Clinical Epidemiology

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ORIGINAL RESEARCH

Adverse Clinical Outcomes Attributable to Socioeconomic and Ethnic Disparities Among People with Type 2 Diabetes in New Zealand Between 1994–2018: A Multiple Linked Cohort Study

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Purpose: The study aimed to examine the separate population-level contributions of the ethnic and socioeconomic disparities among people with type 2 diabetes mellitus (T2DM) and residence in New Zealand (NZ).

Patients and Methods: A prospective cohort enrolled T2DM patients from 01/01/1994 into the Diabetes Care Support Service, a primary care audit program in Auckland, NZ. The cohort was linked to national registry databases (socioeconomic status, pharmaceutical claim, hospitalization, and death registration). Each cohort member was followed up till death or the study end time (31/12/2019), whichever came first. Incident clinical events (stroke, myocardial infarction (MI), heart failure (HF), end-stage renal disease (ESRD), and premature mortality (PM)) were used as outcomes. The attributable fractions (AFs) were estimated for the whole population and for specific population with NZ Europeans (NZE) and/or least deprived population as reference, both unadjusted and with adjustment for covariables by Cox Regression models.

Results: Among 36,267 patients, adjusted population AFs indicated 6.6(−30.8–33.3)% of PM, 17.1(5.8–27.0)% of MI, 35.3(22.6–46.0)% of stroke, 14.3(3.2–24.2)% of HF, and 15.9(6.7–24.2)% of ESRD could be attributed to deprivation; while 14.3(3.3–25.4)% of PM, −3.3(−8.3–1.5)% of MI, −0.5(−6.7–5.3)% of stroke, 4.7(0.3–8.8)% of HF, 13.3(9.9–16.6)% of ESRD could be attributed to ethnicity. Deprivation contributed a significant AF to stroke, while ethnicity was important for ESRD. Gradient of AF for deprivation indicated NZE and Asians were most affected by deprivation across outcomes. Conversely, Māori, with the highest AFs for ethnicity of PM and ESRD, were unaffected by deprivation. At same deprivations, the AFs of MI and stroke were greatest among NZE compared with other ethnic groups; the AF of ESRD was greatest among Māori and Pasifika.

Conclusion: Both socioeconomic deprivation and ethnicity are strongly associated with outcomes in patients with T2DM in NZ, although the extent of the deprivation gradient is greatest among NZE and Asians, and least among Māori.

Keywords: stroke, myocardial infarction, heart failure, end-stage renal disease, premature mortality, Māori

Introduction

In New Zealand (NZ), as with many developed countries, socioeconomic deprivation is associated with increased rates of mortality and cardiovascular disease (CVD).[1–3] This pattern is also observed among patients with type 2 diabetes mellitus

Received: 10 January 2023
Accepted: 17 April 2023
Published: 1 May 2023

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(T2DM) in some specific ethnic groups.[4] However, the relationship between socio-economic deprivation and specific adverse clinical outcomes such as stroke, myocardial infarction (MI), heart failure (HF), end-stage renal disease (ESRD), and premature mortality (PM), among at-risk (i.e. with potential for de novo development of outcomes) patients with T2DM remains unclear. Moreover, few studies have addressed how socio-economic deprivation and ethnicity interact in relation to diabetes complications.

Reducing ethnic and socioeconomic deprivation related disparities in clinical outcomes in both the general population and those with T2DM is an important goal behind several health-care policies in NZ.[5] However, most efforts to enhance adverse clinical events and to lighten disparities are barricaded by a lack of information about how socioeconomic disparities relate to ethnicity and which specific populations are affected the most. Accurate measures are essential to communicate the magnitude of disparities in adverse clinical events to clinicians, and health policymakers. Further, clear implementation plans are needed that include both identifying and targeting interventions, as well as reporting measures and outcomes to monitor the intended gains.

The study aimed to examine the separate population-level contributions of socioeconomic deprivation and/or ethnic disparities to PM, MI, stroke, HF, and ESRD among at-risk patients with T2DM in NZ between 1994 and 2019.

Materials and Methods

Study Design and Data Sources

A prospective cohort was extracted from a primary care audit program dataset established in 1991, the Diabetes Care Support Service (DCSS). After a run-in time (1 January 1991 to 31 December 1993), the DCSS started to audit diabetes and the relevant management from general practices in West, East, and South Auckland, NZ, and ended on 31 July 2018.[6] The DCSS database was linked with national databases incorporating socioeconomic status, pharmaceutical claim death registration, and hospital admission to form the study cohort incorporating T2DM patients aged 18 years and over in Auckland (Supplemental Figure 1). The final cohort incorporated anonymised data on demographics, lifestyle information (such as the status of smoking), anthropometric measurements (blood pressure (BP) and body mass index (BMI)), diabetes routine clinical measurements (duration of diabetes, hemoglobin A1c (HbA1c), and lipid profiles), and routine medications prescribed to diabetes patients (anti-diabetes, statin, antihypertensive, antiplatelet, anticoagulant, and/or antiplatelet treatment). The data used in the study were validated through internal quality control policies and enumeration assessment with regular cross-checks by DCSS auditors, routine and random sampling and checking of data entry, and active data management (e.g. queries, checking unusual figures, duplicate checking, and ranking of columns). Data extracted from the national pharmaceutical claim database included every prescription issued to patients and was cross validated with the DCSS recorded prescription data. The national pharmaceutical claim database was only available after 2006 as the National Health Index Numbers were not universal before 2006.

The North Health Ethics Committee approved the DCSS for research purposes in 1992 and then as an ongoing audit in 1996.

The NZ Health and Disability Ethics Committee waived ethic review as the DCSS study only used anonymied data for analysis. Written informed consent to attend the DCSS was provided by the authorized signatory for every general practice. The NZ Ministry of Health granted access to the DCSS and the linked databases for this study.

Study Population

Patients with T2DM and aged 18 years and over were included. The enrolment date was defined as patients' entry dates to the DCSS database. Patients were excluded if they had outcomes (except premature mortality) before the enrolment date or within 12 months of enrolment to avoid potential information bias.

Exposure

Socioeconomic Deprivation

The NZDep2013 Index of Deprivation[7] as a neighborhood /area deprivation measurement was used to define patients' socioeconomic status. The NZDep2013 compiles an Index of Multiple Deprivation (IMD) score for every NZ meshblock (geographic unit/neighborhood covering a median of 81 people). The NZDep2013 score indicates scales of deprivation and ranges from 1 to 10, with 1 indicating least deprivation and 10 indicating most deprivation. To sustain statistical power for the current study, the IMD was redefined by regrouping the NZDep2013 into five categories: IMD-1 (least deprivation: 1–2 of NZDep2013 scores); IMD-2, IMD-3, and IMD-4 (3–4, 5–6, and 7–8 of NZDep2013 scores, respectively); and IMD-5 (most deprivation: 9–10 of NZDep2013 scores).

Ethnicity

Patients' self-reported ethnicity was used and grouped into 5 categories (New Zealand European (NZE), Māori, Pasifika, Asian and other) using a prioritization methodology when multiple ethnic affiliations were present. The prioritisation grouped Māori (Indigenous Polynesian) as patients identifying as having any ancestry of Māori. Pasifika (93% Polynesian) were defined as patients identifying as having any Pasifika ancestry except for Māori. Asian were defined as patients having Asian ancestry (including Chinese, Indian, Southeast Asian) but neither Māori nor Pasifika. Those individuals with only European ancestry (from anywhere in the European continent) were defined as NZE. The individuals not included in the above 4 ethnic categories were defined as the "other ethnicity" group. The NZE individuals were assigned as the reference ethnic group compared with each of the other 4 ethnic groups.

Outcomes

We collected and assessed data on PM (all-cause death before 65 years) after enrolment. Incident events of MI, stroke, HF, and ESRD before the enrolment or within 12 months of enrolment were excluded to avoid potential information bias. All outcomes were extracted from linked national inpatient, outpatient, and death registration data. The censor time was defined as the date of having the outcome or 31 December 2019, whichever came first. All outcomes were defined based on the primary International Classification of Diseases, 9th Revision (ICD-9) and International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) codes.

Statistical Analysis

We first used Chi-square test to compare all outcomes between ethnic groups and IMD for deprivation. Each outcome data for patients with the least deprivation status and/or with NZE ethnicity was used as reference and applied to the whole study population or a specific population to calculate the expected counts of outcomes.

The attributable fraction (AF) was estimated as the absolute difference in the observed and expected counts of patients with outcome, then divided by the observed count of patients with that outcome.

The AF was interpreted as the proportion of outcome that would not have occurred in a population if the rate of the outcome was equal as the patients in the reference population. The AF compares the reference subpopulation either with the whole population, producing a population attributable fraction (PAF), or with a specific population, producing a group-specific AF, also known as AF in the exposed.[8] A negative AF means decreased risk compared with the reference group.

Cox regression models were used to predict expected counts of patients with outcomes, with adjustment of ethnicity or deprivation, age by enrolment, gender, period of having diabetes, the status of smoking, blood pressure, body mass index (BMI), HbA1c, lipids, estimated glomerular filtration rate (eGFR), antihypertensive treatments, anticoagulant treatment, statin, and anti-diabetes treatment. Incidence of any outcome was calculated per 1000 person-years. The unadjusted and adjusted AF with their 95% confidence intervals (CI) were computed. The percentage of missing data on the covariates ranged 0–6% of eligible patients. Using the worst-case scenario of 6% of cohort members with one or more missing covariates, 6 imputed datasets were generated using the multiple imputation with chained equations.[9] Final adjusted estimations were derived from imputed models.

We also implemented a series of sensitivity analyses by estimating adjusted estimations in the sub-populations including male and female populations, population aged less and more than 60 years, populations with non-smoking and current/ex-smoking status, and populations enrolled before and after 2006.

All analyses were performed with Stata 17.0 (StataCorp, College Station, TX, USA).

Results

A total of 36,267 patients with T2DM were enrolled in the DCSS between January 1, 1994 and July 31, 2018, without any outcome before the enrolment date or any outcome (except premature mortality) within 12 months since the enrolment date. The mean age at enrolment was 55.4 (standard deviation (SD), 13.5) years, and 49.6% were female. Clinical measurements and treatment at baseline (enrolment) are presented in Table 1.

Table 1 Patient Characteristics in the Type 2 Diabetes Cohort at Baseline (Enrolment)

Characteristics	**T2DM Cohort**
	N=36,267
Age, years	55.4 (13.5)
Female gender, n (%)	18,004 (49.6)
Ethnicity group, n (%)	
New Zealand European	13,321 (36.7)
Māori	5809 (16.0)
Pasifika	9383 (25.9)
Asian	5774 (15.9)
Other	1980 (5.5)
Index of multiple deprivation, n (%)	
Least deprivation	3502 (9.7)
IMD-2	4814 (13.3)
IMD-3	4365 (12.0)
IMD-4	6639 (18.3)
Most deprivation	16,947 (46.7)
Smoking status, n (%)	
Never smoker	24,586 (67.8)
Ex-smoker	6375 (17.6)
Current smoker	5306 (14.6)
Duration of having diabetes, years	4.8 (1.2)
Body mass index, kg/m^2	32.7 (7.6)
Systolic blood pressure, mmHg	134 (18)
Diastolic blood pressure, mmHg	81 (11)

(*Continued*)

Table 1 (Continued).

Characteristics	T2DM Cohort
	N=36,267
HbA1c, mmol/mol	61.3 (14.4)
Total cholesterol, mmol/L	5.1 (1.2)
High density lipoprotein cholesterol, mmol/L	1.2 (0.4)
Low density lipoprotein cholesterol, mmol/L	2.5 (1.1)
Triglyceride, mmol/L	2.3 (1.1)
Estimated Glomerular filtration rate (eGFR), mL/min/1.73 m^2	72.4 (21.3)
Antihypertensive treatment, n (%)	25,208 (69.5)
Lowering-lipids treatment, n (%)	21,133 (58.3)
Anticoagulant treatment, n (%)	736 (2.0)
Antidiabetes treatment, n (%)	
Oral antidiabetes drug and insulin	28,565 (78.8)
Oral antidiabetes only	7192 (19.8)
Insulin only	6262 (17.3)

Note: Continuous variables are presented as mean (standard deviation).

The rate of PM increased with socioeconomic deprivation, from 0.9 per 1000 person-years in the least deprived population to 4.9 per 1000 person-years in the most deprived population (Figure 1, Supplemental Table 1). The PAF for socioeconomic deprivation was 38.1 (95% confidence interval: 25.1–48.8)% unadjusted (Supplemental Table 2) and 6.6 (−30.8–33.3)% adjusting for ethnicity, age, sex, smoking status, body measurements, HbA1c, lipid profiles, eGFR, and treatment (Table 2). Risk of PM varied with ethnicity ranging from 1.5 per 1000 person-years among Asians to 8.2 per 1000 person-years among Māori (Figure 1, Supplemental Tables 1 and 2). The PAF for ethnicity was 33.8 (28.2–39.1)% before adjustment (Supplemental Table 2) and dropped to 14.3 (3.3–25.4)% after adjusting for socioeconomic group, age, sex, smoking status, body measurements, HbA1c, lipid profiles, eGFR, and treatment (Table 2).

The risk of MI varied by socioeconomic deprivation, ranging from 8.4 per 1000 person-years in the least deprived group to 10.4 per 1000 person-years in the 3rd socioeconomic deprivation quintile group (Figure 1, Supplemental Table 1). The PAF for socioeconomic deprivation was 14.6 (6.1–22.3)% before adjustment (Supplemental Table 2) and 17.1 (5.8–27.0)% after adjustment (Table 2). Risk of MI also varied by ethnicity, ranging from 7.3 per 1000 person-years among Asians to 11.8 per 1000 person-years among NZE (Figure 1, Supplemental Table 1). The PAF for ethnicity was −21.6 (−25.4 to −17.9)% unadjusted (Supplemental Table 2) and −3.3 (−8.3–1.5) % adjusted (Table 2).

The risk of stroke increased with socioeconomic deprivation, from 5.1 per 1000 person-years in the least deprived group, to 6.3 per 1000 person-years in the 3rd socioeconomic deprivation quintile group (Figure 1, Supplemental Table 1). The PAF for socioeconomic deprivation was 14.1 (3.1–23.8)% unadjusted (Supplemental Table 2) and 35.3 (22.6–46.0)% adjusted (Table 2). The risk of stroke varied with ethnicity, ranging from 4.3 per 1000 person-years among Asians to 6.9 per 1000 person-years among NZE (Figure 1, Supplemental Table 1). The PAF for ethnicity was −18.2 (−22.9 to −13.7) % unadjusted (Supplemental Table 2), and −0.5 (−6.7–5.3)% adjusted (Table 2).

The risk of HF increased with socioeconomic deprivation, from 10.2 per 1000 person-years in the least deprived to 13.8 per 1000 person-years in the most deprived (Figure 1, Supplemental Table 1). The PAF for socioeconomic deprivation was 19.7 (12.5–26.4)% unadjusted (Supplemental Table 2) and 14.3 (3.2–24.2)% adjusted (Table 2). The risk of HF varied with ethnicity ranging from 6.6 per 1000 person-years among Asians to 16.6 per 1000 person-years

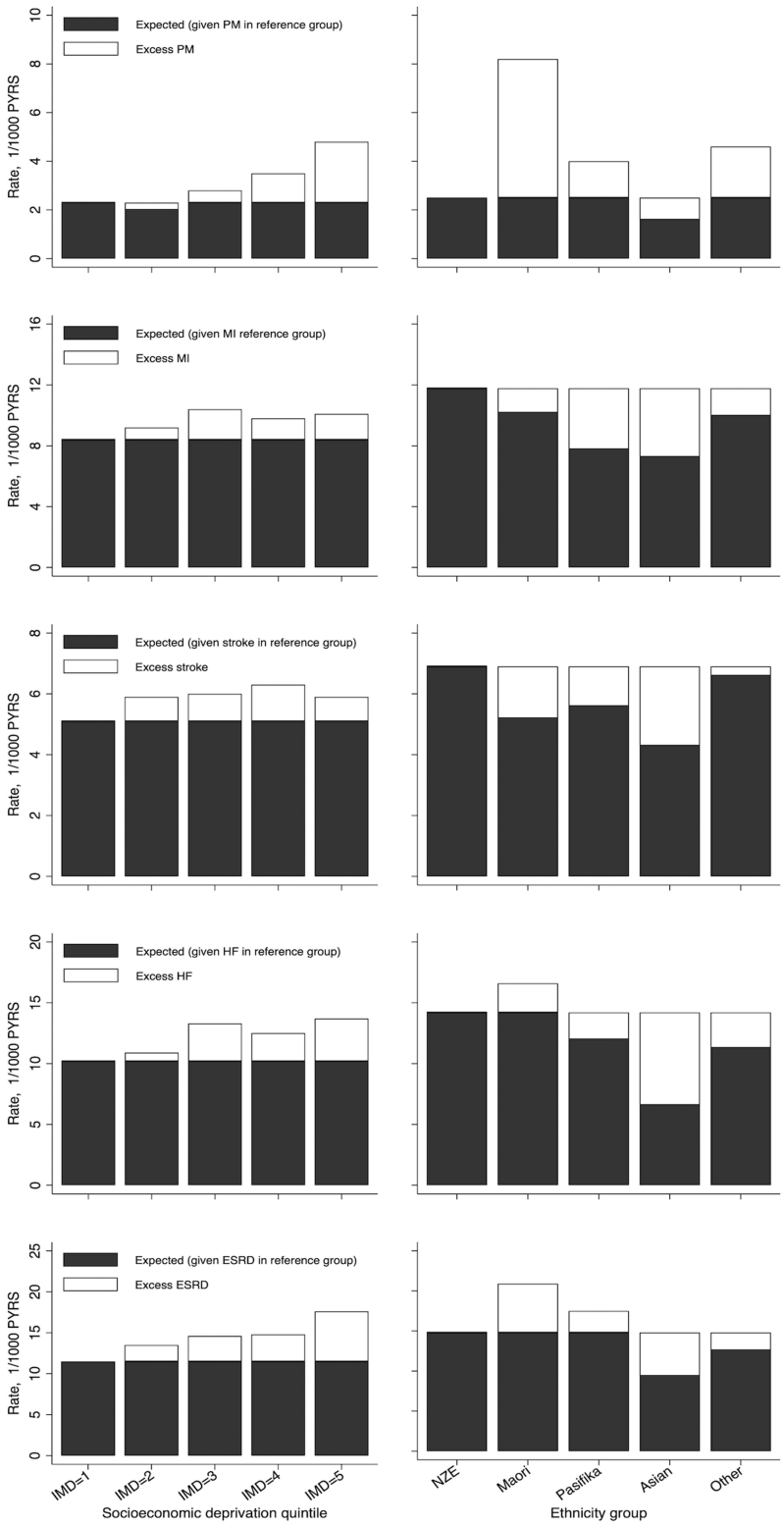


Figure 1 Premature mortality, myocardial infarction, stroke, heart failure, and end-stage renal disease rates by socioeconomic deprivation quintile and ethnic group in type 2 diabetes cohort. NZE, PM, MI, HF, ESRD, and PYRS indicates New Zealand European, premature mortality, myocardial infarction, end-stage renal disease, and person-years. Rates in IMD=1 and NZE group was used as reference group, respectively.

Table 2 Population Attributable Fractions of Premature Mortality, Myocardial Infarction, Stroke, Heart Failure, End-Stage Renal Disease by Socioeconomic Deprivation and Ethnicity

Outcome	Socioeconomic Deprivation[§]	Ethnicity[¶]
	Overall	
Premature mortality	6.6 (−30.8 to 33.3) %	14.3 (3.3 to 25.4) %
Myocardial infarction	17.1 (5.8 to 27.0) %	−3.3 (−8.3 to 1.5) %
Stroke	35.3 (22.6 to 46.0) %	−0.5 (−6.7 to 5.3) %
Heart failure	14.3 (3.2 to 24.2) %	4.7 (0.3 to 8.8) %
End-stage renal disease	15.9 (6.7 to 24.2) %	13.3 (9.9 to 16.6) %
	Women	
Premature mortality	−52.8 (−140.2 to 2.8) %	27.0 (3.5 to 44.8) %
Myocardial infarction	10.2 (−9.7 to 26.5) %	−3.9 (−12.1 to 3.7) %
Stroke	30.5 (9.3 to 46.7) %	4.7 (−4.5 to 13.1) %
Heart failure	15.3 (−2.4 to 29.9) %	3.1 (−3.8 to 9.5) %
End-stage renal disease	20.9 (6.0 to 33.5) %	14.9 (9.4 to 20.0) %
	Men	
Premature mortality	35.9 (−4.4 to 60.7) %	5.2 (−16.2 to 22.5) %
Myocardial infarction	21.6 (7.7 to 33.5) %	−2.64 (−9.2 to 3.5) %
Stroke	39.6 (22.9 to 52.7) %	−5.5 (−14.1 to 2.4) %
Heart failure	13.8 (1.9 to 25.8) %	6.1 (0.4 to 11.4) %
End-stage renal disease	13.0 (1.0 to 23.6) %	12.2 (7.8 to 16.4) %
	<60 years	
Premature mortality	17.8 (5.9 to 29.7) %	16.9 (6.0 to 26.6) %
Myocardial infarction	21.7 (3.8 to 36.3) %	−0.8 (−10.4 to 7.9) %
Stroke	45.9 (24.4 to 61.2) %	−3.0 (−16.3 to 8.7) %
Heart failure	20.3 (0.8 to 36.0) %	16.4 (8.2 to 23.8) %
End-stage renal disease	19.7 (5.1 to 32.0) %	16.1 (10.1 to 21.7) %
	≥60 years	
Premature mortality	2.1 (−37.1 to 30.1) %	9.7 (−6.6 to 23.5) %
Myocardial infarction	13.5 (−1.5 to 26.3) %	−5.4 (−10.4 to −0.6) %
Stroke	29.5 (12.9 to 43.0) %	−0.6 (−6.6 to 5.0) %
Heart failure	10.0 (−3.9 to 22.0) %	−3.1 (−7.6 to 1.3) %
End-stage renal disease	12.9 (0.8 to 23.5) %	8.8 (5.2 to 12.3) %
	Non-smokers	
Premature mortality	13.6 (−33.8 to 44.2) %	−1.7 (−24.4 to 16.8) %
Myocardial infarction	15.4 (1.8 to 27.0) %	−2.7 (−8.8 to 3.1) %
Stroke	30.4 (14.8 to 43.2) %	3.7 (−3.3 to 10.2) %
Heart failure	10.0 (−3.6 to 21.8) %	2.5 (−2.9 to 7.5) %
End-stage renal disease	19.0 (8.2 to 28.6) %	11.1 (6.9 to 15.2) %
	Current smokers and Ex-smokers	
Premature mortality	−8.3 (−82.3 to 35.7) %	32.0 (10.3 to 48.5) %
Myocardial infarction	21.7 (−0.1 to 38.7) %	−5.4 (−15.0 to 3.3) %

(*Continued*)

Table 2 (Continued).

Outcome	Socioeconomic Deprivation[§]	Ethnicity[¶]
Stroke	46.2 (20.8 to 63.4) %	−11.0 (−24.5 to 1.0) %
Heart failure	25.3 (4.0 to 41.9) %	7.5 (2.4 to 12.7) %
End-stage renal disease	7.5 (−11.4 to 23.1) %	17.7 (11.7 to 23.2) %
	Enrolment year ≤ 2006	
Premature mortality	−1.9 (−47.4 to 29.6) %	18.0 (0.8 to 32.2) %
Myocardial infarction	14.8 (1.8 to 26.1) %	−2.6 (−8.0 to 2.6) %
Stroke	34.7 (19.8 to 46.9) %	0.3 (−6.6 to 6.6) %
Heart failure	13.6 (0.8 to 24.7) %	6.6 (3.8 to 9.4) %
End-stage renal disease	15.8 (4.9 to 25.5) %	17.8 (14.1 to 21.5) %
	Enrolment year > 2006	
Premature mortality	27.9 (−62.7 to 68.0) %	−14.0 (−61.4 to 19.4) %
Myocardial infarction	22.4 (−2.7 to 41.4) %	−5.5 (−19.74 to 7.1) %
Stroke	37.1 (8.6 to 56.7) %	−6.7 (−24.0 to 8.2) %
Heart failure	15.4 (−9.7 to 34.8) %	2.1 (−9.1 to 12.1) %
End-stage renal disease	15.2 (2.9 to 27.5) %	4.6 (0.6 to 8.6) %

Notes: [§]Adjusting for ethnicity, Age, sex, smoking status, body measurements, HbA1c, lipid profiles, eGFR, antihypertensive, anticoagulant, lipid-lowering, and antidiabetes treatments; [¶]Adjusting for socio-economic group, age, sex, smoking status, body measurements, HbA1c, lipid profiles, eGFR, treatments, antihypertensive, anticoagulant, lipid-lowering, and antidiabetes treatments.

among Māori (Figure 1, Supplemental Table 1). The PAF for ethnicity was −12.4 (−15.7 to −9.2)% unadjusted (Supplemental Table 3) and 4.7 (0.3–8.8)% adjusted (Table 2).

The risk of ESRD increased with socioeconomic deprivation, from 11.5 per 1000 person-years in the least deprived group to 17.6 per 1000 person-years in the most deprived group (Figure 1, Supplemental Table 1). The PAF for socioeconomic deprivation was 26.2 (19.9–31.9)% unadjusted (Supplemental Table 3) and 15.9 (6.7–24.2)% adjusted (Table 2). The risk of ESRD varied with ethnicity, ranging from 9.4 per 1000 person-years among Asians to 20.9 per 1000 person-years among Māori (Figure 1, Supplemental Table 1). The PAF for ethnicity was 4.7 (1.9–7.4)% unadjusted (Supplemental Table 2) and 13.3 (9.9–16.6)% adjusted (Table 2).

The PAF for each outcome by gender, age-strata, smoking status revealed similar findings (Table 2; Supplemental Tables 3–10).

Figure 2 compares the group-specific AF by deprivation-ethnic group with reference to NZE with the least socio-economic deprivation. The group-specific AF for PM was higher in the most deprived NZE group (53.6 (48.1–59.0)%), each socioeconomic group in Māori (81.8 (79.8–83.8)%, 67.8 (65.7–70.0)%, 76.7 (76.1–77.0)%, 74.3 (72.7–75.9)%, and 79.2 (75.8–82.6)%), more deprived Pasifika with 62.0 (59.5–64.5)% and 54.5 (47.3–61.6)% for IMD quintile 4–5, and for IMD quintiles 1 and 5 in the other ethnic group (64.4 (58.3–70.4)% and 70.4 (69.7–71.0)%).

The group-specific AF for MI was higher in more deprived NZE (13.2 (12.1–14.4)%, 24.8 (24.8–24.9)%, 21.2 (21.1–21.3)%, and 19.4 (17.5–21.3)%) for IMD quintile 2–5; Māori (17.5 (11.6–23.5)%, 9.5 (5.9–13.0)%, 8.1 (5.5–10.8)%) for IMD quintile 3–5, and lower among Asians (−98.6 (−124.5 to −72.8)%, −48.7 (−59.7 to −37.7)%, −6.7 (−15.6 to 2.2)%, −47.2 (−58.0 to −36.5)%, and −3.2 (−4.9 to −1.5)%) for IMD quintile 1–5.

The group-specific AF for stroke was higher in the more deprived NZE (20.9 (18.8–22.9)%, 19.9 (18.3–21.6)%, 27.4 (24.6–30.3)%, and 29.3 (25.6–33.0)%) for IMD quintile 2–5; some deprived other ethnic groups (35.8 (27.9–43.6)%, 40.8 (33.4–48.2)%, 7.4 (0.8–14.0)%) for IMD quintile 2, 3 and 5, but lower in each deprivation group among Asians (−50.8 (−70.5 to −31.1)%, −32.0 (−42.7 to −21.3)%, −35.1 (−52.0 to −18.2)%, −14.0 (−21.7 to −6.2)%, and −17.7 (−21.3 to −14.1)%) for IMD quintile 1–5.

		Ethnic group				
		NZE	Māori	Pasifika	Asian	Other
Premature mortality						
	Socioeconomic deprivation					
	IMD=1 (Least deprivation) %	Reference	81.8 (79.8 to 83.8) %	15.3 (-11.7 to 42.2) %	-28.0 (-48.5 to -7.5) %	64.4 (58.3 to 70.4) %
	IMD=2	2.4 (0.1 to 4.7) %	67.8 (65.7 to 70.0) %	17.9 (2.9 to 32.9) %	-100.3 (-134.5 to -66.5) %	38.2 (27.0 to 49.5) %
	IMD=3	14.3 (-14.8 to 13.8) %	76.5 (76.1 to 77.0) %	40.7 (35.0 to 46.5) %	17.3 (8.2 to 26.4) %	45.9 (36.1 to 55.8) %
	IMD=4	23.1 (18.5 to 27.6) %	74.3 (72.7 to 75.9) %	62.0 (59.5 to 64.5) %	-17.9 (-29.3 to -6.4) %	47.9 (40.6 to 55.3) %
	IMD=5 (Most deprivation) %	53.6 (48.1 to 59.0) %	79.2 (75.8 to 82.6) %	54.5 (47.3 to 61.6) %	-7.8 (-10.6 to -5.0) %	70.4 (69.7 to 71.0) %
Myocardial infarction						
	Socioeconomic deprivation					
	IMD=1 (Least deprivation) %	Reference	10.1 (-8.6 to 28.5) %	-61.0 (-103.8 to -18.2) %	-98.6 (-124.5 to -72.8) %	24.2 (12.3 to 36.1) %
	IMD=2	12.7 (11.3 to 14.1) %	-4.6 (-18.3 to 9.0) %	-15.8 (-31.4 to 0.3) %	-48.7 (-59.7 to -37.7) %	-6.1 (-21.6 to 9.4) %
	IMD=3	12.4 (11.1 to 13.6) %	17.5 (11.6 to 23.5) %	-8.2 (-19.4 to 2.9) %	-6.7 (-15.6 to 2.2) %	29.8 (21.4 to 38.3) %
	IMD=4	25.7 (23.5 to 27.9) %	9.5 (5.9 to 13.0) %	-26.7 (-31.0 to -22.3) %	-47.2 (-58.0 to -36.5) %	-41.3 (-64.8 to -17.8) %
	IMD=5 (Most deprivation) %	34.0 (31.2 to 36.8) %	8.1 (5.5 to 10.8) %	-17.7 (-22.6 to -12.9) %	-3.2 (-4.9 to -1.5) %	9.1 (3.9 to 14.2) %
Stroke						
	Socioeconomic deprivation					
	IMD=1 (Least deprivation) %	Reference	4.4 (-19.8 to 28.6) %	25.2 (8.8 to 41.5) %	-50.8 (-70.5 to -31.1) %	-0.6 (-22.6 to 21.3) %
	IMD=2	20.88 (18.81 to 22.94) %	-18.3 (-38.6 to 2.0) %	-41.2 (-67.5 to -14.8) %	-32.0 (-42.7 to -21.3) %	35.8 (27.9 to 43.6) %
	IMD=3	19.91 (18.27 to 21.56) %	0.5 (-10.0 to 11.0) %	-36.0 (-56.6 to -15.5) %	-35.1 (-52.0 to -18.2) %	40.8 (33.4 to 48.2) %
	IMD=4	27.44 (24.57 to 30.32) %	7.2 (2.7 to 11.8) %	5.5 (3.4 to 7.6) %	-14.0 (-21.7 to -6.2) %	3.5 (-11.8 to 18.8) %
	IMD=5 (Most deprivation) %	29.31 (25.61 to 33.00) %	-7.2 (-10.3 to -4.2) %	6.5 (0.8 to 12.3) %	-17.7 (-21.3 to -14.1) %	7.4 (0.8 to 14.0) %
Heart failure						
	Socioeconomic deprivation					
	IMD=1 (Least deprivation) %	Reference	32.6 (10.6 to 54.7) %	-25.2 (-92.2 to 41.9) %	-108.9 (-148.6 to -69.3) %	46.6 (33.1 to 60.1) %
	IMD=2	6.6 (5.3 to 7.9) %	20.8 (8.3 to 33.3) %	-28.1 (-57.8 to 1.7) %	2.3 (-42.0 to 46.5) %	43.6 (35.7 to 51.4) %
	IMD=3	12.8 (11.7 to 13.9) %	31.9 (26.6 to 37.2) %	1.2 (-12.0 to 14.4) %	37.4 (-4.2 to 78.9) %	30.7 (4.9 to 56.5) %
	IMD=4	20.9 (18.7 to 23.2) %	22.8 (20.0 to 25.5) %	-3.1 (-4.4 to 1.7) %	1.8 (-7.8 to 11.4) %	-47.1 (-124.8 to 30.7) %
	IMD=5 (Most deprivation) %	27.2 (24.3 to 30.2) %	30.3 (27.6 to 33.0) %	2.4 (-3.4 to 8.2) %	24.3 (16.8 to 31.8) %	16.8 (10.2 to 23.5) %
End-stage renal disease						
	Socioeconomic deprivation					
	IMD=1 (Least deprivation) %	Reference	33.4 (13.3 to 53.5) %	-11.3 (-27.4 to 4.8) %	-101.5 (-118.2 to -84.9) %	-10.1 (-24.1 to 3.9) %
	IMD=2	9.7 (9.3 to 10.1) %	29.8 (20.4 to 39.3) %	23.5 (13.1 to 33.9) %	-26.8 (-27.3 to -26.1) %	-17.0 (-25.8 to -8.3) %
	IMD=3	11.3 (10.3 to 12.3) %	35.4 (30.9 to 39.8) %	30.7 (24.7 to 36.8) %	-31.1 (-31.9 to -30.3) %	-2.7 (-15.3 to 10.0) %
	IMD=4	15.1 (14.7 to 15.4) %	38.0 (36.7 to 39.3) %	24.2 (23.6 to 24.8) %	-53.0 (-59.0 to -47.1) %	-34.4 (-38.4 to -30.3) %
	IMD=5 (Most deprivation) %	24.7 (24.2 to 25.3) %	41.6 (39.2 to 44.1) %	29.1 (28.5 to 29.6) %	-14.9 (-15.9 to -13.9) %	17.4 (12.4 to 22.5) %

Figure 2 Attributable fractions of premature mortality, myocardial infarction, stroke, heart failure, end-stage renal disease by socioeconomic deprivation and ethnic group. Data are attributable fraction (AF) (95% CI), calculated by comparison with NZE in the least deprived quintile. Gray, green, light blue, and dark blue colors indicate the AF is not significant, significant and less than zero, significant and less than 50%, and significant and more than 50%, respectively.

The group-specific AF for HF was higher among more deprived NZE (6.6 (5.3–7.9)%, 12.8 (11.7–13.9)%, 20.9 (18.7–23.2)%, and 27.2 (24.3–30.2)%) for IMD quintile 2–5, each socioeconomic group among Māori (32.6 (10.6–54.7)%, 20.8 (8.3–33.3)%, 31.9 (26.6–37.2)%, 22.8 (20.0–25.5)%, and 30.3 (27.6–33.0)%) for IMD quintile 1–5, the most deprived Asian (24.3 (16.8–31.8)%) and other ethnic groups (16.8 (10.2–23.5)%).

The group-specific AF for ESRD was higher among the more deprived NZE (9.7 (9.3–10.1)%, 11.3 (10.3–12.3)%, 15.1 (14.7–15.4)%, and 24.7 (24.2–25.3)%) for IMD quintile 2–5, each deprivation group among Māori, some deprived Pasifika, and the most deprived other ethnic group (17.4 (12.4–22.5)%) but lower in each Asian deprivation group.

As the IMD was based on national socioeconomic information in the general population, the population size or outcomes were not evenly distributed in each IMD category for each ethnicity. To further understand the AF presented in Figure 2, attributable absolute numbers of each outcome for each IMD category in each ethnicity (except least deprived of NZE) are presented in Supplemental Table 11 with the population size in each ethnic cohort by IMD in Supplemental Table 12.

Discussion

Main Findings

Based on the AF derived from a population-based cohort of T2DM in Auckland, NZ from 1994–2018, if event rates for the entire population were those of people with the least deprivation, 17% of MI, 35% of stroke, 14% of HF, and 16% of ESRD would not have occurred, even after adjusting for ethnicity, demographic characteristics, lifestyle, body and clinical measurements and treatment. In the same population, 14% of PM, 5% of HF, and 13% of ESRD were attributable to ethnicity, after adjustment for socioeconomic deprivation, demographic characteristics, lifestyle, body and routine clinical measurements, and treatment. However, a new finding, and particularly important from a health equity perspective is that the relationship between deprivation and different complications varied with ethnicity. Increasing deprivation contributed to a growing AF of PM, MI, stroke, HF and ESRD among NZE and Asians. Among NZE, as

much as 54% of PM, 34% of MI, 29% of stroke, 27% of HF, and 25% of ESRD were attributable to disparities in deprivation. However, among Māori, with the greatest rates of PM, HF, and ESRD, increasing deprivation was not related to a significant increase in any of the complications. Among Pasifika, increasing deprivation contributed to more PM and ESRD, while among other ethnic groups, increasing deprivation contributed to more PM. Asians actually had a lower group-specific AF of PM, MI, stroke, and ESRD compared with NZE with the least deprivation. NZE with the most deprivation had the greatest AF for MI and stroke while Pasifika, Māori, and Asians generally had the lowest.

Strengths and Limitations

There are some strengths in the current study, incorporating being the largest study of a multi-ethnic prospective cohort of at-risk population with T2DM in NZ (and one of the largest globally) to report important diabetes-relevant clinical outcomes covering 25 years by both deprivation and ethnicity. The current cohort incorporated all T2DM patients from participating general practices, and a high proportion of general practices in the area. By linking national representative databases, patients were followed up to ensure all incident outcomes, including mortality and cause-specific outcomes. All clinical outcomes utilized in the current study were based on the linkage of specific national registration datasets that provided well-validated outcomes. The accuracy of clinical diagnoses and records in the current study have been validated for a range of comorbidities, and for cause-specific outcomes the study also used primary ICD codes that have high validity.[10]

Limitations of the current study include heterogeneity in the NZE, Māori, Pasifika, Asian, and other ethnic populations (and need for definition of single ethnicity where multiple ancestries exist), the representativeness of the population at national level and of the participating general practices. Metabolic measures reflect time of entry into the DCSS rather than "lifelong" exposure and other measures (e.g. smoking) have been simplified in terms of degree of exposure. The use of the IMD, based on meshblocks of 81 houses, to represent the SES of a person, their family and indeed individual ethnic groups may also be imprecise. For example, the IMD reflects the documented residency of an individual who might actually live elsewhere for substantial periods of time, perhaps particularly if part of an extended family. However, our previous investigation found that the SES has not shifted between 2001–2016 among 95% of DCSS cohort members.[11] Restricted by outcomes, the group-specific attributable risk of adverse prognostic outcomes was not implemented in the subgroup analysis (e.g. by gender, age-group, and smoking status). Further external replication studies with more outcomes to apply these subgroup analyses are warranted.

Interpretation and Implications

The AF is interpreted as the percentage of PM/diabetes complications that would not have happened if patients with T2DM were exposed to a similar background assuming that there are no other biases and no modification effect.[8] It is unlikely that this assumption was fully met in the context of the current study.

For the current study, this assumption was unlikely to be met in full, as exposures of interest such as ethnicity and socioeconomic deprivation are associated with other factors or circumstances, covering general health status, health relevant behavior, nutrition intake/status, and other known risk factors associated with adverse clinical outcomes in T2DM.[12,13]

Ethnic disparities in outcomes within patients with T2DM in NZ and other high-income nations are reported widely and often purported to be largely due to socioeconomic differences.[14,15] Such disparities are associated with causal pathways that can be long and complex.[16] Socioeconomic deprivation is linked to a wider spectrum of adverse circumstances, incorporating restricted access to new treatment, reduced access to affordable healthy foods, and wider social determinants of health.

Other potential pathways through which socioeconomic disparities can have influence on prognostic outcomes are environmental exposure (e.g. air pollution); paucity of social cohesion and social isolation; restricted access to an integrated care system able to respond to their needs; and increased mental health issues related to insecure employment, economic strain, and more frequent stressful events. Institutional racism and/or unconscious bias that exists in policy decisions and the health system have also been reported as important factors.[17,18]

We now show that the association between deprivation, as a proxy for socioeconomic status, and ethnicity is much more complex and nuanced. NZE are likely to be similar to other populations of European descent, and the growing

contribution of deprivation with increasing IMD to the risk of each complication including premature mortality, reflects the findings in many comparable populations.[19,20] This relationship between complications and deprivation was also shown in Asians, a non-European population, who had lower absolute risks than Europeans. Again, this is reflected in other studies among Asians:[21] both South[22] and East[23] Asians. However, this lower risk may also partly result from the selection processes for entry into NZ to live which are likely to favour healthier individuals. The findings among other ethnicities could well reflect the heterogeneity of the group.

However, it is the patterns among Māori and Pasifika that indicate the complexity of the relationship between deprivation and ethnicity. Among Māori, somewhat counter-intuitively, deprivation showed no major contribution to the high rates of PM and ESRD, with lower rates of cardiovascular complications compared with NZE of comparable degrees of deprivation. One possible explanation is that the incidence of these complications is so high, possibly from non-diabetes related causes, that they have almost "bottomed out", with no additional effect of deprivation. The high rates of diabetic kidney disease may indeed be due to a predisposition to renal disease rather than the T2DM itself.[24] Other suggested causes such as reduced access to care by Māori and Pasifika patients[25,26] was previously not shown in the current cohort, with more consultations for diabetes and similar or more screening programs or tests than patients in Europe.[27] However, these diabetes consultations and screening programs were clearly not sufficient for their actual needs in relation to glycemic (but not blood pressure or lipid) management[28] and complications[11] and do not echo, for example, health literacy or whether culturally safe care was provided. Even though the prescription of antihyperglycemic drugs, antihypertensives, statins, remained higher among Māori and Pasifika T2DM patients than NZE T2D patients, averagely higher HbA1c could have some contribution to this excess risk. It has been suggested the diagnosis in Māori patients is later than it is in NZE patients. However, a previous study implemented in the neighboring Waikato district reported the prevalence of retinopathy at diagnosis was low,[29] as was the prevalence of undiagnosed diabetes,[30] which suggested the lag between progress and diagnosis of T2DM was not very large. Others have proposed that the disparities among Māori are built upon a history of colonization[31] and racism;[32] further metrics are required to demonstrate this relationship objectively.[33]

Pasifika still showed a relationship between deprivation and PM but not consistently with the other adverse outcomes. Pasifika had lower attributable fractions for cardiovascular events than NZE. Unlike Māori, Pasifika in this sample were less likely to have admixture with other non-Māori ethnic groups, which may alter their risk of adverse outcomes.[34] Pasifika communities may also differ from Māori in the timing of screening for and diagnosis of diabetes. In an earlier Auckland-based study, Pasifika had the lowest proportion of undiagnosed diabetes (Pasifika 17%, Māori 24%, NZE 31%) suggesting timely diagnosis.[35] However, in a more recent study Pasifika had the highest proportion of undiagnosed diabetes (41%) compared with Māori (22%) and NZ European (24%).[36] Our work demonstrates the need for further research into the contribution of biological, environmental, societal and social determinants to the differences described here, and the extent to which they reflect disparities elsewhere.

Understanding such differences may be able to inform policy initiatives to reduce adverse outcomes among those with T2DM in New Zealand. Our findings support the recent policy of increasing access to newer diabetes agents (e.g. sodium–glucose co-transporter-2 inhibitors) for Māori and Pasifika patients with T2DM in NZ in response to their higher risk of ESRD in particular.[11,37] However, our findings also support improved access for NZE and other ethnic groups of low socioeconomic status. These findings also validate the need for greater research to provide evidence for interventions that are culturally tailored such as the DEFEND trial.[38] Such evidence should then be rapidly translated into evaluated, funded initiatives.

Most existing initiatives with goals to decrease adverse clinical outcomes recommend that primary care concentrates on individual risk factors and on specific populations predicted to have high risk.[17] This study provides further support for targeting Māori and Pasifika communities for additional culturally tailored interventions, considering their higher burden of adverse outcomes and major influence of the extended family and wider community. An approach that emphasises the ability of a patient to manage risk factors that are at least partly due to their societal attitudes and social context may be unhelpful.[14] For those at the highest risk, integrating the care available from diabetes, mental health, cultural and social services to lessen the risk of outcomes might be more likely to have a positive impact by tackling the consequences of ethnic and socioeconomic disparities, as well as the complexities of modern diabetes management.

Conclusion

Results of the current study indicate that both ethnic and socioeconomic disparities contributed a significant proportion of adverse outcomes in patients with T2DM in NZ. Māori had the highest rate of PM, HF and ESRD, Pasifika the next highest rate of ESRD, but NZE the highest rate of MI and stroke. Asians consistently had lower rates of adverse outcomes. The largest within-group disparities with regard to socio-economic deprivation were observed in the NZE group, especially for PM, with 53.6% of AF in the most deprived group. Interestingly, risk remained similar regardless of socio-economic deprivation within Māori. In addition to ethnically targeted programs, new policies are urgently needed supporting intensive management and addressing socioeconomic deprivation, whilst tailored to the cultural needs of each ethnic group.

Acknowledgments

We thank the participating GPs, audit nurses and the DCSS executive group for their support.

Funding

The DCSS was funded by the New Zealand Ministry of Health through Counties Manukau District Health Board. The funders had no role in the study design, data collection, data analysis, data interpretation, or writing of the report.

Disclosure

The authors declare that they have no competing interests in this work.

References

1. Singh GK, Siahpush M, Azuine RE, Williams SD. Increasing Area deprivation and socioeconomic inequalities in heart disease, stroke, and cardiovascular disease mortality among working age populations, United States, 1969–2011. *Int J MCH AIDS*. 2015;3(2):119–133.
2. Metcalf PA, Scragg RR, Schaaf D, Dyall L, Black PN, Jackson RT. Comparison of different markers of socioeconomic status with cardiovascular disease and diabetes risk factors in the diabetes, heart and health survey. *N Z Med J*. 2008;121(1269):45–56.
3. Mackenbach JP, Bakker MJ. European network on interventions and policies to reduce inequalities in health: tackling socioeconomic inequalities in health: analysis of European experiences. *Lancet*. 2003;362(9393):1409–1414. doi:10.1016/S0140-6736(03)14639-9
4. Ezzatvar Y, Ramirez-Velez R, Izquierdo M, Garcia-Hermoso A. Racial differences in all-cause mortality and future complications among people with diabetes: a systematic review and meta-analysis of data from more than 2.4 million individuals. *Diabetologia*. 2021;64(11):2389–2401. doi:10.1007/s00125-021-05554-9
5. New Zealand Ministry of Health. Reducing Inequalities in Health. Available from: https://www.health.govt.nz/system/files/documents/publications/reducineqal.pdf. Accessed April 21, 2023.
6. Simmons D, Fleming CE, Cutfield R, Patel A, Innes J, Wellingham J. The diabetes care support service for general practitioners in Auckland. *N Z Med J*. 1997;110(1038):48–50.
7. Atkinson J, Salmond C, Crampton P. NZDep2013 Index of Deprivation. Available from: https://www.otago.ac.nz/wellington/otago069936.pdf. Accessed April 21, 2023.
8. Mansournia MA, Altman DG. Population attributable fraction. *BMJ*. 2018;360:k757. doi:10.1136/bmj.k757
9. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. *Stat Med*. 2011;30(4):377–399. doi:10.1002/sim.4067
10. Maru S, Koch GG, Stender M, et al. Antidiabetic drugs and heart failure risk in patients with type 2 diabetes in the U.K. Primary care setting. *Diabetes Care*. 2005;28(1):20–26. doi:10.2337/diacare.28.1.20
11. Yu D, Zhao Z, Osuagwu UL, et al. Ethnic differences in mortality and hospital admission rates between Maori, Pacific, and European New Zealanders with type 2 diabetes between 1994 and 2018: a retrospective, population-based, longitudinal cohort study. *Lancet Glob Health*. 2021;9(2):e209–e217. doi:10.1016/S2214-109X(20)30412-5
12. Herbert A, Gilbert R, Cottrell D, Li L. Causes of death up to 10 years after admissions to hospitals for self-inflicted, drug-related or alcohol-related, or violent injury during adolescence: a retrospective, nationwide, cohort study. *Lancet*. 2017;390(10094):577–587. doi:10.1016/S0140-6736(17)31045-0
13. Daly B, Toulis KA, Thomas N, et al. Increased risk of ischemic heart disease, hypertension, and type 2 diabetes in women with previous gestational diabetes mellitus, a target group in general practice for preventive interventions: a population-based cohort study. *PLoS Med*. 2018;15(1):e1002488. doi:10.1371/journal.pmed.1002488
14. Havranek E, Mujahid M, Barr D, et al. Social determinants of risk and outcomes for cardiovascular disease: a scientific statement from the American heart association. *Circulation*. 2015;132(9):873–898. doi:10.1161/CIR.0000000000000228
15. Tatulashvili S, Fagherazzi G, Dow C, Cohen R, Fosse S, Bihan H. Socioeconomic inequalities and type 2 diabetes complications: a systematic review. *Diabetes Metab*. 2020;46(2):89–99. doi:10.1016/j.diabet.2019.11.001
16. Grintsova O, Maier W, Mielck A. Inequalities in health care among patients with type 2 diabetes by individual socio-economic status (SES) and regional deprivation: a systematic literature review. *Int J Equity Health*. 2014;13:43–9276. doi:10.1186/1475-9276-13-43
17. Jansen RM, Sundborn G, Cutfield R, Yu D, Simmons D. Ethnic inequity in diabetes outcomes-inaction in the face of need. *N Z Med J*. 2020;133(1525):8–10.
18. Adler NE, Newman K. Socioeconomic disparities in health: pathways and policies. *Health Aff*. 2002;21(2):60–76. doi:10.1377/hlthaff.21.2.60

19. Monsivais P, Amiri S, Denne J, Amram O. Area deprivation amplifies racial inequities in premature mortality: analysis of 242,667 deaths in Washington State, USA 2011–15. *Health Place*. 2020;61:102261. doi:10.1016/j.healthplace.2019.102261
20. Saint-Jacques N, Dewars R, Cui Y, Parker L, Dummer T. Premature mortality due to social and material deprivation in Nova Scotia, Canada. *Int J Equity Health*. 2014;13(1):94. doi:10.1186/s12939-014-0094-2
21. Chaturvedi N. Ethnic differences in cardiovascular disease. *Heart*. 2003;89(6):681–686. doi:10.1136/heart.89.6.681
22. Saeed S, Kanaya AM, Bennet L, Nilsson PM. Cardiovascular risk assessment in South and Middle-East Asians living in the Western countries. *Pak J Med Sci*. 2020;36(7):1719–1725. doi:10.12669/pjms.36.7.3292
23. Ueshima H, Sewa A, Miura K, et al. Cardiovascular disease and risk factors in Asia: a selected review. *Circulation*. 2008;118(25):2702–2709. doi:10.1161/CIRCULATIONAHA.108.790048
24. Thompson C, Simmons D, Collins J, Cecil A. Predisposition to nephropathy in Polynesians is associated with family history of renal disease, not diabetes mellitus. *Diabetic Med*. 2001;18(1):40–46. doi:10.1046/j.1464-5491.2001.00406.x
25. Whitehead J, Pearson AL, Lawrenson R, Atatoa-Carr P. Spatial equity and realised access to healthcare - a geospatial analysis of general practitioner enrolments in Waikato, New Zealand. *Rural Remote Health*. 2019;19(4):5349. doi:10.22605/RRH5349
26. Reddy R, Welch D, Lima I, Thorne P, Nosa V. Identifying hearing care access barriers among older Pacific Island people in New Zealand: a qualitative study. *BMJ Open*. 2019;9(8):e029007–2019. doi:10.1136/bmjopen-2019-029007
27. Robinson T, Simmons D, Scott D, et al. Ethnic differences in type 2 diabetes care and outcomes in Auckland: a multiethnic community in New Zealand. *N Z Med J*. 2006;119(1235):U1997.
28. Yu D, Cai Y, Osuagwu UL, et al. Metabolic profiles of Maori, Pacific, and European New Zealanders with type 2 diabetes over 25 years. *Diabetes Care*. 2021;44:e183–e185. doi:10.2337/dc21-1255
29. Lim S, Chellumuthi C, Crook N, Rush E, Simmons D. Low prevalence of retinopathy, but high prevalence of nephropathy among Maori with newly diagnosed diabetes-Te Wai o Rona: diabetes prevention strategy. *Diabetes Res Clin Pract*. 2008;80(2):271–274. doi:10.1016/j.diabres.2007.12.018
30. Simmons D, Rush E, Crook N. Prevalence of undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose among Maori in Te Wai o Rona: diabetes prevention strategy. *N Z Med J*. 2009;122(1288):30–38.
31. Axelsson P, Kukutai T, Kippen R. The field of Indigenous health and the role of colonisation and history. *J Popul Res*. 2016;33:1–7. doi:10.1007/s12546-016-9163-2
32. Reid P, Cormack D, Paine S. Colonial histories, racism and health-the experience of Māori and Indigenous peoples. *Public Health*. 2019;172:119–124. doi:10.1016/j.puhe.2019.03.027
33. Daniel M, Cargo M, Marks E, et al. Rating health and social indicators for use with indigenous communities: a tool for balancing cultural and scientific utility. *Soc Indic Res*. 2009;94(2):241–256. doi:10.1007/s11205-008-9420-7
34. Risch N, Burchard E, Ziv E, Tang H. Categorization of humans in biomedical research: genes, race and disease. *Genome Biol*. 2002;3(7):comment2007–2002. doi:10.1186/gb-2002-3-7-comment2007
35. Sundborn G, Metcalf P, Scragg R, et al. Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand. *N Z Med J*. 2007;120(1257):U2607.
36. Coppell K, Mann J, Williams S, et al. Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey. *N Z Med J*. 2013;126(1370):23–42.
37. PHARMAC. Decision to fund two new medicines for type 2 diabetes - Amended with Q&A. Available from: https://pharmac.govt.nz/news-and-resources/consultations-and-decisions/decision-to-fund-two-new-medicines-for-type-2-diabetes/. Accessed April 21, 2023.
38. Tan J, Manley P, Gamble G, et al. DEFEND investigators: long-term effectiveness of a community-based model of care in Maori and Pacific patients with type 2 diabetes and chronic kidney disease: a 4-year follow up of the DElay Future End Stage Nephropathy due to Diabetes (DEFEND) study. *Intern Med J*. 2015;45(8):843–849. doi:10.1111/imj.12788

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