

Ethnic differences in mortality and hospital admission rates between Māori, Pacific, and European New Zealanders with type 2 diabetes between 1994 and 2018: a retrospective, population-based, longitudinal cohort study

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Summary

Background Type 2 diabetes affects Indigenous and non-European populations disproportionately, including in New Zealand, where long-term temporal trends in cause-specific clinical outcomes between Māori, Pacific, and European people remain unclear. We aimed to compare the rates of mortality and hospital admission between Māori, Pacific, and European patients with type 2 diabetes in Auckland, New Zealand, over a period of 24 years.

Methods In this retrospective, population-based, longitudinal cohort study, we identified a cohort of patients (aged 35–84 years) with type 2 diabetes enrolled between Jan 1, 1994, and July 31, 2018, to the primary care audit programme, the Diabetes Care Support Service (DCSS) in Auckland, New Zealand. Patients with type 1 diabetes, prediabetes, and gestational diabetes were excluded. We linked data from the DCSS with national death registration, hospital admission, pharmaceutical claim, and socioeconomic status databases. Patients were followed up until death or July 31, 2018 (date of last enrolment to the DCSS). Incident clinical events (all-cause mortality, cardiovascular mortality, cancer mortality, cardiovascular hospital admission, cancer hospital admission, and end-stage renal disease hospital admission) were identified. Event rates were stratified by ethnic group, age group, sex, socioeconomic status, and time period (<1998, 1999–2013, 2004–08, 2009–13, and 2014–18). Incidence rate ratios (IRRs) and absolute risk differences were adjusted for sex, age, smoking status, obesity, socioeconomic status, and time period by use of age-period-cohort modelling.

Findings Between Jan 1, 1994, and July 31, 2018, 45 072 patients with type 2 diabetes (21 936 [48.7%] female; mean age 56.7 years [SD 13.8]) were enrolled in the DCSS and followed up for a median of 9.7 years (IQR 5.8–13.6). 16 755 (37.2%) were European, 7093 (15.7%) were Māori, and 12 044 (26.7%) were Pacific patients. Despite a similar temporal trend (decreasing mortality and increasing hospital admissions) across the three ethnic groups, Māori and Pacific patients had consistently higher hospital admission rates than European patients. Māori but not Pacific patients had higher adjusted IRRs for all-cause mortality (1.96 [95% CI 1.80–2.14]), cardiovascular mortality (1.93 [1.63–2.29]) and cancer mortality (1.64 [1.40–1.93]) rates compared with European patients.

Interpretation Compared with European patients, poorer health outcomes have persisted among Māori and Pacific people with type 2 diabetes for more than 20 years. New policies supporting prevention and more intensive management of type 2 diabetes are urgently needed. Research into the biological and societal mechanisms underlying these disparities, and the associated differences between Māori and Pacific patients is also needed.

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Introduction

Type 2 diabetes affects approximately 422 million people worldwide, including 240 000 people in New Zealand. Despite improvements in diabetes therapies over the past two decades, mortality remains high,¹ with disparities associated with ethnicity and socioeconomic status.² The cost of hospital admissions associated with type 2 diabetes is high; in the USA, this cost is predicted to reach more than \$622 billion per year by 2030.³ The economic costs of type 2 diabetes are expected to increase, with a projected increase in the prevalence of this condition of 54% by

2030.³ Consequently, attempts to reduce the substantial social, health, and economic burden of type 2 diabetes have become a top public health priority.⁴

The 2–4-times greater risk of type 2 diabetes among Māori and Pacific people in New Zealand was first reported in the 1960s;⁵ Māori and Pacific people also have an earlier age of onset and a 2–4-times higher risk of diabetes-related complications compared with New Zealanders of European descent. Biological differences (eg, body composition),⁵ health inequities,⁶ or both have been proposed as possible mechanisms

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Research in context

Evidence before this study

In New Zealand, there is an epidemic of type 2 diabetes leading to increasing health-care utilisation. Health disparities between Māori (Indigenous New Zealanders), Pacific, and European ethnic groups in New Zealand are well known. However, the extent of the disparities in mortality and hospital admission rates between Māori, Pacific, and European New Zealanders with type 2 diabetes in the 21st century is unknown. We searched PubMed using the search terms “Māori” and “type 2 diabetes” in different combinations with “surviv*”, “rate*”, “outcome*”, “mortality”, “death*”, “hospital*”, “prognos*”, and “trend*”. We searched for primary research published between Jan 1, 2000, and Dec 5, 2019, written in English, comparing outcome trends in type 2 diabetes between Indigenous and European patients in New Zealand. We found 152 studies, and reviewed the full text of relevant articles to assess their appropriateness for inclusion. Most studies addressed the general population and focused on short-term outcomes. We found no studies reporting cause-specific mortality or hospital admission trends over the last two decades among Māori, Pacific, and European patients with type 2 diabetes in New Zealand.

Added value of this study

The Diabetes Care Support Service (DCSS) was a long-running audit of diabetes management in primary care. By linking the DCSS with national death registration, national hospital admissions, national pharmaceutical claims, and national primary care databases to identify a cohort of patients with type 2 diabetes, we were able to evaluate long-term health disparities between Māori, Pacific, and European ethnic groups

in New Zealand between 1994 and 2018. Cause-specific mortality rates and incidence rates of hospital admissions were consistently higher in Māori patients than in European and Pacific patients with type 2 diabetes over the past 20 years. These health disparities persisted after adjusting for birth cohort and period effects, and for population confounders (age, sex, smoking status, obesity, socioeconomic status, and time periods). Most cause-specific mortality rates between Pacific and European patients with type 2 diabetes did not differ significantly over the same time period. Hospital admission rates were significantly higher in both Māori and Pacific patients than in European patients.

Implications of all the available evidence

Disparities in mortality and hospital admission rates persist between Māori, Pacific, and European patients with type 2 diabetes in New Zealand, and remain a substantial social, health, and economic burden. These health disparities for type 2 diabetes were not driven by population-level confounders, such as age, sex, smoking status, obesity, socioeconomic status, birth cohort effects, or time period effects. The perpetuation of these poor outcomes in patients with type 2 diabetes, particularly among Māori patients, over such a long period of time, calls for the introduction of new, more intensive, approaches for the early recognition and management of this condition, while broader strategies are needed to address social disparities and prevention. Differences in outcomes between Māori and Pacific patients with type 2 diabetes warrant further research into the underlying biological mechanisms, and the role of current or historical inequities.

underlying these disparities. Contemporary population-level data on emerging outcome trends among different ethnic groups of people with type 2 diabetes in New Zealand are not available, and cause-specific mortality and hospital admission rates in people with type 2 diabetes will enable demand projections. Such trends are also likely to inform our understanding of the global epidemiology of type 2 diabetes. We aimed to investigate whether ethnic differences in mortality and hospital admission rates among patients with type 2 diabetes in New Zealand have persisted. We also aimed to estimate the risk difference of these outcomes between ethnic groups using a novel age-period-cohort analysis.

Methods

Study design and population

We did a retrospective, population-based, longitudinal cohort study of Māori, Pacific, and European patients (aged 35–84 years) with type 2 diabetes enrolled between Jan 1, 1994, and July 31, 2018 to the Diabetes Care Support Service (DCSS). The DCSS, a primary care audit programme established in 1991, audits general practice diabetes

management in south, east, and west Auckland to improve standards of care.⁷ We linked data from the DCSS with national death registration, hospital admission, pharmaceutical claim, and socioeconomic status databases to identify a cohort of patients with type 2 diabetes in Auckland, New Zealand. All patients were included in the audit. Patients from other individual ethnic groups were excluded from the ethnic group-specific analyses due to insufficient numbers. The longitudinal, linked, anonymised DCSS database included data on patient demographics, risk factors, clinical measurements, diagnosed diabetes complications, and diabetes medications, all of which have been validated through enumeration assessment and internal quality control policies, with auditors regularly cross-checking data, and undertaking random and routine sampling or data entry checks, and active data management (eg, checking unusual numbers, ranking columns, and checking duplicates).^{7–9} Pharmaceutical claims data included all prescriptions issued for patients, and was used to cross-validate the prescription data in the DCSS. Only pharmaceutical claims data after 2006 were available for linkage. Historical claims before

2006 were not linked because National Health Index numbers were not universal until 2006. Data for all patients from their first DCSS enrolment date (Jan 1, 1994) up to July 31, 2018, (date of last enrolment to the DCSS) were included in the study.

The North Health Ethics Committee approved the DCSS for research purposes in 1992, and then as an ongoing audit in 1996 (92/006). Ethics review was waived by the New Zealand Health and Disability Ethics Committees on March 25, 2019. Anonymised data were used for this analysis. Signed consent to participate was provided by an authorised signatory for each general practice.

Procedures

Type 2 diabetes was defined by primary care record coding, with validation by trained diabetes auditors. Baseline characteristics included smoking, diabetes duration, body-mass index (BMI), blood pressure, glycated haemoglobin (HbA_{1c}), blood lipids, and antihypertensive, antidiabetes, statin, and antiplatelet or anticoagulant treatment (or both).

Patients were categorised into exposure groups by ethnicity (Māori, European, and Pacific); age (<35 years, 35–44 years, 45–54 years, 55–64 years, 65–74 years, 75–84 years and ≥85 years); sex; and socioeconomic status. Māori (Indigenous Polynesian) patients were defined as those with any Māori ancestry, and Pacific patients (93% Polynesian) were defined as those with any Pacific ancestry except Māori. As only six all-cause death events occurred in those aged <35 years, and the numbers of Māori (n=31) and Pacific patients (n=67) aged ≥85 years were low, analyses in these two strata were excluded.

The area deprivation indicator from the Department of Public Health, University of Otago (Otago, New Zealand), NZDep2013 was used to define socioeconomic status. NZDep2013 provides an Index of Multiple Deprivation (IMD) score for each New Zealand meshblock (Statistics New Zealand-defined geographical units, each of which included a median of approximately 81 people in 2013).¹⁰ NZDep2001, NZDep2006, and NZDep2013 scores were compared at a patient level to observe shifts in socioeconomic status over time (appendix pp 1–5). As consistent stability in socioeconomic status was observed using NZDep2013, this index was chosen for analyses. The NZDep2013 scale of deprivation ranges from 1 to 10, and divides New Zealand into tenths of the distribution of the first principal component scores. To maintain statistical power, the IMD was redefined by re-ranking NZDep2013 into 5 groups: IMD-1 (least deprived; NZDep2013 score 1–2), IMD-2 (NZDep2013 score 3–4), IMD-3 (NZDep2013 score 5–6), IMD-4 (NZDep2013 score 7–8), and IMD-5 (most deprived; NZDep2013 score 9–10).

Outcomes

The primary objective was to compare all-cause, cardiovascular, and cancer mortality rates, and the incidence

rates of hospital admission due to cardiovascular disease, cancer, and end-stage renal disease (ESRD) between Māori, Pacific, and European patients with type 2 diabetes. Sensitivity analyses were done by adjusting for confounders.

The linked national death registration database includes all deaths registered in New Zealand and was used to obtain the date and cause of death of patients in our cohort. The count of individual deaths was used to estimate mortality. Hospital admission was defined as all non-elective admissions occurring after, but not on, the date of enrolment to the DCSS. The linked national hospital admissions database, which includes all inpatient hospital admissions in New Zealand, provided the date and cause of hospital admission (defined as the primary cause of hospital admission). The overall number of hospital admissions (events) were counted for each patient, with follow-up until death or the end of the study (July 31, 2018). All clinical events were defined by the primary International Classification of Disease (ICD)-ninth edition and ICD-tenth edition (appendix p 6). Incident clinical events (all-cause mortality, cardiovascular mortality, cancer mortality, cardiovascular hospital admission, cancer hospital admission, and ESRD hospital admission) between Jan 1, 1994 and Dec 31, 2018, were identified.

Statistical analysis

Descriptive statistics are presented as numbers and proportions for dichotomous variables, and as mean (SD) or median (IQR) for continuous variables. Incident clinical event rates with 95% CIs are shown for the whole cohort, and stratified by ethnic group, age group, sex, socioeconomic status, and time period (<1998, 1999–2013, 2004–08, 2009–13, and 2014–18).

Māori and Pacific patients were compared with European patients by use of Poisson regression models (with 95% CIs), to estimate absolute risk differences and the incidence rate ratio (IRR) of clinical events, adjusting for population-level confounders overall, and by sex, age, smoking, obesity (defined as a BMI of ≥30 kg/m²), socioeconomic status, and time period.

We modelled age-period-cohort effects on clinical events from 1994 to 2018. Since the birth cohort is defined as the time period of the study minus the age of the patient, standard regression models to simultaneously estimate the additive effects of age, time period, and cohort effects cannot achieve a unique solution. Two approaches were therefore used to provide a robust check on the results of the age-period-cohort analysis.¹¹ The first approach to overcome dependency, was to estimate a variable (referred to as a drift variable) for the overall linear trend in clinical event rates that cannot be attributable uniquely to either time period or cohort effects. Derivation from linearity (referred to as curvature), which can be uniquely attributable to time period or cohort effects, and is not dependent on any model constraint, was then estimated as

See Online for appendix

	All patients (n=45 072)	European patients (n=16 755)	Māori patients (n=7093)	Pacific patients (n=12 044)
Age at enrolment, years	56.7 (13.8)	62.2 (13.2)	51.5 (12.6)	52.8 (12.8)
Age distribution, years				
<35	550 (1.2%)	75 (0.4%)	168 (2.4%)	231 (1.9%)
35–44	6351 (14.1%)	1228 (7.3%)	1509 (21.3%)	2226 (18.5%)
45–54	11736 (26.0%)	3135 (18.7%)	2336 (32.9%)	3858 (32.0%)
55–64	12372 (27.4%)	4577 (27.3%)	1958 (27.6%)	3330 (27.6%)
65–74	9087 (20.2%)	4526 (27.0%)	863 (12.2%)	1819 (15.1%)
75–84	4147 (9.2%)	2633 (15.7%)	228 (3.2%)	513 (4.3%)
≥85	829 (1.8%)	581 (3.5%)	31 (0.4%)	67 (0.6%)
Sex				
Female	21936 (48.7%)	7560 (45.1%)	3613 (50.9%)	6437 (53.5%)
Male	23136 (51.3%)	9195 (54.9%)	3480 (49.1%)	5607 (46.5%)
IMD group (NZDep13 scale)*				
IMD-1 (1 or 2)	5503 (12.2%)	3235 (19.3%)	277 (3.9%)	329 (2.7%)
IMD-2 (3 or 4)	5428 (12.0%)	2857 (17.1%)	515 (7.3%)	596 (4.9%)
IMD-3 (5 or 6)	5006 (11.1%)	2615 (15.6%)	718 (10.1%)	643 (5.3%)
IMD-4 (7 or 8)	10709 (23.8%)	4360 (26.0%)	1665 (23.5%)	2502 (20.8%)
IMD-5 (9 or 10)	18426 (40.9%)	3688 (22.0%)	3918 (55.2%)	7974 (66.2%)
Current smoker	6360 (14.1%)	1812 (10.8%)	2124 (30.0%)	1737 (14.4%)
Duration of having diabetes, years	4.8 (1.2)	4.7 (1.2)	5.0 (1.4)	4.9 (1.1)
Body-mass index, kg/m ²	32.5 (7.4)	31.2 (6.5)	35.9 (7.8)	35.0 (7.4)
Obesity	26867 (59.6%)	8960 (53.5%)	5596 (78.9%)	8946 (74.3%)
Blood pressure, mm Hg				
Systolic	135 (18)	137 (18)	135 (19)	132 (18)
Diastolic	81 (11)	80 (10)	83 (12)	82 (11)
HbA _{1c}	7.6% (4.0)	7.1% (3.7)	8.1% (4.2)	8.3% (4.2)
HbA _{1c} , mmol/mol	60 (20)	54 (17)	65 (22)	67 (22)
Total cholesterol, mmol/L	5.1 (1.2)	5.0 (1.2)	5.2 (1.2)	5.0 (1.2)
Triglyceride, mmol/L	2.2 (1.5)	2.2 (1.4)	2.6 (1.6)	2.1 (1.6)
LDL, mmol/L	2.5 (0.9)	2.5 (0.9)	2.6 (1.0)	2.6 (1.0)
HDL, mmol/L	1.2 (0.3)	1.2 (0.3)	1.1 (0.3)	1.2 (0.3)
Antihypertensive treatment	30165 (66.9%)	11687 (70.0%)	5060 (71.3%)	8070 (67.0%)
Statin treatment	24676 (54.8%)	9417 (56.2%)	4197 (59.2%)	6380 (53.0%)
Antidiabetes treatment†				
Oral antidiabetes drug and insulin	7130 (15.8%)	2510 (15.0%)	1398 (19.7%)	2182 (18.1%)
Oral antidiabetes drug only	26968 (59.8%)	9337 (55.7%)	4180 (58.9%)	7569 (62.8%)
Insulin only	4922 (10.9%)	1776 (10.9%)	994 (14.0%)	1237 (10.3%)
Antiplatelet or anticoagulant treatment	1072 (2.4%)	574 (3.4%)	198 (2.8%)	182 (1.5%)

Data are n (%) or mean (SD). IMD=Index of Multiple Deprivation. HbA_{1c}=glycated haemoglobin. *IMD, defined by the NZDep13 Index of Deprivation, was used to divide patients into groups ranging from IMD-1 (least deprived) to IMD-5 (most deprived). †Neither GLP-1 receptor agonists nor SGLT2 inhibitors are available in New Zealand, despite evidence to show that they improve outcomes in patients with type 2 diabetes.

Table 1: Characteristics of study participants with type 2 diabetes at enrolment to the Diabetes Care Support Service

time period or cohort effects. We estimated clinical event rates using parametric smooth functions based on natural splines with five knots for age, time period, and cohort variable to detect non-linear effects.

To address the effect of population-level confounders as a sensitivity analysis, age-period-cohort analyses were re-run by sex, socioeconomic status, smoking status, and obesity for each clinical event. All statistical analyses were done by use of Stata MP 15.1.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data and had final responsibility for the decision to submit for publication.

Results

45 072 patients with type 2 diabetes were enrolled in the DCSS between Jan 1, 1994, and July 31, 2018. 21 936 (48.7%) were female and the mean age was 56.7 years (SD 13.8). Patients were followed up for a median of 9.7 years (IQR 5.8–13.6). The characteristics of the 16 755 (37.2%) European, 7093 (15.7%) Māori, and 12 044 (26.7%) Pacific patients are shown in table 1. Median follow-up was 9.5 years (IQR 5.3–13.4) for European patients, 9.9 years (5.9–14.0) for Māori patients, and 9.8 years (5.9–14.2) for Pacific patients. 9180 (20.4%) patients from Africa, south Asia, east Asia, and the Middle East were excluded from ethnicity-specific analyses. Compared with Māori and Pacific patients, European patients were older, had the lowest deprivation scores, included fewer women and fewer current smokers, and had the lowest BMI, HbA_{1c}, and diastolic blood pressure, but the highest systolic blood pressure. There were no differences in diabetes duration or cholesterol concentrations between the three ethnic groups. Antihypertensive and statin therapy use was lowest among Pacific patients, and insulin therapy was lowest among European patients. Māori and Pacific patients were similar in terms of age, sex, and BMI. Māori patients had the highest proportion of current smokers, even after adjusting for socioeconomic status. Compared with Māori and European ethnic groups, the Pacific group had the highest proportion of patients in the most deprived category, but systolic and diastolic blood pressure was lower and HbA_{1c} was higher.

The overall crude all-cause mortality rate in all patients increased from 12.62 deaths per 1000 person-years (95% CI 11.05–14.37) in 1994–98, to 19.35 deaths per 1000 person-years (18.00–20.77) in 2004–08, before decreasing to 16.90 deaths per 1000 person-years (15.85–17.99) in 2009–13, and 9.90 deaths per 1000 person-years (9.14–10.71) in 2014–18 (table 2). Although the trend was similar across the three ethnic groups by sex, age group, socioeconomic status (appendix pp 7–8), and smoking and obesity status (appendix p 15), the overall all-cause mortality rate was highest among Māori patients and lowest among Pacific patients at each time period, both overall and within subgroups.

	All-cause mortality	Cardiovascular mortality	Cancer mortality	Cardiovascular hospital admission	Cancer hospital admission	End-stage renal disease hospital admission
All patients						
<1998	12.62 (11.05–14.37)	3.38 (2.59–4.34)	2.29 (1.65–3.10)	24.94 (24.38–25.50)	13.22 (12.40–14.09)	1.11 (0.81–1.49)
1999–2003	19.35 (18.00–20.77)	5.88 (5.16–6.69)	5.28 (4.59–6.04)	51.16 (50.61–51.72)	36.82 (35.90–37.76)	17.83 (16.97–18.72)
2004–08	17.40 (16.39–18.45)	4.02 (3.55–4.54)	5.41 (4.86–6.01)	76.93 (76.28–77.59)	66.80 (65.75–67.86)	27.19 (26.28–28.14)
2009–13	16.90 (15.85–17.99)	4.12 (3.62–4.67)	5.22 (4.65–5.84)	131.63 (130.40–132.86)	119.94 (118.16–121.74)	42.40 (41.06–43.78)
2014–18	9.90 (9.14–10.71)	2.00 (1.67–2.37)	3.46 (3.02–3.95)	264.87 (262.07–267.69)	210.20 (207.13–213.30)	75.34 (73.36–77.36)
European patients						
<1998	12.32 (10.15–14.83)	3.83 (2.67–5.33)	1.86 (1.08–2.98)	26.86 (26.05–27.69)	11.97 (10.95–13.07)	0.87 (0.49–1.44)
1999–2003	20.83 (18.80–23.02)	7.00 (5.85–8.31)	6.03 (4.96–7.25)	55.28 (54.43–56.14)	36.78 (35.56–38.04)	12.21 (11.11–13.39)
2004–08	19.35 (17.83–20.97)	4.98 (4.22–5.82)	6.43 (5.57–7.38)	83.49 (82.48–84.50)	67.56 (66.18–68.97)	21.86 (20.64–23.14)
2009–13	18.27 (16.71–19.94)	4.63 (3.87–5.50)	5.94 (5.07–6.91)	128.99 (127.16–130.84)	117.90 (115.57–120.26)	37.38 (35.52–39.30)
2014–18	11.33 (10.11–12.65)	2.10 (1.60–2.70)	4.96 (4.18–5.85)	266.88 (262.50–271.30)	253.37 (248.62–258.20)	71.35 (68.45–74.35)
Māori patients						
<1998	15.17 (11.02–20.36)	2.74 (1.18–5.39)	3.76 (1.88–6.72)	25.57 (24.31–26.88)	19.02 (16.64–21.63)	1.34 (0.67–2.39)
1999–2003	25.08 (21.32–29.31)	6.90 (5.02–9.27)	5.80 (4.09–8.00)	50.07 (48.86–51.31)	40.18 (37.82–42.65)	27.21 (24.93–29.66)
2004–08	22.48 (19.68–25.58)	5.45 (4.13–7.06)	5.93 (4.55–7.60)	76.16 (74.74–77.59)	70.50 (67.84–73.24)	39.68 (37.20–42.27)
2009–13	25.18 (22.08–28.60)	5.92 (4.48–7.67)	7.59 (5.95–9.55)	135.23 (132.61–137.90)	138.89 (134.26–143.64)	57.92 (54.36–61.66)
2014–18	13.08 (10.92–15.54)	3.07 (2.09–4.36)	3.88 (2.76–5.30)	290.98 (284.30–297.78)	209.36 (201.77–217.17)	102.13 (96.49–108.02)
Pacific patients						
<1998	11.15 (7.97–15.18)	1.94 (0.78–3.99)	2.22 (0.96–4.36)	19.54 (18.41–20.72)	14.94 (12.79–17.36)	1.36 (0.72–2.32)
1999–2003	14.58 (12.19–17.30)	3.97 (2.78–5.49)	3.64 (2.50–5.11)	42.26 (41.18–43.36)	32.90 (30.89–35.00)	21.22 (19.39–23.18)
2004–08	12.34 (10.52–14.39)	1.72 (1.09–2.58)	3.30 (2.40–4.43)	65.97 (64.68–67.27)	56.79 (54.41–59.25)	29.61 (27.64–31.67)
2009–13	14.49 (12.34–16.91)	3.01 (2.09–4.21)	3.46 (2.46–4.73)	131.99 (129.35–134.68)	121.95 (117.22–126.82)	51.58 (48.34–54.98)
2014–18	8.19 (6.82–9.76)	1.74 (1.15–2.53)	1.55 (0.99–2.31)	264.40 (258.93–269.95)	154.33 (148.57–160.25)	82.90 (78.72–87.24)

All data are incidence rates per 1000 person-years (95% CI).

Table 2: Incidence rate (per 1000 person-years) of clinical outcomes, stratified by ethnicity and time period

Compared with European patients, the adjusted overall IRR for all-cause mortality was 1.96 (95% CI 1.80–2.14) in Māori patients, with an adjusted absolute risk difference of 22.85 per 1000 person-years (95% CI 19.03–25.77; table 3). Similar ethnic differences were also found by sex, age, smoking, obesity, socioeconomic status, and time period (appendix pp 19–20). However, the adjusted IRRs for all-cause mortality in Pacific patients compared with European patients indicated that this outcome did not differ significantly between the two ethnic groups (appendix pp 19–20). The adjusted absolute risk difference for all-cause mortality in Pacific patients compared with European patients was marginally higher in some subgroup analyses (eg, in men), but was significantly lower in the 2014–18 cohort (appendix pp 21–22).

The overall crude cardiovascular mortality rate decreased from 3.38 deaths per 1000 person-years (95% CI 2.59–4.34) in 1994–98 to 2.00 deaths per 1000 person-years (1.67–2.37) in 2014–18 (table 2). Although the pattern fluctuated between time periods, cardiovascular mortality rate patterns were similar across the three ethnic groups, irrespective of sex, age, socioeconomic status, smoking status, and obesity (appendix pp 8–9, 15–16). The

cardiovascular mortality rate was highest among Māori patients and lowest among Pacific patients in each time period and by sex, age group, smoking status, and obesity (appendix pp 8–9, 15–16).

Compared with European patients, the adjusted IRR for cardiovascular mortality in Māori patients was 1.93 (95% CI 1.63–2.29), with an absolute risk difference of 6.97 deaths per 1000 person-years (95% CI 5.14–8.80; table 3). These ethnic differences were also observed when patients were grouped by sex, age, smoking status, obesity, socioeconomic status, and time period (appendix pp 19–20). However, the adjusted IRRs and absolute risk differences for cardiovascular mortality in Pacific patients indicated that the incidence of this outcome was not significantly different to that in European patients (appendix pp 21–22).

The overall cancer mortality rate in the whole cohort increased from 2.29 deaths per 1000 person-years (95% CI 1.65–3.10) in 1994–98 to 5.41 deaths per 1000 person-years (4.86–6.01) in 2004–08, before decreasing to 3.46 deaths per 1000 person-years (3.02–3.95) in 2014–18 (table 2). Although the pattern was similar across ethnic groups by sex, age (appendix pp 9–10), socioeconomic status, smoking, and obesity (appendix pp 16–17), the overall

	Māori patients		Pacific patients	
	Adjusted IRR (95% CI)	Adjusted absolute risk difference per 1000 person-years (95% CI)	Adjusted IRR (95% CI)	Adjusted absolute risk difference per 1000 person-years (95% CI)
All-cause mortality	1.96 (1.80 to 2.14)	22.85 (19.93 to 25.77)	1.04 (0.95 to 1.15)	1.00 (-0.12 to 2.13)
Cardiovascular mortality	1.93 (1.63 to 2.29)	6.97 (5.14 to 8.80)	0.87 (0.71 to 1.05)	-1.00 (-1.56 to -0.44)
Cancer mortality	1.64 (1.40 to 1.93)	3.08 (2.25 to 3.92)	0.75 (0.62 to 0.91)	-1.20 (-1.45 to -0.96)
Cardiovascular hospital admission	1.26 (1.25 to 1.28)	42.70 (41.32 to 44.08)	1.09 (1.07 to 1.10)	14.45 (13.70 to 15.20)
Cancer hospital admission	1.31 (1.28 to 1.34)	44.76 (42.37 to 47.15)	1.03 (1.00 to 1.06)	4.05 (2.81 to 5.30)
End-stage renal disease hospital admission	2.05 (1.96 to 2.14)	31.44 (29.67 to 33.21)	1.59 (1.52 to 1.67)	17.29 (16.12 to 18.46)

IRRs and absolute risk differences adjusted for sex, age group, Index of Multiple Deprivation, smoking status, obesity status, and time period. European ethnicity was used as the reference group. IRR=incidence rate ratio.

Table 3: Adjusted IRRs and absolute risk differences for clinical outcomes in Māori and Pacific patients with type 2 diabetes compared with European patients with type 2 diabetes in New Zealand

cancer mortality rate was highest among Māori patients and lowest among Pacific patients in each time period.

Compared with European patients, the adjusted overall IRR for cancer mortality was 1.64 (95% CI 1.40–1.93) in Māori patients, with an absolute risk difference of 3.08 per 1000 person-years (2.25–3.92; table 3). These ethnic differences were also observed when patients were grouped by sex, age, socioeconomic status, smoking status, obesity and time period (appendix pp 19–20). However, the adjusted IRRs and absolute risk differences for cancer mortality in Pacific people indicated that the incidence of this outcome was similar to that in European patients (appendix pp 21–22).

Overall, ethnic and age-specific crude incidence rates of cardiovascular hospital admissions increased substantially over time (table 2 and appendix pp 10–11, 17–18). The incidence rates of this outcome were higher among European patients than Māori patients in 1999–2008, whereas the opposite trend was observed from 2009 onwards. Compared with European patients, the adjusted overall IRR for cardiovascular hospital admission was higher among Māori patients (1.26 [95% CI 1.25–1.28]) and Pacific patients (1.09 [1.07–1.10]; table 3) across the different time periods (appendix pp 19–20). These ethnic differences were largely present over the whole time period (1998–2018) when patients were grouped by sex, socioeconomic status, obesity, and smoking status (appendix pp 19–22). Even though the adjusted overall absolute risk difference reflected the adjusted overall IRR for cardiovascular hospital admission, the absolute risk differences in Pacific patients were generally significantly lower than in Māori patients in all subgroup analyses (appendix pp 21–22).

The overall, ethnic and age-specific incidence rates of cancer hospital admissions increased substantially over time (table 2 and appendix pp 11–12, 17). The adjusted IRRs of this outcome were higher among Māori and Pacific patients (table 3 and appendix pp 19–20); however, Māori patients had a greater adjusted absolute risk difference for cancer hospital admission than European

and Pacific patients overall, and when patients were grouped by sex, age, socioeconomic status, smoking status, obesity, and time period (table 3 and appendix pp 21–22).

The overall, ethnic, sex, and age-specific incidence rates of ESRD hospital admission increased substantially over time, with low incidence rates of this outcome before 1998 (table 2 and appendix pp 13–14, 18). When compared with European patients, the incidence rates of ESRD hospital admission were generally highest among Māori patients (IRR 2.05 [95% CI 1.96–2.14]; absolute risk difference 31.44 per 1000 person-years [95% CI 29.67–33.21]), followed by Pacific patients (1.59 [1.52–1.67]; 17.29 per 1000 person-years [16.12–18.46]; table 3). These ethnic patterns were observed from 1999 onwards (appendix pp 19–20). Additionally, the incidence rates of ESRD hospital admission were higher in European patients with the highest deprivation scores than in those with the lowest deprivation scores; however, the incidence rates of this outcome were higher in Māori and Pacific patients overall than in the least deprived European patients (appendix pp 21–22).

Adjusted all-cause, cardiovascular, and cancer mortality rates, and incidence rates of cardiovascular, cancer and ESRD hospital admissions all increased with age (figure). The adjusted incidence rates of all six outcomes were highest among Māori patients aged older than 45 years, even after adjusting for birth cohort and period effects. Pacific patients generally had similar adjusted mortality rates as European patients, but had higher adjusted incidence rates of cardiovascular (45–80 years), cancer (60–75 years), and ESRD (≥ 45 years) hospital admissions. The incidence rates of ESRD hospital admissions in European patients showed a U-shaped association, and among those younger than 40 years, the incidence rates of ESRD hospital admissions were significantly higher in European patients than in Pacific patients. There was no significant birth cohort effect (IRR with the birth year of 1960 as a reference in each ethnic group), after adjusting for age and time period effects in each ethnic group.

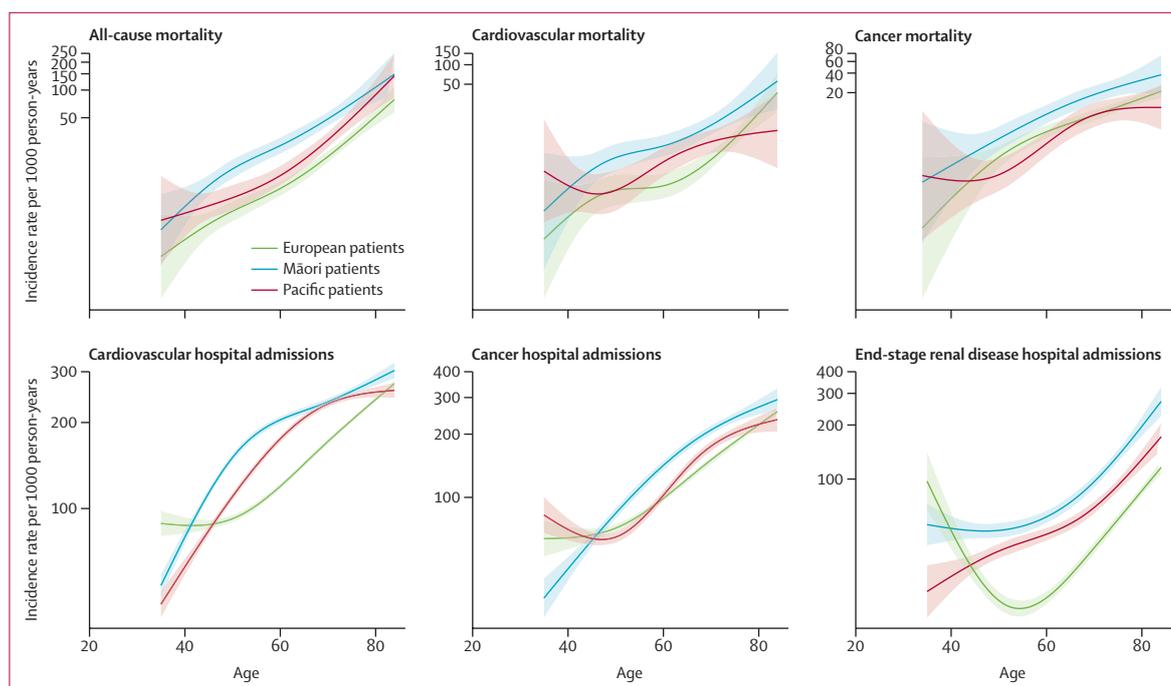


Figure: Adjusted incidence rate (per 1000 person-years) of clinical outcomes in a New Zealand population with diabetes enrolled to the Diabetes Care Support Service between 1994 and 2018, by Māori, Pacific, and European ethnicity. Patients were aged 35–84 years. Period and birth cohort effects were adjusted by use of age-period-cohort models. Solid lines indicate point estimations and shaded areas indicate the 95% CIs.

The adjusted time period effect (IRR with the calendar year of 2013 as reference in each ethnic group) was similar across the three ethnic groups, as the IRRs for all-cause, cardiovascular, and cancer mortality first increased in the 1990s, reached a plateau in the 2000s, and then significantly decreased after 2013 (appendix pp 23–58). A continuously increasing trend in adjusted IRR for cardiovascular, cancer, and ESRD hospital admissions was observed over the whole study period (appendix pp 59–94). Similar age-period-cohort estimations for all outcomes were observed when patients were grouped by sex, socioeconomic status, smoking status, and obesity according to time of enrolment (appendix pp 23–94).

Discussion

To our knowledge, this study is the first to compare all-cause, cardiovascular, and cancer mortality, and cardiovascular, cancer, and ESRD hospital admissions among Māori, Pacific, and European patients with type 2 diabetes in New Zealand over 25 years, showing that disparities remain as marked now as they did almost 25 years ago. The results show that Māori patients had worse outcomes than European New Zealander patients for all clinical outcomes measured. This difference was present after adjusting for age, sex, socioeconomic status, obesity, and smoking status. Although health disparities in cancer mortality and hospital admissions improved between 2014 and 2018, cardiovascular and ESRD outcomes have been consistently worse in Māori patients

than in European patients for over 20 years. The incidence rates of ESRD and cardiovascular hospital admissions in Pacific patients were also higher than in European patients, although incidence rates of cancer-related outcomes were lower, and rates of all-cause and cardiovascular mortality were similar. These results remained consistent when age-period-cohort models were used to estimate the incidence rates of health outcomes after adjusting for birth cohort and time period effects.

Higher IRRs for all-cause mortality (1.79–2.54), cardiovascular mortality (1.75–3.38), and cancer mortality (1.26–1.78) among Māori patients compared with the New Zealand general population between 1981 and 2011 have been shown.¹² In a cohort study of patients with type 2 diabetes in South Auckland, standardised mortality ratios in European patients were 2.78 (95% CI 2.19–3.48), 6.55 (4.61–9.03) in Māori patients, and 4.49 (3.07–6.34) in Pacific patients between 1991 and 1996, although the sample size was small and data were not adjusted for socioeconomic status.¹³ Data from a national primary health-care diabetes annual review programme (2000–05) found that, compared with European patients, the adjusted hazard ratios for the first cardiovascular event were 1.30 (1.19–1.41) in Māori patients and 1.04 (0.95–1.13) in Pacific patients.¹³ Based on national prevalence data,¹⁴ these figures suggest that the annual number of extra deaths in Māori patients with type 2 diabetes in New Zealand was 12 298 due to any cause, 4481 due to cardiovascular disease, and 2023 due to cancer.

These findings raise two key questions. The first is about the effectiveness of diabetes-related policy for addressing health disparities and inequities for Māori and Pacific people over this time period. The second, more fundamental question, is why such substantial differences exist in all six outcomes between two Polynesian populations: Indigenous Māori and non-Indigenous Pacific peoples.

Previous studies have attributed health disparities between Māori and European New Zealanders to several factors, including socioeconomic status,¹⁵ smoking,¹⁶ and obesity.¹⁷ However, the poorer outcomes in Māori patients compared with European patients in our study were independent of these variables. Health inequities through, for example, reduced access to care have also been suggested as a possible cause of health disparities;^{18,19} but we have previously shown that Māori (and Pacific) patients in this cohort had more diabetes consultations and similar or more screening tests than European patients.⁸ However, these consultations and screening tests could have been insufficient for their needs, and do not reflect, for example, health literacy or the cultural safety of the health care provided. Even though the prescription of statins, anti-hypertensive, and antihyperglycaemic drugs remained higher in Māori (and Pacific) patients than European patients, diastolic blood pressure, LDL cholesterol, and HbA_{1c} were, on average, higher, which could have contributed to some of the excess risk. It has also been suggested that Māori patients are diagnosed later than European patients. However, in a study in the neighbouring Waikato district, the prevalence of retinopathy at diagnosis was low,²⁰ as was the prevalence of undiagnosed diabetes,²¹ suggesting that the delay between development and diagnosis of type 2 diabetes was small.

Pacific people arrived in New Zealand approximately 1000–1200 years after Māori, and they have a similar genetic and cultural ancestry. Compared with Māori patients, Pacific patients in our study had the same or worse socioeconomic status, access to health care, and metabolic control, and a higher proportion of Pacific patients had obesity compared with Māori patients. Despite these disadvantages, Pacific patients had largely similar mortality rates to European New Zealander patients after adjustment for sex, age group, IMD, smoking status, obesity status, and time period. This finding suggests that the worse outcomes among Māori patients are for reasons beyond these confounding factors. We speculate that there could be several possible reasons for this observation. Firstly, the adjustment measures are likely to be imperfect. NZDep13 as a measure of deprivation is thought to only reflect 50–75% of the actual socioeconomic status.¹⁰ Even though our comparison of the three NZDep measures indicated consistent stability in socioeconomic status in most patients, there is no annual measure of socioeconomic status, and a shifting socioeconomic status in a small number of patients might have led to a degree of information bias in some patients.

Secondly, the proportion of patients reported as current smokers, which was higher in Māori patients than in European patients, could have been under-reported and under-recorded, and does not include smoking severity (eg, smoking pack-years). Finally, even though a higher BMI criterion for obesity is recommended for Polynesian people,²² we used standard criteria, which is likely to attenuate the differences rather than enhance them; the effect would be expected to be similar among Māori and Pacific people.

One important difference between Pacific and Māori Polynesian populations is that a European heritage could contribute to the increased risk of some complications, without necessarily attenuating others (eg, ESRD); a factor that warrants further research. For example, European people have a higher frequency of a range of genes associated with cancer than several non-European ethnic groups.²³ Whether these genes contributed to the increased mortality rates observed in Māori patients in our study is unclear. The excess risk of ESRD among Polynesian people appears to be a common feature between Māori and Pacific people,²⁴ and was associated with a family history of renal disease, but not diabetes, among Polynesian people.²⁵ Māori people could also be at greater risk of ESRD than other Polynesians because of their unique status as those who survived (or were selected for) the long oceanic journey to New Zealand.²⁶ A further mechanism is that Māori people have been affected by the colonisation process.⁶ This is a complex subject requiring further research that cannot be ignored.

Our study has several strengths, including being the largest study of a multi-ethnic cohort of patients with type 2 diabetes in New Zealand to report key diabetes-related health outcomes over 25 years. The cohort included all patients from participating general practices. By linking large, nationally representative databases, we were able to follow up patients to ascertain all incident health outcomes, including cause-specific mortality and hospital admissions. All health outcomes used in this study were based on the linkage of specific registration datasets, which provided good validation of outcomes. The accuracy of clinical recording and diagnoses in this study have been validated for a range of comorbidities, and we also used primary ICD codes for cause-specific outcomes, which have high precision.²⁷ The study limitations include heterogeneity in the Pacific, Māori, and European populations, and the national representativeness of the population and of the participating general practices. However, the population covered by the DCSS includes a substantial proportion of Pacific and Māori populations in New Zealand, and possibly European people at higher risk of diabetes complications due to their lower socioeconomic status overall. The high incidence rate of ESRD hospital admissions among younger (ie, aged <40 years) European patients suggests that people with type 1 diabetes were also included in the cohort. Misdiagnosis of type 1 diabetes as type 2 diabetes is a well known issue in primary care,²⁸

although this would probably have attenuated the observed differences between ethnic groups.

In conclusion, population-level ethnic disparities in type 2 diabetes outcomes are a major and persistent public health challenge in New Zealand. Over the past two decades, poor outcomes have persisted among Māori and Pacific people with type 2 diabetes after adjusting for age, sex, smoking, obesity, socioeconomic status, time period, and birth cohort effects. The perpetuation of these poorer outcomes among Māori and Pacific patients with type 2 diabetes compared with European patients over such a long period of time calls for the introduction of new, more intensive approaches to prevention, early recognition, and management of type 2 diabetes, while broader strategies are needed to address social disparities. Research into the biological and societal mechanisms underlying these disparities, and the differences in outcomes between Māori and Pacific patients with type 2 diabetes is needed.

Contributors

DY, ZZ, and DS contributed to the design and conduct of the study during all phases. DY, YC, ZZ, and DS contributed to the processing and the statistical handling of the data to generate the results. All authors contributed to discussions on the interpretation of the results and their appropriate presentation. DS contributed to patient and public input at all stages of the study. DS initiated the project, and DS, KP, and JB secured the funding. All authors contributed to the first draft of the manuscript, and commented on the final draft of the manuscript.

Declaration of interests

We declare no competing interests.

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