate national prevalence estimates as we included youth seen at tertiary centres only, Indigenous Australians were clearly overrepresented in this cohort compared with non-Indigenous youth with type 2 diabetes.

Our data demonstrate females were diagnosed with type 2 diabetes at a younger age, and had higher LDL levels than males, which has not been previously described in population studies to date. This age difference may be secondary to the earlier puberty seen in females. The SEARCH study did not specifically report the age of diagnosis by sex, but found a higher prevalence of type 2 diabetes in females under 19 years of age in some ethnic groups.[19] Similarly, the TODAY study showed that female participants with type 2 diabetes were younger than males despite a similar duration of diabetes, implying a younger age at diagnosis in females.[23] The rates of raised LDL levels, however, were similar between the sexes.

The median HbA1c at first visit was lower at 7.3% (56mmol/mol) than earlier reports in Western Australia (10%)[8] and Canada (8.7%)[6] but consistent with the HbA1c of 7.3% reported in New South Wales after a median diabetes duration of 1.3 years.[3]

The discrepancy may be explained by the time at which some of these studies took place: the Western Australian study covered a period from 1990-2002. It may be that greater awareness and screening for type 2 diabetes in youth in the decades since these studies were conducted has resulted in earlier detection of type 2 diabetes. This is supported by the median HbA1c of 7.7% in the Western Australian participants in our study, which is based on data collected up to 27 years later than the previous study.[8]

Our data describe a more aggressive disease profile in Indigenous Australians and Maori/Pacifica. Compared to their non-Indigenous counterparts, they presented at a younger age and with higher HbA1c at the first visit. A greater proportion of Indigenous Australian and Maori/Pacifica youth were aged <10 years at diagnosis (13% and 9% respectively) compared to non-Indigenous youth (3%). This is consistent with the Canadian Aboriginal population, in which 11% of youth with type 2 diabetes presented before age 10 years.[6] Despite this poor metabolic profile, Indigenous Australian children presented with a lower BMI than Maori/Pacifica youth and similar BMI to non-Indigenous Australian youth. Such differences in BMI between indigenous and non-indigenous youth with type 2 diabetes were also observed in the SEARCH study. Navajo youth with type 2 diabetes were underweight or normal weight in around 16% of cases, compared with approximately 10% of non-Hispanic white youth and <10% of African American youth.[24-26]

The proportion of overweight and obesity in our participants varied across ethnic groups. However, BMI SDS can be a poor measure of adiposity. Studies using skin fold thickness and bioelectric impedance have reported a higher fat mass for a given BMI in Indigenous Australian adults compared with European Australians.[27,28] Conversely, a study in Maori and Samoan adults found that a BMI above 25 kg/m^2 was associated with a higher fat-free mass on DXA scan than their European counterparts at the same BMI.[29] These findings highlight the complexity in comparing BMI across ethnicities. In contrast, the higher relative fat mass at a given BMI in Indigenous Australians indicates that the current BMI thresholds for overweight and obesity may result in underdiagnosis in this group. Additionally, a greater waist circumference and intra-abdominal fat distribution has been reported in Indigenous Australian adults.[27]

Interpretation of the higher BMI in Maori and Pacifica participants of this study is also difficult. Although a higher lean tissue mass for a given BMI has been reported in Maori and Pacifica peoples, there is evidence that this group displays a similar central/visceral fat distribution to that seen in Indigenous Australians. Elite athletes of Polynesian descent were found on MRI and DEXA to have higher relative abdominal adiposity despite a higher overall lean mass compared with their Caucasian counterparts, which is associated with increased cardiometabolic risk.[30]

Given that both Indigenous Australian and Maori/Pacifica groups may present with central adiposity, and the difficulty interpreting BMI in these groups, it is possible that a waist circumference measurement would be a more useful marker of type 2 diabetes risk. Waist circumference and waist/height ratio Z-scores are more strongly associated than the BMI Z-score with abnormal lipid profiles in children and adolescents.[16] Similarly, a meta-analysis has found that these measures are more predictive than BMI of adverse cardiometabolic outcomes in adults.[31] We were unable to compare waist circumference between ethnicities from the available data in our participants. However, waist circumference and waist/height ratio may be particularly useful to determine metabolic risk and provide an indication for screening in young people of Indigenous Australian and Maori/Pacifica descent. This is reflected in the recent APEG guidelines recommending screening in young people of Indigenous backgrounds with a waist/height ratio of >0.5.[18] Furthermore, the higher HbA1c noted in the participants who presented in the normal weight range may partly reflect the difficulty in using BMI to predict metabolic risk.

Differences in HbA1c between ethnicities have been reported previously. Significant differences in HbA1c at presentation based on ethnicity were found in the SEARCH cohort, even after the data were adjusted for age, sex and insurance payer (as a surrogate for socioeconomic status), however the HbA1c in Native American youth was not specified.[32] This differs from a previous report in New South Wales which found no difference in HbA1c at diagnosis between Indigenous and non-Indigenous Australian youth.[20]

Although the more aggressive metabolic profile in Indigenous Australian and Maori/Pacifica youth may relate, in part, to genetic differences, social inequity likely plays a key role. Indigenous Australians and Maori have a lower life expectancy than their non-Indigenous counterparts.[33,34] The *Close the Gap* 2016 progress report lists access to health care, institutional racism and provision of culturally competent services as key hurdles for improving the health of Indigenous Australians.[34] Reduced healthcare accessibility for Indigenous youth may explain the higher HbA1c at diagnosis in our cohort, indicating more advanced disease at presentation and a potential diagnostic delay. This trend would likely be amplified in rural and remote areas where access to health care is even more problematic.[35] These concerns around diagnostic delay and healthcare access again emphasize the importance of early and targeted screening in this population, including in urban areas where BMI may be higher.[36]

Despite the substantial caseload reported across these six tertiary centres, our analysis was limited by incomplete case ascertainment, as evidenced by the variable number of cases reported across centres. Data collected by the National Diabetes Services Scheme (NDSS) provides a useful comparison to the number cases of type 2 diabetes captured by ADDN. The NDSS is an Australian Government service which enables access to subsidised diabetes products for people with diabetes.[37] Based on data obtained from the NDSS, ADDN captured around one third of the cases of type 2 diabetes in youth registered with the NDSS in 2016 and 2017. The three Australian states and territories yet to be included as ADDN centres were adjusted for, so this result is a reasonable reflection of reduced case ascertainment in states with ADDN centres.

ADDN does not ascertain all youth with type 2 diabetes managed solely by general paediatricians, adult endocrinologists and general practitioners. A higher proportion of Indigenous than non-Indigenous youth in Australia live in rural and remote areas, which are less likely to be captured in tertiary hospital-based data. Hence, despite being over-represented, this patient group is likely underreported. Some ADDN centres do provide outreach services to rural areas and these participants were included in our data. Despite this, the inclusion of participants solely from tertiary services may explain why the case ascertainment was much lower than the NDSS

(any patient with diabetes can register with the NDSS). However, the NDSS data may also over-represent cases of type 2 diabetes. As the number of ADDN centres expands, especially into areas with a high Indigenous Australian population such as the Northern Territory, our ability to capture data on this high-risk subgroup will improve.

The diagnosis of type 2 diabetes was based on physician report. Since the data were obtained from paediatric endocrinology units at tertiary hospitals (where diabetes autoantibodies are routinely performed to exclude type 1 diabetes, along with measurement of C-peptide and targeted screening for monogenic diabetes), it is reasonable to assume that the diagnosis was correct in the majority of cases. While the inability to independently confirm the diagnosis is an inherent limitation of a registry, the ADDN data dictionary clearly specifies that diagnosis of T2D is based on negative diabetes autoantibodies and exclusion of monogenic diabetes.

In summary, Indigenous Australian and Maori or Pacifica youth with type 2 diabetes in this cohort presented at a younger age and had worse glycaemic control than non-Indigenous youth. This is alarming as a younger age at diagnosis of type 2 diabetes and a higher HbA1c are associated with higher mortality and risk of diabetes complications.[1,38] Indigenous Australians and Maori/Pacifica are overrepresented amongst youth with type 2 diabetes in Australia and New Zealand captured by ADDN, and have higher risk of developing type 2 diabetes in youth combined with poorer health outcomes and a lower life-expectancy.[7,20,39] These results support the recommendation for targeted screening for type 2 diabetes in overweight or obese youth from high risk populations (including Indigenous Australian and Maori) in the newly compiled guidelines for type 2 diabetes in youth from the Australasian Paediatric Endocrine Group.[18] Increasing data-capture by expanding the number of ADDN centres, particularly into areas with a higher Indigenous Australian population, will improve our understanding of type 2 diabetes in this high-risk group.

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Table 1: Clinical and demographic characteristics of children and adolescents with type 2 diabetes reported to the ADDN registry between 2012-2017

	All Centres	Australia	New Zealand
Number of children and adolescents	269	201	68
Sex (male)	113 (42%)	90 (45%)	23 (34%)
Age at diagnosis[†] (years)	13.7 [12.0-14.9]	13.9 [12.1-15.1]	12.8 [11.7-14.4]
Age at first visit (years)	14.3 [12.7-15.6]	14.4 [12.8-15.6]	14.1 [12.3-15.3]
Duration of diabetes (years)	0.2 [0.0-0.8]	0.1 [0.0-0.6]	0.3 [0.1-1.4]
HbA1c (%)	7.3 [6.0-9.2]	7.1 [6.0-9.2]	7.5 [6.1-9.2]
HbA1c (mmol/mol)	56 [42-77]	55 [42-78]	58 [43-77]
BMI-SDS	2.2 [1.8-2.5]	2.1 [1.7-2.5]	2.3 [2.0-2.6]
Overweight[‡]	46 (18%)	39 (20%)	7 (10%)
Obese[‡]	199 (77%)	141 (73%)	58 (87%)
Waist circumference percentile[§]	97 [93-99]	97 [93-99]	-
Waist/height ratio	0.61 [0.55-0.66]	0.61 [0.55-0.66]	-
Hypertension[¶]	63 (35%)	59 (35%)	4 (57%)
Raised LDL[#]	20 (29%)	17 (28%)	3 (38%)
Raised triglycerides[††]	39 (43%)	37 (44%)	2 (33%)

Data are n (%) or median [IQR]
[†] Where date of diagnosis was not reported (5 participants) the date of first visit was used as a surrogate for calculating age at diagnosis
[‡] Overweight and obesity defined according to the International Obesity Task Force cut-offs for age and sex
[§]Waist circumference percentiles based on data from the US National Health and Nutrition Survey. Waist circumference reported in 22/269 participants.
[¶] Hypertension defined as systolic and/or diastolic blood pressure >95th centile in age <13 years or >130/80mmHg in age ≥ 13 years
[#] Raised LDL cholesterol >3.36mmol/L (131mg/dL)
[††]Raised triglycerides defined as >1.7mmol/L (150mg/dL). Fasting status unknown in 22/39 participants with hypertriglyceridemia.

Table 2: Clinical characteristics of Indigenous Australian, New Zealand Maori/Pacifica and non-Indigenous children and adolescents with type 2 diabetes

	Non-Indigenous	Indigenous Australian	NZ Maori or Pacifica	P value
Number of children and adolescents	140 (52%)	56 (23%)	47 (19%)	
Age at diagnosis[†] (years)	14.1 [12.6-15.3]	13.3 [11.3-14.7]	13.1 [12.3-14.5]	0.005
Age <10yrs at diagnosis	4 (3%)	7 (13%)	4 (9%)	0.031
BMI-SDS	2.2 [1.7-2.5]	2.1 [1.5-2.4]	2.3 [2.0-2.6]	0.011
Overweight[‡]	28 (20%)	13 (26%)	2 (4%)	0.017
Obesity[‡]	100 (73%)	33 (65%)	43 (94%)	0.003
HbA1c (%)	6.7 [5.9-7.9]	9.4 [7.5-12.5]	7.8 [6.4-9.4]	0.000
HbA1c (mmol/mol)	50 [41-63]	79 [58-113]	62 [47-79]	0.000
HbA1c >6.5%[§]	71 (52%)	44 (85%)	34 (74%)	0.000
Hypertension[¶]	37 (35%)	17 (36%)	5 (56%)	0.456
Raised LDL[#]	10 (25%)	8 (40%)	2 (33%)	0.490
Raised triglycerides[††]	23 (43%)	13 (45%)	2 (50%)	0.949

Data are n (%) or median [IQR]

[†] Where date of diagnosis was not reported, the date of first visit was used as a surrogate for calculating age at diagnosis

[‡] Overweight and obesity defined according to the International Obesity Task Force cut-offs for age and sex

[§]Treatment target for HbA1c in youth with type 2 diabetes is ≤6.5% in Australasian Paediatric Endocrine Group (APEG) guidelines (awaiting publication)

[¶] Hypertension defined as systolic and/or diastolic blood pressure >95th centile in age <13 years or >130/80mmHg in age ≥ 13 years

[#] Raised LDL cholesterol >3.36mmol/L (131mg/dL)

[††]Raised triglycerides defined as >1.7mmol/L (150mg/dL). Fasting status unknown in 22 of 38 participants with hypertriglyceridemia.

Appendix 1:

Australasian Diabetes Data Network (ADDN) Study Group participants

Professor Peter Colman (Royal Melbourne Hospital, Melbourne)
Professor Maria Craig (The Children's Hospital at Westmead, Sydney)
Clinical Professor Tim Jones (Perth Children's Hospital, Perth)
Professor Richard Sinnott (eResearch, University of Melbourne)
Clinical Professor Geoff Ambler (The Children's Hospital at Westmead, Sydney)
Dr Helen Barrett (The Mater Private Hospital, Brisbane)
Professor Jenny Batch (Queensland Children's Hospital, Brisbane)
Dr Philip Bergman (Monash Children's Hospital, Melbourne)
Professor Fergus Cameron (Royal Children's Hospital, Melbourne)
A/Prof Louise Conwell (Queensland Children's Hospital, Brisbane)
A/Prof Andrew Cotterill (Queensland Children's Hospital, Brisbane)
Professor Jennifer Couper (Women's and Children's Hospital, Adelaide)
A/Prof Elizabeth Davis (Perth Children's Hospital, Perth)
Professor Kim Donaghue (The Children's Hospital at Westmead, Sydney)
Dr Jan Fairchild (Women's and Children's Hospital, Adelaide)
Dr Gerry Fegan (Fiona Stanley Hospital, Perth)
Dr Leonie Gray (Mater Medical Centre, Rockhampton)
A/Prof Shane Hamblin (Western Health, Melbourne)
Professor Paul Hofman (University of Auckland, New Zealand)
A/Prof Jane Holmes-Walker (Westmead Hospital, Sydney)
Dr Neville Howard (Royal North Shore Hospital, Sydney)
Dr Michelle Jack (Royal North Shore Hospital, Sydney)
Dr Steven James (University of the Sunshine Coast)
Dr Craig Jefferies (Starship Children's Hospital, New Zealand)
A/Prof Bruce R King (John Hunter Children's Hospital, Newcastle)
Dr Antony Lafferty (The Canberra Hospital, Canberra)
Dr Robert McCrossin (Gladstone Hospital, Gladstone)
Dr Mark Pascoe (Royal Hobart Hospital, Hobart)
Dr Alexia Peña (The University of Adelaide)
A/Prof Darrell Price (Pacific Private Clinic, Gold Coast)
A/Prof Christine Rodda (Monash Children's Hospital, Melbourne)

Dr Alan Sive (Townsville Hospital, Townsville)

Dr Carmel Smart (John Hunter Children's Hospital, Newcastle)

Dr Monique Stone (Royal Darwin Hospital, Darwin)

Dr Elaine Tham (Women's and Children's Hospital, Adelaide)

Dr Charles Verge (Sydney Children's Hospital, Sydney)

Professor Jerry Wales (Queensland Children's Hospital, Brisbane)

A/Prof Ben Wheeler (Dunedin School of Medicine, New Zealand)

Dr Judy Williams (Bundaberg Diabetes Centre, Bundaberg)

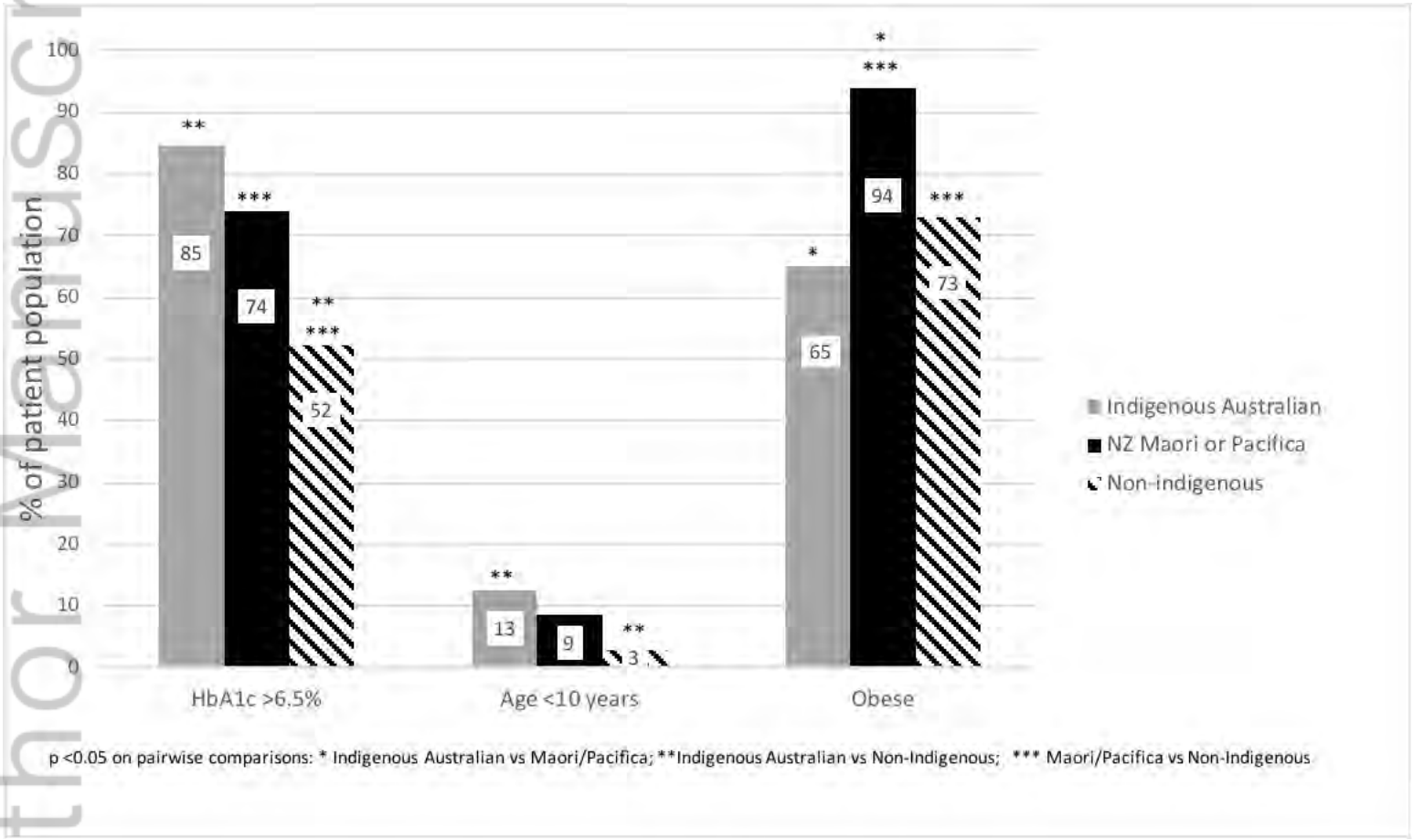
A/Prof Esko Wiltshire (Wellington Children's Hospital, New Zealand)

Dr Helen Woodhead (University of New South Wales, Sydney)

Dr Nick Woolfield (Caboolture Hospital, Caboolture)

Dr Anthony Zimmermann (Lyell McEwin Hospital, Elizabeth Vale)

Professor Sophia Zoungas (Monash Medical Centre, Melbourne)



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