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Ethnic differences in risk of renal disease progression amongst young-onset type 2 diabetes in New Zealand

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ABSTRACT

Aim: Māori and Pacific adults in New Zealand (NZ) with type 2 diabetes are at high risk of Diabetic Kidney Disease (DKD). This study assessed whether the same was true in young-onset type 2 diabetes.
Methods: We conducted a secondary analysis of young adults 18–40 years enrolled in a (1994–2018) NZ primary care cohort. DKD risk was classified as minimal or elevated using Urine Albumin-Creatinine Ratio (UACR) and Estimated Glomerular Filtration Rate (eGFR), with hyperfiltration (eGFR $\geq$ 120 mL/min/1.73 m^2) considered an early marker. Logistic regression identified predictors of elevated DKD risk.
Results: Among 2,184 participants (46 % Pacific people, 31 % Māori, 23 % NZ European: 54 % female, mean age 33.9 $\pm$ 4.9 years, mean BMI 38.0 $\pm$ 8.7 kg/m^2, diabetes duration 1.7 years), elevated DKD risk was more common in Pacific People (37.4 %) and Māori (33.5 %) than NZE (23.3 %; $p < 0.001$) with adjusted odds ratio (vs NZE) of 1.96 (95 % CI: 1.50–2.57) and 1.41 (1.06–1.87) respectively. Māori had less risk than Pasifika (odds ratio 0.72 (0.58–0.89)). Independent predictors of DKD risk included ethnicity, triglyceride-HDL ratio, systolic blood pressure, antihypertensive use, and HbA_{1c}: BMI was not significant.
Conclusions: Pacific and Māori with young-onset type 2 diabetes face a disproportionately higher DKD risk.

1. Introduction

The global burden of type 2 diabetes is increasing, with a notable rise among adults under 40 years of age, now termed "young-onset type 2 diabetes" [1–3]. Once considered a disease of older populations, young-onset type 2 diabetes now presents a growing public health concern due to its early onset, prolonged exposure to hyperglycaemia, accelerated risk of long-term complications, including cardiovascular disease, neuropathy, and diabetic kidney disease (DKD) [4,5] and relative resistance to current treatment [6].

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DKD remains one of the most serious microvascular complications, characterized by progressive renal damage that is often asymptomatic in its early stages [7]. Two key biomarkers are used to assess kidney function: the estimated glomerular filtration rate (eGFR), which reflects renal filtration capacity, and the urine albumin-creatinine ratio (UACR), which indicates albuminuria. Elevated UACR often precedes declines in GFR and serves as an early indicator of kidney injury [8,9]. Conversely, glomerular hyperfiltration, elevated GFR, may signal early intraglomerular hemodynamic changes and is associated with progressive renal decline [8]. The combined use of eGFR and UACR offers enhanced risk stratification and supports earlier clinical intervention [8,9].

In New Zealand, type 2 diabetes has reached epidemic proportions, particularly among young adults and Māori and Pacific communities. Māori and Pacific People are disproportionately affected, with complication rates approximately two to three times higher than New Zealand Europeans (NZE) [10,11]. Evidence suggests type 2 diabetes follows a more aggressive course in Māori and Pacific adults [12]; however, it is unclear whether those with young-onset type 2 diabetes from these groups also exhibit greater risk for DKD progression [10,12].

The aim of this study is to assess whether DKD progression risk was higher in Māori and Pacific people with young-onset type 2 diabetes than their NZE counterparts using a composite classification of UACR and eGFR to better reflect overall kidney disease burden.

2. Methods

2.1. Study design

This study involved NZE, Māori, and Pacific young adults, aged 18–40 years, with type 2 diabetes who were enrolled in the Diabetes Care Support Service (DCSS) between 1994 and 2018. The DCSS is a longitudinal primary care diabetes audit program based in South and West Auckland, New Zealand across 217 primary care practitioners (general practitioners). The DCSS contains detailed data on participant demographics, risk factors, clinical measurements, diagnosed diabetes complications, and medications [13]. Data accuracy has been ensured through enumeration assessments and robust internal quality control measures, including regular audits, random and routine sampling, and active data management [14,15].

2.2. Risk factors

Young-onset type 2 diabetes was defined by primary care record coding and validated by trained diabetes auditors [16,17]. Baseline socio-demographic and clinical characteristics included ethnicity, age at diagnosis, duration of type 2 diabetes, body mass index (BMI), smoking status, blood pressure, HbA_{1c}, blood lipids, and treatments for hypertension, diabetes, and antiplatelet/anticoagulants. Socioeconomic position was assessed using the NZDep2013 index from the Department of Public Health, University of Otago (Otago, New Zealand), which categorizes deprivation levels across 5 groups: IMD-1 (least deprived), IMD-2, IMD-3, IMD-4, and IMD-5 (most deprived) [18].

Participants were grouped by self-identified ethnicity: Māori (Indigenous Polynesian), Pacific people (93 % Polynesian, 7 % Melanesian, Micronesian), and NZE. Māori were defined as those with any Māori ancestry, Pacific people as individuals identifying as Samoan, Tongan, Fijian, Niuean, or other Pacific ethnicities (excluding Māori), and NZE as those identifying with European ancestry. This approach ensures statistical power for ethnic comparisons.

2.3. Diabetes definition and classification

Type 2 diabetes was identified using diagnostic coding from general practice electronic medical records within the DCSS. In accordance with prior DCSS publications [13], diagnoses were based on general practitioner and/or hospital-entered Read or ICD-10 codes consistent with type 2 diabetes, with manual case validation undertaken by trained diabetes nurses and audit staff. Individuals with diagnostic codes for type 1 diabetes or other specified diabetes type (e.g., secondary or monogenic) were excluded.

As part of the data validation process, we initially performed a subgroup analysis excluding participants prescribed insulin therapy to assess potential misclassification. However, because there were no significant differences in clinical characteristics between insulin-treated and non–insulin-treated participants, the full cohort was retained for the final analyses.

2.4. Determination of kidney risk

The primary outcome of this study was the risk of progression to DKD, assessed using a composite classification of UACR and eGFR. Following guidelines from the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 clinical practice framework [8], participants were categorized into a 9-cell matrix combining three eGFR categories ($\geq$120, 90–119, and $<$90 mL/min/1.73 m^2) with three UACR categories ($<$3, 3–30, and $>$30 mg/mmol). While sex-specific UACR cut-offs (2.5 mg/mmol for males and 3.5 mg/mmol for females) have been recommended for Māori and Pacific populations to improve risk stratification, we applied the standard KDIGO thresholds across all participants to maintain consistency and comparability within the cohort and with international studies. Importantly, eGFR $\geq$120 mL/min/1.73 m^2 was used as a proxy for glomerular hyperfiltration, which is increasingly recognized as an early marker of DKD, particularly in young individuals with type 2 diabetes [19]. Hyperfiltration reflects increased intraglomerular pressure and is associated with subsequent decline in kidney function, even in the absence of elevated UACR [8,12].

Participants were stratified into two DKD risk grades based on the combined eGFR/UACR matrix:

- **Minimal risk** (green), typically characterized by normal eGFR with low UACR
- **Elevated risk** (orange/red), which includes those with reduced eGFR and/or raised UACR, as well as those with hyperfiltration (eGFR $\geq$ 120 mL/min/1.73 m^2) and elevated UACR

This modified KDIGO heat map (Fig. 1) allows early detection of renal dysfunction across a spectrum of presentations, from subtle albuminuria to hyperfiltration, and facilitates proactive risk stratification and individualized intervention.

For this study, eGFR was recalculated using the CKD-Epi 2021 equation, which has been shown to offer improved performance over the MDRD equation, especially at higher eGFR levels, with greater accuracy and without ethnicity adjustment. The CKD-Epi 2021 equation is as follows:

$$\begin{aligned}\text{eGFR} = 142 \times \min(\text{standardized}\, S_{cr}/\kappa, 1)^{\alpha} \\ \times \max(\text{standardized}\, S_{cr}/\kappa, 1)^{-1.200} \times 0.9938^{\text{age in years}} \\ \times 1.012\ [\text{if female}]\end{aligned}$$

where: S_{cr} = serum creatinine in mg/dL, $\kappa = 0.7$ (females) or 0.9 (males), α = -0.241 (females) or -0.302 (males), min(standardized Scr/K,1) = the minimum of S_{cr}/κ or 1, max(standardized S_{cr}/K,1) = the maximum of S_{cr}/κ or 1.

2.5. Statistical analysis

Descriptive statistics were used to summarize the data: frequencies and percentages for categorical variables and means and standard deviations for continuous variables. Ethnic differences in baseline socio-demographic and clinical characteristics were analysed using Chi-square tests for categorical variables and ANOVA with post-hoc Tukey

DKD Risk Classification		UACR categories (mg/mmol)		
		Normoalbumuria (*UACR < 3*)	Microalbuminuria (*3 ≤ UACR ≤ 30*)	Macroalbuminuria (*UACR > 30*)
eGFR categories (ml/min/1.73m^2)	Normal (*90 ≤ eGFR < 120*)	Minimal Risk	Minimal Risk	Moderate Risk
	Hyperfiltration (*eGFR ≥ 120*)	Minimal Risk	Moderate Risk	High Risk
	Mild-Severe (*eGFR < 90*)	Moderate Risk	High Risk	High Risk

Fig. 1. Modified KDIGO risk grading for DKD based on a composite UACR/eGFR threshold.

tests for continuous variables. Variables with skewed distributions were log-transformed as needed, and geometric means with 95 % confidence intervals were presented for continuous outcomes.

To examine the impact of socio-demographic and clinical factors on DKD risk, we employed a logistic regression with stepwise variable selection to identify significant predictors of DKD risk. The process forced the inclusion of ethnicity as a variable and then systematically evaluated additional demographic and clinical predictors including sex, BMI, deprivation status, smoking status, medication use, age, blood pressure, lipid ratio, and HbA_{1c}. Variables were considered for entry into the model if they demonstrated a statistical significance of $p < 0.30$. After each addition, all variables currently in the model were reassessed, and any that no longer maintained a significance of $p < 0.35$ were removed. These thresholds were selected to allow for the inclusion of potentially meaningful predictors during exploratory analysis, whilst maintaining control over model complexity. This iterative forward–backward selection process continued until no additional variables met the criteria for entry and all included variables remained above the significance threshold for retention.

To evaluate the consistency of DKD risk factors across clinical strata, exploratory subgroup analyses were conducted by obesity status (BMI $\geq$30 kg/m^2 vs <30 kg/m^2) and by antihypertensive prescription (yes vs no). The obesity-stratified models were undertaken to assess whether metabolic predictors such as TG:HDL ratio and HbA_{1c} differed by adiposity level, and to address potential misclassification of diabetes type among lean, insulin-treated individuals. Analyses stratified by antihypertensive use examined whether associations between systolic blood pressure and DKD risk persisted among those under active blood-pressure management. Although BMI was not expected to be a primary determinant of DKD in this cohort, a relationship between obesity and DKD risk has been reported in other populations [20,21,22]; therefore, subgroup analyses by obesity status were conducted to examine whether this association was evident in the DCSS cohort. These subgroup analyses were pre-specified as sensitivity checks to determine whether key predictors of DKD risk were robust across differing metabolic and treatment contexts.

Separate multivariable logistic regression models were fitted within each subgroup, adjusting for the same covariates as in the primary analysis. Heterogeneity was assessed by comparing regression coefficients and confidence intervals across strata. All statistical analyses were performed using SAS version 9.4, and statistical significance was evaluated at a two-sided α level of 0.05, unless otherwise specified.

3. Results

Of the 2757 participants enrolled in the cohort, we excluded those self-identifying into an ethnic group other than NZE, Māori, or Pacific People (n = 573), resulting in 2184 participants being included in the current analysis.

Baseline socio-demographic and clinical characteristics stratified by ethnicity are presented in Tables 1 and 2. Of the 2,184 participants with young-onset type 2 diabetes, the sample comprised of 46 % Pacific people, 31 % Māori, 23 % NZE; 54 % female, with a mean age 33.9 ± 4.9 years. Māori and Pacific People at elevated risk for DKD had higher BMI, lower socioeconomic status, and higher HbA_{1c} compared with NZE. They were also more likely to be prescribed antihypertensive, anti-diabetes, and lipid-lowering medications. Pacific people had lower systolic and diastolic blood pressure compared with Māori. Smoking prevalence was highest among Māori, followed by Pacific people, and lowest among NZE. When stratified by DKD risk, a significantly greater proportion of Pacific people (37.4 %) and Māori (33.5 %) were classified as being at elevated risk for DKD progression compared with NZE (23.3 %; $p < 0.001$). Of those prescribed insulin therapy, 79.6 % were obese (BMI $\geq$ 30 kg/m^2) and 20.4 % were non-obese, suggesting that insulin use predominantly occurred among individuals with obesity and long-standing type 2 diabetes rather than lean individuals with possible type 1 diabetes. Diabetes duration was right-skewed, with many participants recorded as newly diagnosed (median 0.0 years). Participants treated with insulin had a longer duration of diabetes than those not on insulin (median 1.0 [0.0–4.0] vs 0.0 [0.0–1.0] years; Wilcoxon rank-sum $p < 0.0001$). However, the majority of participants in both groups had a recorded duration of 0 years. Among individuals with eGFR $\geq$120 mL/min/1.72 m^2, 43.5 % had normoalbuminuria (UACR <3 mg/mmol), indicating that a proportion of those with hyperfiltration may not be identified using UACR alone.

When examined independently, glomerular hyperfiltration was observed in 18.9 % of participants. Hyperfiltration was significantly more common among Māori and Pacific individuals than NZE ($p < 0.0048$). It was also associated with younger index age, younger age at diabetes diagnosis, lower diastolic blood pressure, lower total cholesterol, lower total and HDL cholesterol, higher UACR, higher HbA_{1c}, lower likelihood of insulin treatments, higher likelihood of anti-lipid treatment, and a higher likelihood of antihypertensive treatment (all $p < 0.05$).

Table 3 presents the results of a stepwise logistic regression analysis examining independent predictors of elevated DKD risk among young-onset type 2 diabetes participants. Ethnicity emerged as a significant predictor, with Pacific People showing a markedly elevated risk compared to NZE (odds ratio [OR] 1.96, 95 % CI 1.50–2.57; $p < 0.001$), as did Māori (OR 1.41, 95 % CI 1.06–1.87; $p = 0.017$). However, Māori had a lower risk than Pacific people (OR 0.72, 95 % CI 0.58–0.89; $p = 0.003$). Systolic blood pressure (SBP) was another significant factor; each 10 mmHg increase in SBP was associated with a statistically significant increase in the risk of DKD progression (OR 1.10, 95 % CI: 1.00–1.22, $p < 0.001$). A higher triglyceride-to-HDL (TG:HDL) cholesterol ratio had a strong association (OR 1.21, 95 % CI: 1.13–1.31, $p < 0.001$), as did hyperglycaemia, with higher HbA_{1c} significantly increasing DKD risk (OR 1.21, 95 % CI: 1.09–1.33, $p < 0.001$). The use of antihypertensive medications was also associated with increased odds of DKD risk (OR 1.74, 95 % CI: 1.35–2.26, $p < 0.001$), likely reflecting both

Table 1
Baseline demographic and metabolic characteristics of DCSS young adult participants – continuous variables (N = 2184).

Characteristic	Overall (N = 2184)	NZE (n = 497)	Māori (n = 675)	Pacific People (n = 1012)	*p*
Age at Enrolment, Years	33.9 ± 4.9	34.3 ± 4.6	33.6 ± 5.2	33.9 ± 5.0	0.026[a]
Age at Type 2 Diabetes Diagnosis, Years	32.2 ± 5.5	32.8 ± 5.4	31.7 ± 5.8	32.2 ± 5.4	0.001[a]
Duration of Type 2 Diabetes*, Years	2.5 (2.3–17.9)	2.4 (2.1–18.0)	2.7 (2.4–19.8)	2.4 (2.2–17.4)	0.100
Body Mass Index, kg/m^2	38.0 ± 8.7	35.6 ± 8.2	38.8 ± 8.6	38.8 ± 8.7	<0.001[a,b]
Blood Pressure, mmHg					
Systolic	127.8 ± 15.7	129.3 ± 14.5	128.5 ± 17.0	126.5 ± 15.4	0.002[b,c]
Diastolic	82.2 ± 11.0	81.8 ± 10.0	83.2 ± 11.8	81.7 ± 10.9	0.015[c]
Lipids, mmol/l					
Total Cholesterol (TC)	5.1 ± 1.2	5.1 ± 1.3	5.2 ± 1.2	5.0 ± 1.2	0.092
Low-Density Lipoprotein (LDL)	2.8 ± 1.1	2.7 ± 1.0	2.8 ± 1.2	2.8 ± 1.1	0.391
High-Density Lipoprotein (HDL)	1.1 ± 0.3	1.1 ± 0.4	1.1 ± 0.3	1.1 ± 0.3	<0.001[a,c]
Triglyceride (TG) *	2.1 (1.6–16.9)	1.9 (1.8–14.0)	2.5 (2.4–18.2)	1.9 (1.9–13.9)	<0.001[a,c]
TG:HDL*	1.9 (1.9–13.9)	1.8 (1.6–12.9)	2.4 (2.3–17.6)	1.7 (1.7–12.6)	<0.001[a,c]
HbA_{1c}, %	8.5 ± 2.1	7.6 ± 1.9	8.6 ± 2.1	8.8 ± 2.2	<0.001[a,b]
HbA_{1c}, mmol/mol	69 ± 23	60 ± 21	71 ± 23	72 ± 24	<0.001[a,b]
UACR*, mg/mmol	3.3 (3.1–24.4)	1.3 (1.2–10.2)	3.9 (3.5–29.7)	4.6 (4.2–34.5)	<0.001[a,b]
eGFR, mL/min/1.73 m^2	107.0 ± 18.3	106.3 ± 17.5	108.3 ± 17.6	106.6 ± 19.0	0.105

Data presented as mean ± SD.
Post-hoc Tukey comparisons were performed when an overall difference was found ($p < 0.05$) and indicated with different letter superscripts: [a]NZE vs. Maori; [b]NZE vs. Pacific People; [c]Maori vs. Pacific People.
*Geometric means calculated from log-transformed data and back-transformed. 95% confidence intervals derived from standard error of the mean of log values.

treatment need and underlying risk. While age showed a weak inverse relationship with DKD risk (OR 0.98, 95 % CI: 0.97–1.00, $p = 0.087$), it did not reach statistical significance. Notably, body mass index (BMI) was not significantly associated with DKD risk and was excluded from the final model.

In the subgroup analyses stratified by obesity status (obese vs. non-obese) and antihypertensive prescription (prescribed vs. not prescribed) (supplemental Tables 1 and 2), the associations for TG:HDL ratio and HbA_{1c} were directionally consistent across all subgroups, though effect sizes were slightly attenuated and statistical significance varied. Among participants with obesity, DKD risk was independently associated with Pacific and Māori ethnicity, higher TG:HDL ratio, systolic blood pressure, HbA_{1c}, and use of antihypertensive therapy. In contrast, among non-obese participants, TG:HDL ratio was the only significant predictor of DKD risk.

4. Discussion

This study is the first to investigate the potential of combining eGFR and UACR as a tool for assessing DKD risk in young-onset type 2 diabetes, particularly within high-risk groups such as Māori and Pacific populations. We demonstrated that DKD risk is already prevalent among young-onset type 2 diabetes in New Zealand, affecting between 23 % and 37 % of individuals depending on ethnicity, with the highest burden observed among Pacific People, followed by Māori and NZE. Beyond ethnicity, key associations with increased DKD risk included higher TG:HDL cholesterol ratio, elevated SBP, antihypertensive medication use, and higher HbA_{1c}. BMI, in contrast, was not significantly associated, underscoring the limited utility of BMI alone in risk stratification for DKD within this population.

Previous studies examining DKD in young adults have largely focused on albuminuria or reduced eGFR, not their combination, as markers of kidney damage, often underestimating early renal changes [23,24]. However, emerging evidence suggests that hyperfiltration, a state of elevated GFR thought to represent early renal dysfunction, may be an important early marker of DKD, particularly in youth-onset type 2 diabetes [25,26]. Hyperfiltration has been identified as a precursor to subsequent eGFR decline and albuminuria, especially among individuals with poor glycaemic control and metabolic dysregulation [25,27]. Given this, the inclusion of hyperfiltration in our definition of DKD risk is both conceptually and empirically supported [28]. This broader definition allows for earlier detection of at-risk individuals and may enhance clinical decision-making around targeted interventions.

Although individuals with eGFR ≥120 mL/min/1.73 m^2 were included within the elevated DKD risk group, hyperfiltration was not examined as a separate outcome in this analysis. Nonetheless, a notable proportion of these individuals had normoalbuminuria, suggesting that elevated eGFR may identify individuals at risk for DKD who are not detected by albuminuria alone. However, as eGFR is a derived measure with known limitations, particularly in younger, ethnically diverse populations, these observations should be interpreted cautiously. The apparent hyperfiltration may reflect early renal risk but could also arise from inaccuracies in the estimating equations used in this population and the challenges of defining thresholds within a continuous variable.

Independent analysis demonstrated that hyperfiltration was more prevalent among Māori and Pacific individuals and among those with higher BMI, elevated UACR as well as in those receiving antihypertensive or insulin therapy. These findings suggest that glomerular hyperfiltration in young-onset type 2 diabetes may reflect a combination of metabolic and haemodynamic stressors rather than isolated glycaemic exposure. While hyperfiltration is not currently part of the formal DKD definition, its association with these risk factors, particularly among high-risk ethnic groups, supports its value as an early physiological indicator of renal vulnerability [29]. Together, these metabolic and haemodynamic pathways provide a biological basis for early renal injury; however, the markedly higher prevalence of DKD among Māori and Pacific peoples suggests that additional factors, such as genetic predisposition and potentially inequities in healthcare access, also contribute to this disparity.

Both HbA_{1c} and TG:HDL ratio were independently associated with elevated DKD risk, indicating that hyperglycaemia and insulin resistance represent parallel yet interrelated pathways to kidney injury in young-onset type 2 diabetes [12,30,31]. The TG:HDL ratio serves as a surrogate marker of insulin resistance and atherogenic dyslipidaemia [31], while HbA_{1c} reflects chronic glycaemic exposure [12,30]. In the setting of relative insulin deficiency, hyperglycaemia promotes hepatic triglyceride synthesis through increased counter-regulatory hormone activity, linking glucotoxicity and lipotoxicity within a shared pathogenic framework [12,30]. These findings suggest that metabolic dysfunction in young-onset type 2 diabetes operates along both glucose- and lipid-

Table 2
Baseline demographic and metabolic characteristics of DCSS young adult participants – categorical variables (N = 2184).

Characteristic	Overall (N = 2184)	NZE (n = 497)	Māori (n = 675)	Pacific people (n = 1012)	*p*
Sex: Female	1184 (54.2)	227 (45.7)	358 (53.0)	599 (59.2)	<0.001
BMI: Obese (≥30)	1727 (79.1)	376 (75.7)	590 (87.4)	882 (87.2)	<0.001
NZDep13 Scale 9 or 10 (Most Deprived)	1146 (52.5)	108 (21.7)	389 (57.6)	649 (64.1)	<0.001
Smoking Status: Current- or Ex-Smoker	923 (42.3)	153 (30.8)	383 (56.7)	387 (38.2)	<0.001
UACR, mg/mmol					
Normoalbuminuria (<3)	1189 (54.4)	379 (76.3)	342 (50.7)	468 (46.2)	<0.001
Microalbuminuria (3–30)	728 (33.3)	104 (20.9)	247 (36.6)	377 (37.3)	
Macroalbuminuria (>30)	267 (12.2)	14 (2.8)	86 (12.7)	167 (16.5)	
eGFR, mL/min/1.73 m^2					
Hyperfiltration (≥120)	412 (18.9)	68 (13.7)	157 (23.3)	187 (18.5)	<0.001
Normal (90–119)	1417 (64.9)	345 (69.4)	422 (62.5)	650 (64.2)	
Mildly-Severely Decreased (<90)	355 (16.3)	84 (16.9)	96 (14.2)	175 (17.3)	
Risk of DKD					
Minimal	1464 (67.0)	381 (76.7)	449 (66.5)	634 (62.7)	<0.001
Moderate/High	720 (33.0)	116 (23.3)	226 (33.5)	378 (37.4)	
Prescribed Antihypertensive Treatment: Yes	1508 (69.1)	302 (60.8)	499 (73.9)	707 (69.9)	<0.001
Prescribed Insulin Treatment: Yes	764 (35.0)	174 (35.0)	252 (37.3)	338 (33.4)	0.252
Prescribed Antidiabetes Medication: Yes	2016 (92.3)	439 (88.3)	629 (93.2)	948 (93.7)	<0.001
Prescribed Antilipid Medication: Yes	1383 (63.3)	303 (61.0)	472 (69.9)	608 (60.1)	<0.001

Data presented as n (%).

Table 3
Stepwise logistic regression analysis of factors associated with elevated DKD risk.

Outcome: DKD risk	Regression coefficient	χ^2	*p*	Odds ratio (95 % CI)
Intercept	−2.769	24.824	<0.001	–
Prescribed Antihypertensives: Yes vs. No	0.687	35.736	<0.001	1.99 (1.59–2.50)
Ethnicity: Pacific People vs. NZE	0.674	24.249	<0.001	1.96 (1.50–2.57)
Ethnicity: Māori vs. NZE	0.343	5.688	0.017	1.41 (1.06–1.87)
Ethnicity: Māori vs. Pacific People	−0.331	9.057	0.003	0.72 (0.58–0.89)
TG:HDL Ratio (natural log, per 1-unit)	0.297	19.453	<0.001	1.35 (1.18–1.54)
SBP, per 10 mmHg	0.100	11.848	0.001	1.11 (1.05–1.22)
HbA_{1c}, per 1 % ≈ 10 mmol/mol	0.051	4.988	0.026	1.05 (1.01–1.10)
Index Age, per year	−0.016	2.927	0.087	0.98 (0.97–1.00)
NZDep13: Most Deprived vs. All Others	−0.130	1.714	0.191	0.88 (0.72–1.07)
Prescribed Antidiabetes: Yes vs. No	−0.230	1.506	0.220	0.80 (0.55–1.15)

Variables significant in univariate analyses (ethnicity, sex, BMI, socioeconomic position, smoking status, etc.) were included in the stepwise logistic regression model.
HbA_{1c} expressed per 1 % (≈ 10 mmol/mol); SBP rescaled to 10 mmHg increments; TG:HDL ratio natural log-transformed, OR reflects change per 1-unit increase in ln(TG:HDL).

mediated axes, each amplifying renal microvascular injury. This dual mechanism may explain why DKD risk was greatest among participants with poor glycaemic control and elevated TG:HDL ratios, reinforcing the importance of addressing both dyslipidaemia and hyperglycaemia in early, multifactorial prevention strategies.

A subgroup analysis of insulin-treated participants indicated that nearly four in five were obese, supporting that insulin use in this cohort likely reflects more advanced disease and β-cell dysfunction within the context of insulin resistance rather than type 1 diabetes misclassification. Although diabetes duration was statistically longer among insulin-treated participants, most individuals in both groups had a recorded duration of 0 years (diagnosed at or near baseline), so this difference is probably of limited clinical relevance. The coexistence of insulin resistance and declining beta-cell capacity in young-onset type 2 diabetes may explain the higher HbA_{1c} and consequent DKD risk observed among those requiring insulin therapy.

The higher DKD risk observed among Māori and Pacific peoples likely reflects a complex interplay of biological, environmental, and systemic factors. Genetic influences, including variation in renal sodium transport, insulin signalling, and inflammatory pathways, may predispose these groups to early glomerular injury and metabolic dysfunction [32,33]. Disparities in healthcare access have been shown elsewhere, such as later diabetes diagnosis, and reduced access to culturally appropriate care, which could also contribute to delayed detection and suboptimal management of risk factors. Addressing these disparities will require both clinical and public health interventions that integrate culturally grounded models of care and targeted early screening strategies [34,35].

Given these complexities, a more comprehensive and targeted approach to managing DKD risk is required. The combination of eGFR and UACR as a composite measure offers significant promise for early detection and more effective risk stratification. This approach enables healthcare providers to identify individuals at higher risk for progressive renal disease and tailor interventions, accordingly, improving clinical outcomes. Early identification and management of renal impairment are crucial, particularly in young adults, who are at risk for prolonged disease duration and subsequent complications like DKD. By stratifying risk using eGFR/UACR, timely interventions can prevent DKD progression, enhance quality of life, and reduce healthcare costs associated with advanced renal disease.

The lack of association between BMI and DKD risk in our study has also been shown in other research [28,36], and this finding may be mediated through ethnic differences in body composition. Māori and Pacific people typically have a higher proportion of lean muscle mass relative to body fat when compared to other ethnic groups [37]. While BMI can serve as a general indicator of obesity-related risk, it does not distinguish between muscle and fat mass and may overestimate obesity-related risk in these populations. Furthermore, Māori and Pacific people often develop cardiometabolic risk factors such as hypertension, dyslipidaemia, and hyperglycaemia at younger ages or lower BMI thresholds, reducing BMI's predictive power in isolation [38]. Socioeconomic factors, including limited access to healthcare, healthy nutrition, and physical activity opportunities, also contribute substantially to DKD risk, overshadowing BMI's influence [34].

The consistency of associations across several clinically relevant subgroups lends support to the robustness of our primary findings. Specifically, the direction and magnitude of associations with TG:HDL ratio and HbA_{1c} remained generally stable across obesity and

antihypertensive strata. However, subgroup analyses revealed potential effect modification for some predictors likely reflecting the differences in sample size and power. The relationship between Pacific ethnicity and DKD risk, while strong in the overall cohort, appeared attenuated in non-obese and untreated individuals. Likewise, SBP demonstrated a significant association only in subgroups with obesity or antihypertensive use. These variations highlight the importance of considering subgroup-specific risk profiles in young-onset type 2 diabetes and suggest that certain risk factors may operate differently across clinical contexts. While HbA_{1c}, SBP, and dyslipidaemia are established DKD risk factors, their convergence in young-onset Pacific People with type 2 diabetes suggests an urgent need for earlier and more tailored invention strategies in the high-risk group.

These findings align with prior studies emphasising the influence of ethnic and cardiometabolic factors on kidney disease progression in diabetes [7,12,39]. In particular, disparities in DKD risk among Pacific and Māori populations have been attributed to both clinical and social determinants of health[40,41] and the treatment status have been increasingly recognised [42].The elevated risk of DKD observed among Māori and Pacific participants appears to be driven, in part, by a greater burden of metabolic and clinical risk factors. These factors are often interrelated and compounded by socioeconomic disadvantage, and earlier onset of cardiometabolic conditions in Māori and Pacific peoples [43]. Collectively, these findings highlight the need for early, culturally responsive interventions that address dyslipidaemia, hypertension, and hyperglycaemia to reduce DKD burden in these high-risk populations.

The strengths of the eGFR/UACR composite variable represents an important tool for enhancing early detection, risk assessment, and intervention strategies for DKD, especially in high-risk populations like young Māori and Pacific People. With further validation and refinement, this approach could lead to more effective, culturally appropriate healthcare practices and improved outcomes for communities disproportionately affected by diabetes-related kidney disease.

While the eGFR/UACR composite measure holds considerable potential, several limitations should be considered. The generalisability of these findings may be limited to Māori and Pacific people, as the biological and environmental/sociocultural factors influencing kidney disease could differ across ethnic groups, potentially reducing the broader applicability of this approach [44]. Additionally, although this composite measure shows promise, further validation is needed to confirm its reliability and clinical utility across diverse patient groups, particularly the inclusion of hyperfiltration from an estimate of GFR. Variability in measurement techniques, such as laboratory protocols and biological fluctuations in eGFR and UACR, could impact the accuracy of this composite variable. Furthermore, the study's cross-sectional design limits our ability to establish causal relationships or track the progression of DKD over time. Longitudinal studies are necessary to better understand how the eGFR/UACR composite variable performs in predicting long-term kidney function decline and DKD progression. Another limitation is the potential for diagnostic misclassification within primary-care and hospital datasets, including the inadvertent inclusion of individuals with type 1 diabetes and monogenic diabetes [45]. Such errors are inherent to studies relying on routinely collected clinical data and may introduce a small degree of uncertainty into phenotype categorisation. The dataset also did not include information on specific classes of antihypertensive agents, precluding assessment of renal-protective medications such as angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs). While their inclusion may have been of interest, there would have been a problem of confounding by indication, as individuals at higher renal risk are more likely to be prescribed these therapies.

The absence of detailed lifestyle data, such as dietary intake (e.g., salt and protein consumption) and physical activity levels, were not routinely captured in the DCSS database. These factors influence blood pressure, glycaemia, body weight, oxidative stress, and endothelial function, and are known contributors to DKD development and progression [46–48]. Incorporating lifestyle measures in future risk assessments may help identify modifiable pathways and guide preventive interventions to reduce DKD risk, particularly among young adults with type 2 diabetes.

These findings suggest that routine inclusion of eGFR and TG:HDL ratio alongside HbA_{1c} and blood pressure in early risk stratification may improve early DKD risk detection in young-onset type 2 diabetes. Identifying individuals with hyperfiltration and/or dyslipidaemia, even in the absence of albuminuria, could support earlier initiation of reno-protective interventions, such as Sodium-Glucose Transport Protein 2 (SGLT2) inhibitors or Renin-Angiotensin System (RAS) blockade, tailored to ethnic risk profiles.

Based on these findings, we propose a pragmatic screening framework for early DKD risk stratification in young-onset type 2 diabetes. Key markers such as HbA_{1c}, SBP, and TG:HDL ratio should be monitored regularly from diagnosis, with early intervention thresholds adjusted for ethnicity and treatment history. Pacific individuals and those with obesity or hypertension may benefit from more aggressive early screening for microalbuminuria, CKD and hyperfiltration. Importantly, individuals with hyperfiltration (eGFR $\geq$120 mL/min/1.73 m^2), even in the absence of albuminuria, may warrant earlier consideration of reno-protective therapies (e.g., RAS blockade or SGLT2 inhibitors), given their elevated DKD risk. This approach may aid clinicians in identifying high-risk individuals earlier, when kidney function is still preserved and interventions are most effective.

In conclusion, this cohort study underscores the heightened risk of DKD among Māori and Pacific People compared to New Zealand Europeans, as well as the importance in considering both clinical and sociodemographic factors, highlighting the need for targeted interventions aimed at improving health outcomes for these populations, particularly among Māori and Pacific People under 40 years of age in New Zealand. Further research is warranted to explore the underlying causes of these disparities and develop tailored strategies for these high-risk groups.

5. Guarantor

David Simmons is the guarantor of this work and, as such, had full access to all data and takes responsibility for the integrity of the data and accuracy of the analysis. All authors reviewed and approved the final manuscript.

CRediT authorship contribution statement

Kanchana Perera: Writing – original draft, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **John Baker:** Writing – review & editing, Visualization, Funding acquisition. **Kalpa Jayanatha:** Writing – review & editing, Visualization. **Karen Pickering:** Writing – review & editing, Visualization, Funding acquisition. **Richard Cutfield:** Writing – review & editing, Visualization, Funding acquisition. **Brandon Orr-Walker:** Writing – review & editing, Visualization, Funding acquisition. **Gerhard Sundborn:** Writing – review & editing, Visualization. **Andrew Heroy:** Writing – review & editing, Software, Formal analysis. **Thomas Arnold ScM:** . **Dahai Yu:** Writing – review & editing, Resources, Data curation. **David Simmons:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2025.113018.

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