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Association between within-target risk factors and life expectancy free from cardiovascular disease, cancer, and dementia in individuals with type 2 diabetes in New Zealand between 1994 and 2018: a multi-ethnic cohort study

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Abstract

Background The extent to which type 2 diabetes (T2D) reduces life expectancy depends on the risk of complications. We aimed to characterise the relationship between risk factors for diabetes complications and life expectancy, in individuals with T2D, free from major chronic disease, in a regional database linked with national New Zealand health databases.

Methods A prospective cohort study design was employed, analysing data from individuals with T2D drawn from the comprehensive Diabetes Care Support Service database (1994–2018). Participants with known values for all five within-target risk factors (WTRF +) including blood pressure, glycaemia, and LDL cholesterol, alongside being non-smoking with normal renal function at baseline, were included. Life expectancy free from cardiovascular disease (CVD), cancer, and dementia at age 50 years was estimated using multistate life tables, adjusting for demographics and clinical metrics.

Results Women and men with no WTRF + at enrolment had a life expectancy free from CVD, cancer, or dementia of 13.1 (95% confidence interval: 9.1–19.0) and 11.2 (6.7–18.6) years, respectively. For the most socioeconomically deprived groups or Māori with no WTRF + at baseline, life expectancies were markedly lower. Life expectancies were 23.7 (18.9–29.7) years for women and 23.2 years (17.2–31.4) years for men with four or five WTRF + at baseline, respectively. While an increasing number of WTRF + at baseline was significantly associated with improved outcomes

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in men and certain subgroups (e.g. Other ethnicity group), this trend was not statistically significant for women overall or in most subgroups.

Conclusions Having multiple WTRF + at baseline is associated with a considerable increase in life expectancy free from major chronic disease among individuals with T2D. This highlights the importance of lifestyle and clinical interventions in the management of T2D and in the prevention and management of associated chronic conditions.

Keywords Type 2 diabetes, Cardiovascular disease, Cancer, Dementia, Life expectancy, Ethnic disparity, Socioeconomic inequality, Māori, New Zealand, Healthy life expectancy

Background

Over the past few decades, global average life expectancy has witnessed a remarkable increase [1]. However, this rise in longevity has coincided with an increasing prevalence of chronic conditions alongside a growing ageing population, particularly in the context of type 2 diabetes (T2D) [2]. This population shift has led to a significant burden from chronic disease such as cardiovascular disease (CVD), cancer, and dementia [3]. While individuals are now living longer, many older adults, especially those with T2D, are facing the challenges of disability and chronic health issues [4]. Among these chronic conditions, cancer, CVD, and dementia have been found to substantially curtail life expectancy. Research estimates indicate that the reduction in years of life due to these ailments ranges from 7.5 to 20 years in the general population [3], and 10 to 20 years in the population with T2D, contingent on study methodologies and population characteristics [5].

Risk factors for T2D complications such as smoking, hyperglycaemia, overweight and obesity, dyslipidaemia, high blood pressure (BP), and chronic kidney disease (CKD) influence both overall life expectancy and the age of onset of associated chronic disease [6–8]. Previous investigations have demonstrated that these factors are responsible for a significant proportion of premature deaths, accounting for 10–50% of early mortality and a loss of 5–20 years in life expectancy within the population with T2D [9]. Despite these findings, limited attention has been directed towards understanding how various combinations of multiple risk factors for diabetes complications may interact to influence life expectancy devoid of the major diseases—dementia, CVD, and cancer—particularly in individuals with T2D. The significance of estimating life expectancy without chronic disease lies in its encapsulation of both quality of life and mortality risk, serving as an invaluable metric for healthcare professionals, the wider public, and policymakers. Moreover, such estimates facilitate more precise predictions of future healthcare expenditure and aid in devising strategic healthcare plans. This study aims to explore the impact of risk factors for complications on healthy life expectancy (HLE) without cancer, CVD, and dementia using a comprehensive dataset from the DCSS primary care system, linked with national databases in New Zealand. By delving into the relationship between risk factors and disease-free HLE, this research contributes to a more informed understanding of health outcomes for individuals with T2D and offers insights into potential interventions to enhance their quality of life.

Methods

Data setting

The study utilised a prospective cohort drawn from the Diabetes Care Support Service (DCSS), which originated as part of a primary care audit programme established in 1991 [10]. DCSS commenced its operation of auditing diabetes and associated management practices across general practices in West, East, and South Auckland, New Zealand, after an initial period spanning from 1 January 1991 to 31 December 1993, and continued until 31 July 2018 [11, 12]. This database was combined with national databases that included data on socioeconomic status, pharmaceutical claims, cancer registration, death registrations, and hospital admissions. The anonymised dataset encompassed demographic details, lifestyle factors (such as smoking status), anthropometric measures (including body mass index (BMI)), routine diabetes clinical metrics (like diabetes duration, blood pressure (BP), haemoglobin A1c (HbA1c), and lipid profiles), and information on routine medications for diabetes patients (covering treatments such as anti-diabetes drugs, statins, antihypertensives, anticoagulants, and/or antiplatelet medications). Data integrity was maintained through rigorous quality control methods, including internal checks, regular cross-verification by DCSS auditors, systematic and random data entry reviews, and proactive data management practices [13]. The national pharmaceutical claim database, which comprised every prescription dispensed to patients, was corroborated with the recorded prescription data in the DCSS database, although it was only fully integrated post-2006 due to the prior lack of universal National Health Index Numbers.

The DCSS received ethical approval from the North Health Ethics Committee in 1992 for research purposes and subsequently for ongoing audits in 1996.

Additionally, the New Zealand Health and Disability Ethics Committee exempted the study from ethics review since it solely used anonymised data for analysis. Every participating general practice provided written consent through their authorised representatives to participate in the DCSS. The New Zealand Ministry of Health authorised access to the DCSS and its associated linked databases for this research.

Study population

Participants aged 18 years and older diagnosed with T2D were included. The date of their initial registration in the DCSS database was established as the point of enrolment, which served as the baseline for this study. Exclusion criteria for the study encompassed patients who had experienced any of the specified health outcomes (including mortality) prior to their enrolment date or within the first 12 months after their enrolment, as a measure to minimise possible information bias. Participants were enrolled in the cohort at various ages (Supplemental Fig. 1), and the follow-up period was defined from the time of enrolment to death or until December 31, 2019, for those who did not die within the study period.

Within-target risk factors, smoking and CKD (WTRF+)

The current study examined within-target risk factors (WTRF+) at enrolment including optimal blood pressure (≤130 mmHg systolic) [14], controlled blood glycaemia (HbA1c < 64 mmol/mol [8%]) [15], low-density lipoprotein (LDL) cholesterol (< 1.4 mmol/L [54.1 mg/dl]) [16], non-smoking, and normal kidney function (eGFR ≥ 90 ml/min/1.73 m^2) calculated using the CKD-EPI equation [17]. Participants were categorised at baseline based on the number of WTRF+, with 0 to 5 of possible factors. Those with 4 or 5 factors were combined into the highest category due to small sample sizes. Once categorised, participants remained in their initial category throughout the study period. We use the term WTRF+ at baseline to refer to clinically relevant measurements that align with management targets for individuals with T2D. These targets are distinct from the preventive thresholds set for the general population and are based on established guidelines for managing T2D. The study did not explore BMI < 25 kg/m^2 due to a high baseline prevalence of obesity (60%: globally, most people with T2D are either overweight or obese) [18] and previous findings of a stable average BMI > 33 kg/m^2 over 5 years [19]. Incorporating non-obesity or non-overweight would significantly reduce the population size (particular the group with multiple factors) for analysis and limit statistical power. Similarly, stricter glycaemic target criteria (HbA1c < 53 mmol/mol/7.0%) were not applied, aligning with previous observations of stable HbA1c levels around 60 mmol/mol/7.6% over time [19].

Outcomes

Deaths were identified using records from the national death registration database following enrolment. CVD and dementia were recognised as clinical incidents recorded in outpatient, inpatient, and death databases, identified by primary ICD-9 and ICD-10 codes post-enrolment (Additional file 1: Table S1) [12]. Similarly, cancer incidents were documented in the national cancer registry and healthcare databases using the same coding system.

Ethnicity and socioeconomic status measurement

Patients' self-reported ethnicity was used and grouped into 4 categories (New Zealand European (NZE), Māori, Pasifika, and other) using a prioritisation 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. Those individuals with only European ancestry (from anywhere in the European continent) were defined as NZE. Individuals not included in the above 3 ethnic categories were defined as the "Other ethnicity" group.

The socioeconomic status of patients was determined using the NZDep2013 Index of Deprivation, a tool measuring area-based deprivation. The NZDep2013 assigns an index of multiple deprivation (IMD) score to each meshblock in New Zealand, which are small geographical units or neighbourhoods typically comprising about 81 people. This scoring system rates deprivation on a scale from 1 to 10, where 1 represents the least deprivation and 10 the most. For the purposes of this study, the IMD scores were reclassified into five distinct categories for analytical robustness: IMD-1 representing the least deprivation (encompassing scores 1–2 from NZDep2013), followed by IMD-2, IMD-3, and IMD-4 (which group scores 3–4, 5–6, and 7–8 from NZDep2013, respectively), and IMD-5 indicating the highest level of deprivation (covering scores 9–10 from NZDep2013) [20]. In our previous work, NZDep2001, NZDep2006, and NZDep2013 scores were compared at the patient level to observe shifts in socioeconomic status over time. Consistent stability in socioeconomic status was observed using NZDep2013, which is why this index was chosen for our analyses [10].

Statistical analysis

Continuous variables were compared across the number of WTRF+ at baseline by ANOVA after meeting the requirements of normality and homogeneity of

variance. For those whose raw values did not meet the requirements, log transformation was attempted until the requirements were met. Categorical variables across WTRF + at baseline were tested by chi-square. Our approach involved utilising multistate life tables based on the NZ population to ascertain the variations in HLE and the duration of life free from major chronic illnesses for each risk factor and the aggregate score of these factors. Additionally, we tested for a trend in the estimations of HLE by the increase in the number of WTRF + at baseline using linear trend analysis. The null hypothesis for this analysis was that there is no relationship between the number of WTRF + at baseline and HLE. *P* values for linear trends were calculated to determine if there was a significant trend in HLE with increasing numbers of WTRF + at baseline. Participants were enrolled in the cohort across various ages (Additional file 1: Fig. S1), and risk factors and covariates were evaluated at the time of enrolment [7]. Age at enrolment was modelled as specified above. Only participants who did not have any of the three specified outcome conditions or death within 12 months of enrolment were included. This exclusion criterion helped avoid information bias. Given the commonly used benchmark in life expectancy studies, these life tables began at age 50 years and ended at age 85 years from the multistate model, adjusting for factors including age, sex, ethnicity, IMD, enrolment year, and several health metrics and treatments. Age at enrolment was adjusted rather than other specific age during the follow-up. Using age 50 years as the starting point allowed for meaningful midlife health assessments and aligns with standard epidemiological practice, as seen in previous studies [7]. The multistate models for HLE after age 50 years had three conditions (cancer, CVD, and dementia) and three state transitions. This involved considering three health states (free of condition, condition presence, and death) and three state transitions (from a non-condition state to condition occurrence, from a non-condition state to mortality in those without major chronic conditions, and from the point of condition diagnosis to mortality in affected individuals). We then constructed multistate life tables to account for the transition probabilities for each number of WTRF + at baseline [4].

Four distinct multistate life tables were created: one for a combination of cancer, CVD, and dementia, and three for each condition separately. Initially, we determined the overall transition rates for three shifts: mortality without any of the specified conditions, incidence of the specified conditions, and mortality in participants with cancer, CVD, or dementia. We only acknowledged the initial occurrence of a state, disallowing any subsequent condition or reversal of states (Additional file 1: Fig. S2). Next, we determined hazard ratios to evaluate the association between the number of WTRF + at baseline and the transitions using Cox proportional hazards analysis. Separate time-varying Cox proportional hazards models were utilised to analyse the risks associated with mortality without condition, condition incidence, and mortality in participants with cancer, CVD, and dementia. Furthermore, we calculated the proportions of WTRF + at baseline within the sub-study population for each transition. Finally, combining the hazard ratios with the general transition rates and proportions of WTRF + at baseline in the multistate life tables, we computed the overall HLE, as well as HLE without one specific condition but with either of the other two, for each WTRF + at baseline category.

Regarding missing data, which varied from 0 to 6% for different covariates, we addressed this by generating six imputed datasets using multiple imputation with chained equations, assuming a worst-case scenario where 6% of cohort members had at least one missing covariate. Confidence intervals for HLE estimates were calculated using Monte Carlo simulation (parametric bootstrapping) with 10,000 runs. Additional sensitivity analyses were conducted, including adjusted estimations in sub-populations such as ethnic-specific groups and populations segmented by IMD level. All statistical analyses were executed using Stata 18.0 (StataCorp, College Station, TX, USA), with a threshold for statistical significance set at a two-tailed *P* value of less than 0.05.

Results

Table 1 presents the baseline characteristics categorised by the number of WTRF + at baseline present, ranging from zero to four or five. The number of WTRF + at baseline decreased (on average) with age at baseline, younger patients tended to have with more WTRF + at baseline compared to older patients. The number of WTRF + at baseline varied significantly across ethnic groups; NZE had a higher average number of WTRF + (four or five) at baseline, while Māori and Pacific Islands people were more frequently found in the lower range (zero to two WTRF + at baseline). The number of WTRF + at baseline decreased as the level of deprivation increased; a higher proportion of patients with zero WTRF + at baseline were in the most deprived IMD category (IMD-5), compared to those with more WTRF + at baseline. Additionally, the treatment profile showed that patients with fewer WTRF + at baseline were more likely to use anti-diabetes medications, including insulin, while those with more WTRF + at baseline were more likely to be treated with oral anti-diabetes drugs alone.

Women with T2D and 0 WTRF + at enrolment aged 50 years had an HLE of 13.1 (9.1–19.0) years, while those with three WTRF + at baseline had an HLE of

Table 1 Characteristics of patients with type 2 diabetes at baseline according to number of within-target risk factors at baseline in New Zealand between 1994-2018

	Number of within-target risk factors					
	Zero (n=1,101)	**One (n=8,537)**	**Two (n=18,196)**	**Three (n=10,958)**	**Four/Five (n=3,539)**	***P*-value**
Age, years	56.6 (11.4)	58.1 (12.9)	57.5 (13.7)	54.6 (13.6)	51.4 (13.5)	<0.0001
Female, n (%)	464 (42.1)	4012 (47.0)	9058 (49.8)	5153 (47.0)	1697 (48.0)	<0.0001
Ethnicity, n (%)						
New Zealand European	360 (32.7)	3269 (38.3)	6998 (38.5)	3835 (35.0)	956 (27.0)	<0.0001
Māori	317 (28.8)	1724 (20.2)	2801 (15.4)	1424 (13.0)	370 (10.5)	
Pacific Islanders	323 (29.3)	2384 (27.9)	4890 (26.9)	2869 (26.2)	911 (25.7)	
Other Ethnic group	101 (9.2)	1160 (13.6)	3507 (19.3)	2830 (25.8)	1302 (36.8)	
Enrol cohort, n (%)						
1994-1998	449 (40.8)	3092 (36.2)	5411 (29.7)	1931 (17.6)	352 (10.0)	<0.0001
1999-2003	229 (20.8)	1820 (21.3)	5031 (27.7)	2968 (27.1)	882 (24.9)	
2004-2008	128 (11.6)	1319 (15.5)	2880 (15.8)	2037 (18.6)	648 (18.3)	
2009-2013	185 (16.8)	1527 (17.9)	3361 (18.5)	2899 (26.5)	1248 (35.3)	
2014-2018	110 (10.0)	779 (9.1)	1513 (8.3)	1123 (10.3)	409 (11.6)	
IMD group (NZDep13 scale), n (%)						
Least deprivation: IMD-1 (1 or 2)	81 (7.4)	832 (9.8)	2169 (11.9)	1509 (13.8)	504 (14.2)	<0.0001
IMD-2 (3 or 4)	78 (7.1)	923 (10.8)	2200 (12.1)	1423 (13.0)	455 (12.9)	
IMD-3 (5 or 6)	110 (10.0)	953 (11.2)	2091 (11.5)	1196 (10.9)	362 (10.2)	
IMD-4 (7 or 8)	276 (25.1)	2021 (23.7)	4387 (24.1)	2588 (23.6)	851 (24.1)	
Most deprivation: IMD-5 (9 or 10)	556 (50.4)	3808 (44.6)	7349 (40.4)	4242 (38.7)	1367 (38.6)	
Smoking status, n (%)						
Never smoking	0(0.0)	3385 (39.7)	13052 (71.7)	9074 (82.8)	3362 (95.0)	<0.0001
Ex-smoker	535 (48.6)	2906 (34.0)	2843 (15.6)	1047 (9.6)	112 (3.2)	
Current Smoker	566 (51.4)	2246 (26.3)	2301 (12.7)	837 (7.6)	65 (1.8)	
Duration of having diabetes, years	6.0 (1.9)	4.9 (1.1)	4.2 (1.6)	3.8 (1.2)	3.5 (1.8)	<0.0001
Body mass index, kg/m^2	33.7 (7.0)	33.2 (7.2)	32.5 (7.2)	32.0 (7.2)	31.0 (7.1)	<0.0001
Systolic blood pressure, mmHg	145 (15)	143 (17)	138 (18)	128 (17)	118 (11)	<0.0001
Diastolic blood pressure, mmHg	85 (11)	84 (11)	82 (11)	79 (10)	75 (9)	<0.0001
HbA1c, mmol/mol / %	86.1 (18.8)/10.0(3.8)	72.0 (20.5)/8.7(4.0)	60.5 (13.9)/7.7 (3.4)	55.3 (10.7)/7.2(3.1)	50.6 (9.5)/6.8(3.0)	<0.0001
Total cholesterol, mmol/L / mg/dl	5.5 (1.2) / 212.7 (46.4)	5.3 (1.2) / 205.0 (46.4)	5.2 (1.2) / 201.1 (46.4)	4.9 (1.2) / 189.5 (46.4)	4.5 (1.2) / 174.0 (46.4)	<0.0001
Triglyceride, mmol/L[a]	2.7 (1.9) / 239.2(168.3)	2.5 (1.7) / 221.4 (150.6)	2.3 (1.6) / 203.7(141.7)	2.1 (1.4) / 186.0 (124.0)	2.0 (1.3) / 177.2 (115.2)	<0.0001
Low-density lipoprotein cholesterol, mmol/L / mg/dl	2.7 (0.9) / 104.4 (34.8)	2.6 (0.9) / 100.5 (34.8)	2.5 (0.9) / 96.7 (34.8)	2.4 (0.9) / 92.8 (34.8)	2.1 (0.9) / 81.2 (34.8)	<0.0001
High-density lipoprotein cholesterol, mmol/L	1.1 (0.3) / 42.5(11.6)	1.2 (0.3) / 46.4 (11.6)	1.2 (0.4) / 46.4 (15.5)	1.2 (0.4) / 46.4 (15.5)	1.2 (0.3) / 46.4 (11.6)	<0.0001
estimated Glomerular filtration rate<90 ml/min/1.73 m^{2}n (%)	1101 (100)	7157 (83.8)	10551 (58.0)	3809 (34.8)	285 (8.1)	<0.0001
Antihypertensive treatment, n (%)	721 (65.5)	5709 (66.9)	11954 (65.7)	7516 (68.6)	2211 (62.5)	<0.0001
Statin treatment, n (%)	536 (48.7)	4221 (49.4)	9154 (50.3)	6638 (60.6)	2368 (66.9)	<0.0001
Antiplatelet or anticoagulant treatment, n (%)	28 (2.5)	184 (2.2)	410 (2.3)	273 (2.5)	81 (2.3)	<0.0001
Antidiabetes treatment, n (%)						
Oral antidiabetes drug and insulin	315 (28.6)	1613 (18.9)	2783 (15.3)	1596 (14.6)	506 (14.3)	<0.0001
Oral antidiabetes drug only	622 (56.5)	5134 (60.1)	10262 (56.4)	7027 (64.1)	2374 (67.1)	
Insulin only	43 (3.9)	307 (3.6)	531 (2.9)	333 (3.0)	90 (2.5)	

The distribution of continuous variables was tested using ANOVA, and the distribution of categorical variables was tested using the chi-square test

[a] Log-transformation was processed to meet the ANOVA requirement

21.1 (95% CI: 14.4–31.0) years, a difference of 8.0 (3.7–12.3) years. The HLE was slightly higher among women with four or five WTRF+, at 23.7 (18.9–29.7) years, indicating a 10.6 (7.0–14.1) year difference. Similarly, men with T2D and 0 WTRF+at enrolment aged 50 years had an HLE of 11.2 (7.6–18.6) years, while those with three WTRF+at baseline had an HLE of 20.1 (11.4–35.5) years, a difference of 9.0 (2.5–15.4) years. The HLE was higher among men with four or five WTRF+at baseline, at 23.2 (17.2–31.4) years with four or five: a 12.1 (8.1–16.1) year difference. The tests for trend were not statistically significant among women. Among men, HLE increased with increasing number of WTRF+at baseline, as did life expectancy free of CVD (Table 2).

In Additional file 1: Table S2, for women with four or five WTRF+at enrolment, there is a significant reduction in the hazard ratio for CVD, cancer, or dementia to 0.67 (0.57–0.79) over 1994–2018. Additionally, the risk of mortality decreased over this time period, with a hazard ratio of 0.43 (0.21–0.88). This pattern of decreasing risk with an increase in WTRF+at baseline was also consistent but weaker for men, affirming a strong link between WTRF+at baseline and CVD, cancer, dementia, and mortality in this cohort.

As presented in Additional file 1: Table S3, among NZE, the hazard ratios for the risk of CVD, cancer, or dementia were 0.85 (0.73–1.00) for one WTRF+at baseline, 0.74 (0.64–0.86) for two WTRF+at baseline, 0.74 (0.63–0.86) for three WTRF+at baseline, and 0.71 (0.60–0.86) for

Table 2 Healthy life expectancy (in years) at age 50 years in people with type 2 diabetes free of CVD, cancer or dementia according to number of within-target risk factors at baseline in New Zealand between 1994-2018

Women	**Number of within-target risk factors**					***P*-value for linear trend**
	Zero	**One**	**Two**	**Three**	**Four or Five**	
	n=464	**n=4,018**	**n=9,086**	**n=5,158**	**n=1,794**	
Free of CVD, cancer, or dementia						
Life expectancy	13.1 (9.1-19.0)	16.3 (9.9-26.9)	19.0 (11.9-30.4)	21.1 (14.4-31.0)	22.6 (16.8-30.1)	0.63
Difference	Reference	3.2 (-0.4-6.8)	5.9 (1.2-10.6)	8.0 (3.7-12.3)	9.5 (5.8-13.2)	
Free of CVD						
Life expectancy	15.7 (13.0-18.9)	18.7 (15.7-22.2)	20.9 (18.3-23.9)	22.5 (20.5-24.7)	23.7 (22.2-25.2)	0.37
Difference	Reference	3.0 (1.9-4.1)	5.2 (3.8-6.7)	6.9 (5.0-8.7)	8.0 (5.8-10.2)	
Free of cancer						
Life expectancy	14.2 (11.2-18.0)	17.9 (15.4-20.7)	20.6 (18.7-22.8)	22.5 (20.9-24.2)	23.7 (22.4-24.9)	0.13
Difference	Reference	3.6 (2.4-4.9)	6.4 (4.2-8.6)	8.3 (5.5-11.1)	9.5 (6.4-12.6)	
Free of dementia						
Life expectancy	13.8 (9.5-20.1)	18.9 (14.7-24.3)	22.5 (19.1-26.4)	24.4 (22.1-26.9)	25.3 (23.9-26.6)	0.41
Difference	Reference	5.1 (2.7-7.4)	8.7 (4.9-12.4)	10.6 (6.1-15.0)	11.5 (6.7-16.3)	
Men	n=637	n=4,519	n=9,110	n=5,800	n=1,983	
Free of CVD, cancer, or dementia						
Life expectancy	11.2 (6.7-18.6)	14.6 (6.9-30.8)	17.6 (8.6-36.1)	20.1 (11.4-35.5)	22.6 (15.9-33.3)	0.014
Difference	Reference	3.4 (-2.0-8.8)	6.5 (-0.8-13.8)	9.0 (2.5-15.4)	11.3 (6.2-14.7)	
Free of CVD						
Life expectancy	13.8 (11.0-17.4)	17.1 (13.6-21.6)	19.8 (16.4-23.8)	21.7 (19.1-24.7)	23.8 (21.8-26.0)	0.001
Difference	Reference	3.3 (1.9-4.7)	6.0 (4.2-7.7)	7.9 (6.1-9.7)	9.9 (9.1-11.6)	
Free of cancer						
Life expectancy	13.3 (10.3-17.1)	17.1 (14.6-20.1)	20.2 (18.1-22.4)	22.2 (20.6-24.0)	24.0 (22.6-25.5)	0.46
Difference	Reference	3.8 (2.6-5.1)	7.9 (4.6-9.1)	8.9 (6.1-11.8)	10.7 (8.1-13.0)	
Free of dementia						
Life expectancy	12.7 (8.7-18.5)	17.9 (13.5-23.7)	21.8 (18.0-26.5)	24.1 (21.3-27.2)	25.9 (23.6-27.6)	0.53
Difference	Reference	5.1 (2.8-7.5)	9.1 (5.4-12.8)	11.4 (7.2-15.5)	13.2 (8.9-16.3)	

Age, ethnicity, index of multiple deprivation, body mass index, diastolic blood pressure, duration of having diabetes, triglyceride, total cholesterol, high-density lipoprotein cholesterol, anti-diabetes treatment, statin, antihypertension treatment, and antiplatelet/anticoagulant treatment were adjusted. All estimations are derived from multistate models. The number of within-target risk factors (WTRF+) is based on baseline measurements, not at age 50

P-values were derived from trend analyses

four or five WTRF + at baseline, each relative to the 0 WTRF + category at baseline. Similarly, the risk of mortality in patients without these conditions also decreased, with a hazard ratio of 0.75 (0.60–0.95) for those with four or five WTRF + at baseline. This pattern of decreasing hazard ratios with an increasing number of WTRF + at baseline is consistent across Māori, Pacific Islands people, and Other ethnic groups, indicating a strong association between WTRF + at baseline and these health conditions.

Additional file 1: Table S4 shows that within the least deprived group (IMD-1 or 2), the hazard ratios for non-fatal conditions and mortality decreased as the number of WTRF + at baseline increased. For example, in the least deprived group, those with four or five WTRF + at baseline had a hazard ratio of 0.72 (0.50–1.02) for cancer, CVD, and dementia, indicating a trend towards reduced risk, although this result was not statistically significant ($P = 0.06$).

Additional file 1: Table S5 reveals that across NZE, Māori, Pacific Islands people, and Other ethnic groups, the HLE free of CVD, cancer, or dementia increases from zero to four or five WTRF + at baseline, indicating the positive impact of low-risk factors on extending condition-free lifespan across different ethnicities. However, trends were only statistically significant among NZE and the "Other ethnic group" category for those free of CVD and among "Other ethnic groups" for those free of all three diagnoses. Additional file 1: Table S6 reveals that HLE free of CVD, cancer, or dementia at age 50 years increased with an increasing number of WTRF + at baseline; however, the trend was only statistically significant in the IMD-2 group for all three diagnoses.

Figures 1 and 2 reveal that HLE free of cancer, cardiovascular disease, or dementia increased with the number of WTRF + at baseline, with this trend consistent across both sex and ethnic group. Figure 3 and Additional file 1: Fig. S3 show increasing difference in HLE between those with zero and those with one to four or five WTRF + at baseline, across different ages, for men and women, and across ethnicities, using those with zero WTRF + at baseline as a reference.

Additional file 1: Fig. S4 shows that the HLE free of cancer, cardiovascular disease, or dementia increased with the number of WTRF + at baseline for those aged between 51 and 85 years across IMG groups. Additional file 1: Fig. S5 shows an increasing difference in HLE between those with zero and those with one to four or five WTRF + at baseline, for those aged between 51 and 85 years across different IMG groups.

Discussion

In our study utilising the DCSS dataset linked with national databases in NZ, we found that individuals with T2D without WTRF + at baseline faced reduced HLE free from CVD, cancer, or dementia. This pattern was consistently observed across subgroups, with men, Māori, and those of lower socioeconomic status experiencing relatively shorter HLE without WTRF + at baseline. Conversely, those with more WTRF + at baseline saw significantly longer HLE free from these major conditions, uniformly across sex, ethnicity, and socioeconomic status. The research underscores the significant role of WTRF + at baseline in enhancing disease-free HLE, highlighting the importance of lifestyle and clinical interventions in T2D management.

Enhanced life expectancy is evident globally, influenced not only by healthier lifestyles, such as increased exercise and healthy nutrition, but also by pharmaceutical treatment, improved healthcare access, and quality of care. The contributions of these factors can vary significantly by country. Chudasama et al. noted significant disparities in survival post-CVD events among those with T2D in England, and particularly influenced by ethnicity [21]. Gavurova and Vagasova highlighted the effect of CVD and T2D on Slovak life expectancy, suggesting strategies for reducing mortality rates could extend LE [22]. Díaz-Venegas et al. connected T2D to early cognitive decline, emphasising the importance of T2D management on cognitive health [23]. Our study, utilising New Zealand's comprehensive primary care electronic health records and national registry databases, explored how managing blood pressure, lipids, and glycaemia impacted HLE free from these three major conditions in T2D. It highlights the role of lifestyle interventions in enhancing life quality and duration in T2D. Additionally, medication use plays a significant role in risk factor control. Medications such as antihypertensives, statins, and anti-diabetes treatments (e.g. insulin and oral agents), are essential for

(See figure on next page.)

Fig. 1 Healthy life expectancy (in years) at age 51–85 years free of cancer, CVD, or dementia among men and women with type 2 diabetes and different number of within-target risk factors at baseline. Estimations were adjusted for age, ethnicity, index of multiple deprivation, body mass index, diastolic blood pressure, duration of having diabetes, triglyceride, total cholesterol, high-density lipoprotein cholesterol, anti-diabetes treatment, statin, antihypertension treatment, and antiplatelet/anticoagulant treatment. All estimations are derived from multistate models. The number of within-target risk factors (WTRF +) at baseline is based on baseline measurements, not at age 51–85 years

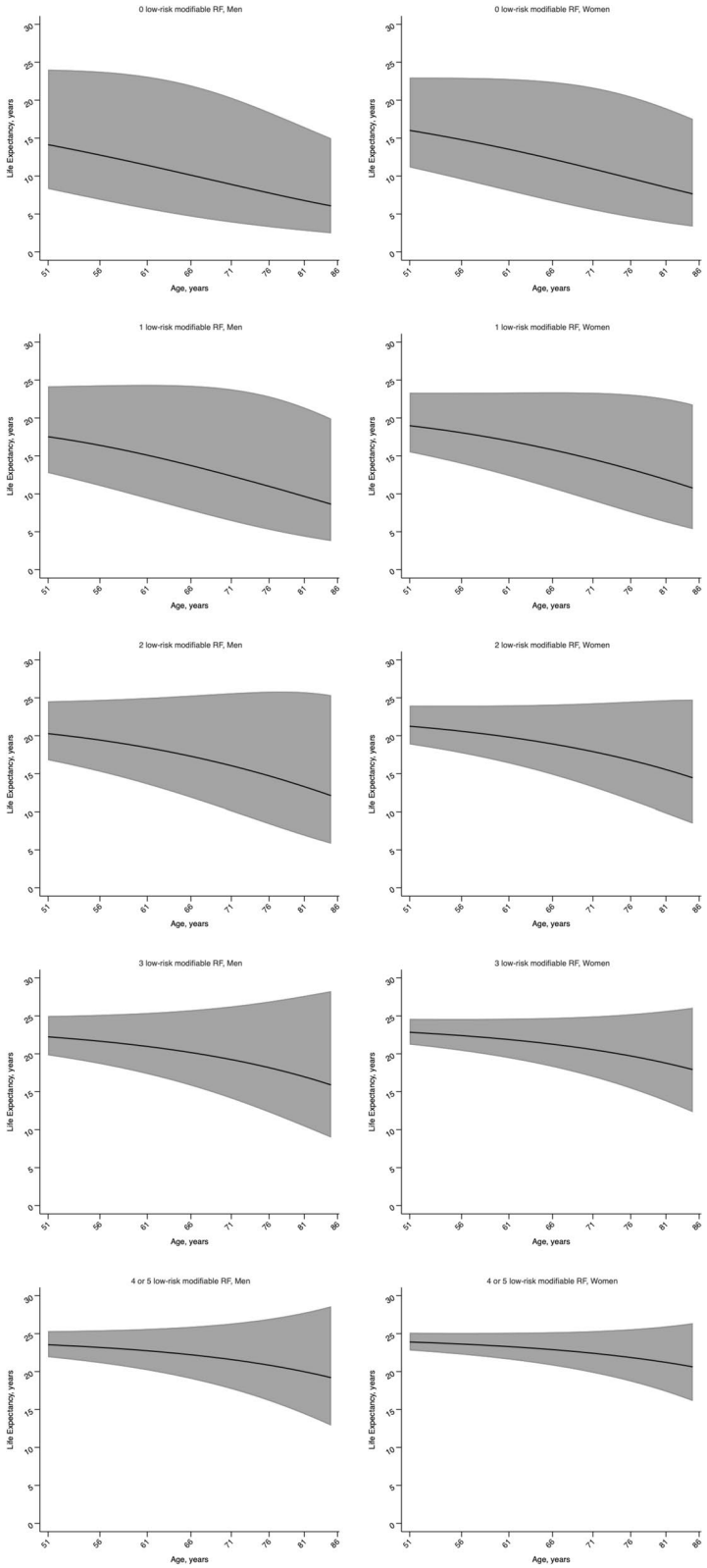

Fig. 1 (See legend on previous page.)

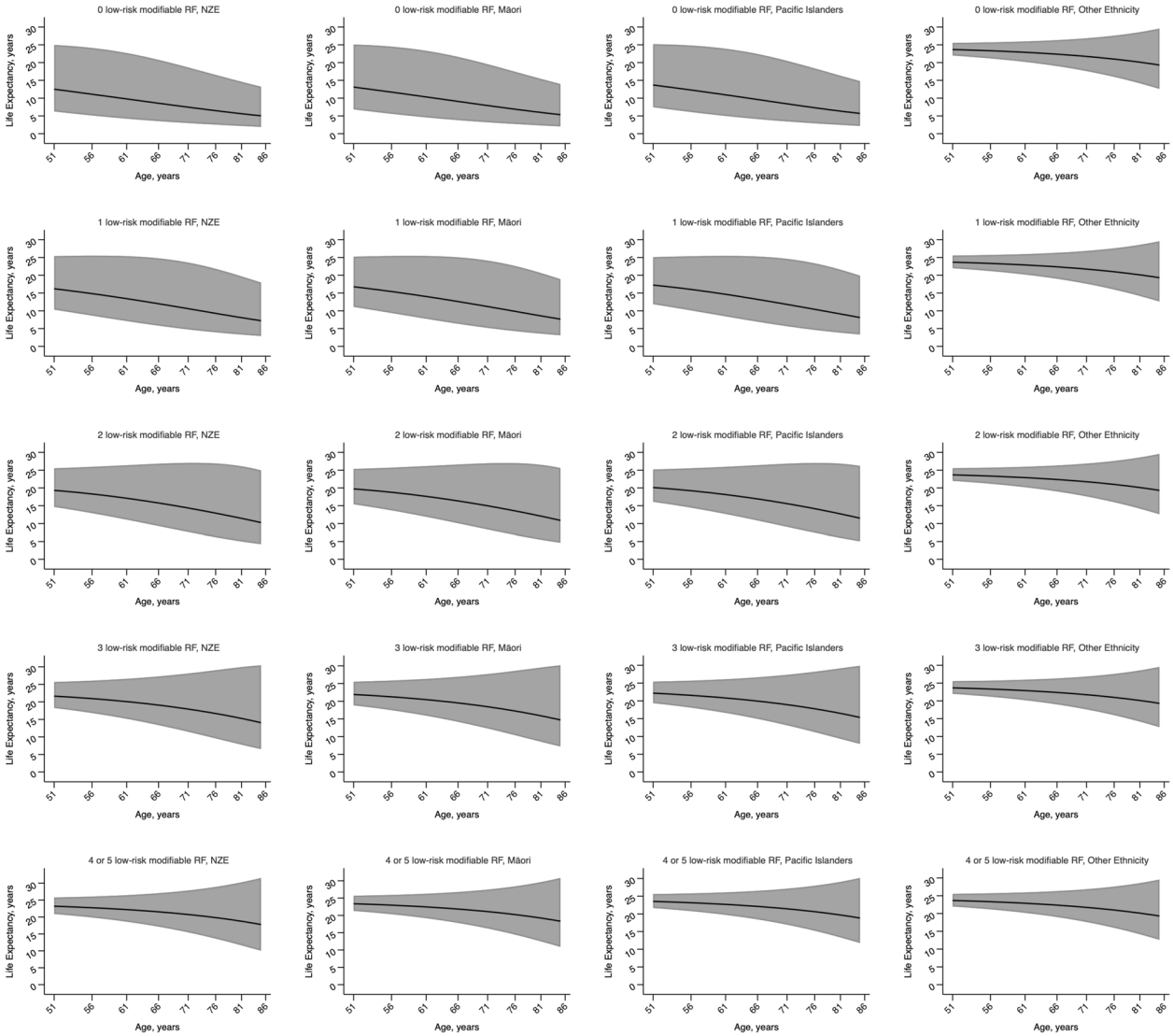

Fig. 2 Healthy life expectancy (in years) at age 51–85 free of cancer, CVD, or dementia among type 2 diabetes patients across different ethnic groups and varying numbers of within-target risk factors at baseline. Estimations were adjusted for age, sex, index of multiple deprivation, body mass index, diastolic blood pressure, duration of having diabetes, triglyceride, total cholesterol, high-density lipoprotein cholesterol, anti-diabetes treatment, statin, antihypertension treatment, and antiplatelet/anticoagulant treatment. All estimations are derived from multistate models. The number of within-target risk factors (WTRF +) at baseline is based on baseline measurements, not at age 51–85 years

managing blood pressure, cholesterol, and glycaemia, respectively. Acknowledging socioeconomic and educational barriers to a healthier lifestyle, characterised by regular exercise and healthy nutrition and medication adherence, is essential for making healthy living accessible to everyone, ensuring widespread benefits.

Based on New Zealand Ministry of Health projections for those newly diagnosed with T2D at age 53 years in 2001, the estimated overall HLE, without considering mortality or adjusting for covariates, was 22.6 years for men and 25.8 years for women [24]. Unlike prior New Zealand studies in the prevalent T2D population that did

(See figure on next page.)

Fig. 3 Healthy life expectancy differences at ages 51–85 free of cancer, CVD, or dementia in type 2 diabetes patients by sex and number of within-target risk factors at baseline (using 0 factors as reference). Estimations were adjusted for age, ethnicity, index of multiple deprivation, body mass index, diastolic blood pressure, duration of having diabetes, triglyceride, total cholesterol, high-density lipoprotein cholesterol, anti-diabetes treatment, statin, antihypertension treatment, and antiplatelet/anticoagulant treatment. All estimations are derived from multistate models. The number of within-target risk factors (WTRF +) at baseline is based on baseline measurements, not at age 51–85 years

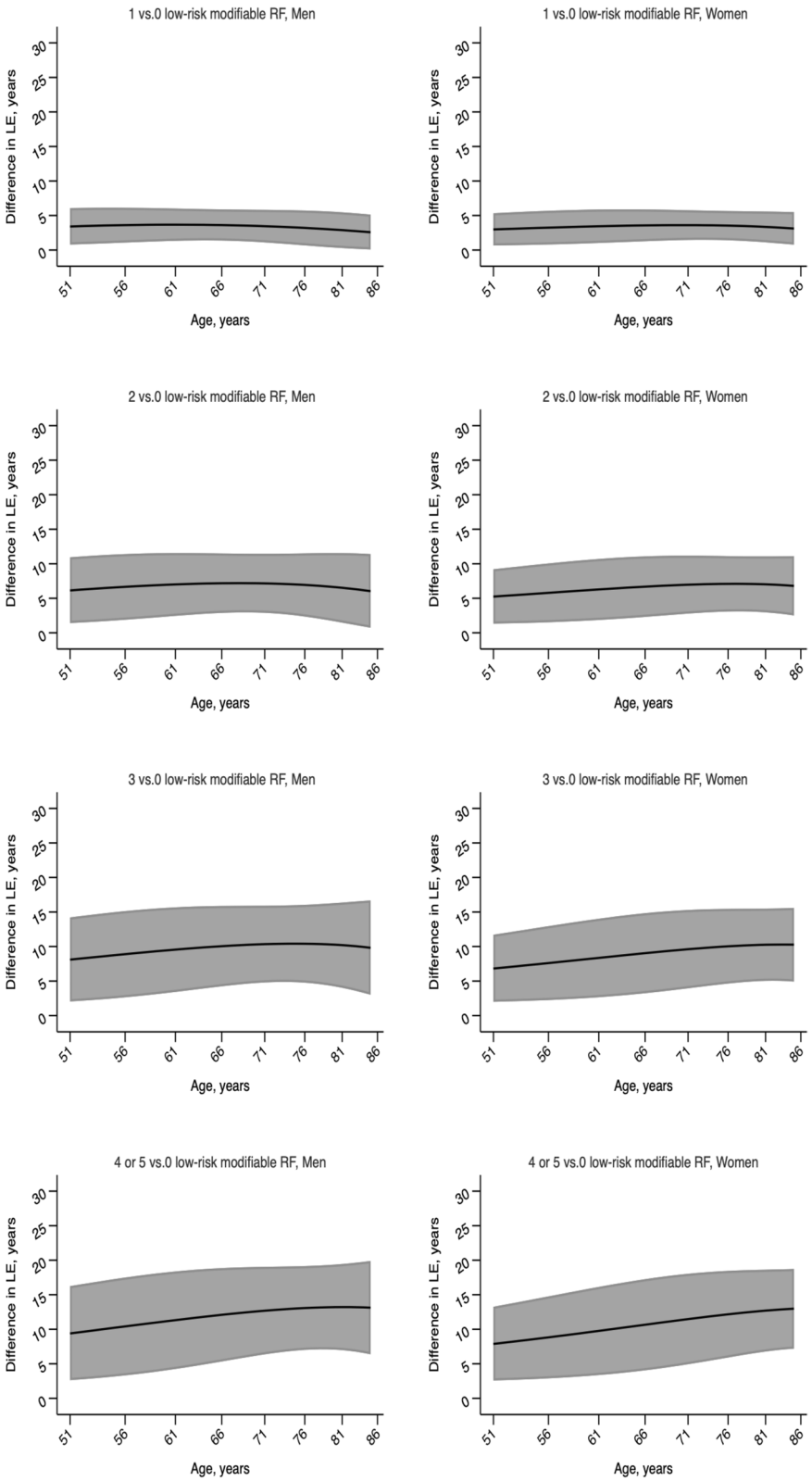

Fig. 3 (See legend on previous page.)

not estimate HLE free of these three chronic conditions for, our study uniquely assesses HLE free of CVD, cancer, or dementia, considering mortality and covariates. For individuals aged 53 years without WTRF + at baseline, HLE was projected at 14 years for men and 15 for women, which could increase to 24.3 for men and 24.5 for women with four or five WTRF + at baseline. In our study, having multiple WTRF + at baseline was associated with an increase in HLE free of these three conditions. Specifically, women aged 50 years with four or five WTRF + at baseline gained 10.6 more years of HLE compared to those without WTRF + at baseline. Men gained 12.1 more years, comparing to those without WRTF + at baseline. This was similar to another study focusing on individuals aged 51–60 years with T2D, where well-managed BMI, BP, HbA1c, and LDL resulted in a life expectancy gain of 10.9 years for women and 12.7 years for men [9]. Broader observations in the general population suggest that, typically, women benefit more from low-risk lifestyle adherence (healthy diet, smoking, physical activities, less alcohol consumption, and low BMI) than men in terms of life expectancy free of major chronic disease [25, 26]. However, the discrepancies between our findings and these broader observations could be due to several factors. First, the T2D population in our study is inherently different from the general population, which may lead to varying health outcomes. Second, the outcomes in our study focus on WTRF + at enrolment, whereas broader lifestyle adherence includes additional factors like diet, physical activity, and alcohol consumption, which were not available in our study. Third, the specific components of WTRF + at baseline may not entirely capture the comprehensive lifestyle adherence considered in previous studies. Future studies in T2D that incorporate available data on diet and physical activities are warranted to provide a more comprehensive understanding.

The relationship between WTRF + at baseline and mortality risk appears more complex compared to the relationship with nonfatal chronic conditions. This complexity can be attributed to several factors. Firstly, mortality in patients with T2D can result from a variety of causes beyond the chronic condition we specifically targeted (CVD, cancer, dementia). These other causes of death may not be as strongly influenced by the WTRF + at baseline we examined, leading to a more complex relationship. Future research could build on our findings by employing more detailed analyses to examine the effects of specific risk factor combinations on life expectancy, particularly for different subgroups within the T2D population. Secondly, patients with fewer WTRF + at baseline may have already developed complications or advanced stages of diseases that increase their overall mortality risk, which can mitigate the apparent benefits of controlling these risk factors on mortality rates. Thirdly, some risk factors may have a more pronounced effect on nonfatal conditions compared to mortality. For instance, while controlling blood pressure and cholesterol may significantly reduce the incidence of CVD, the same level of control might not translate into a proportional reduction in overall mortality due to other competing risks. Finally, the impact of WTRF + at baseline on mortality can vary significantly across different ethnic groups and socioeconomic status. These variations can influence the overall patterns observed in the data. The statistically significant differences observed for variables (shown in Table 1) highlight their potential influence on outcomes related to life expectancy and healthy life expectancy. Adjusting for these factors in our multistate model was essential to accurately isolate the effects of within-target risk factors on healthy life expectancy, ensuring that the observed associations are not confounded by other demographic or health-related factors.

The disparities in HLE at age 50 years between Māori, with the shortest HLE at 10.5 years with no baseline WTRF +, and the NZE population, with a HLE of 19.8 years under the same conditions, underscore significant ethnic health disparities in New Zealand. However, having multiple WTRF + at baseline appears to level these differences across ethnic groups, suggesting that lifestyle intervention and optimal clinical management could be a powerful tool for reducing health disparities [27]. This highlights the importance of tailored public health strategies and high-quality clinical care that not only promote healthier lifestyles and timely and effective treatment but also address the specific needs, challenges, and barriers experienced by diverse ethnic populations to improve their HLE and quality of life [27]. Additionally, the significant reduction in the risk of dementia with an increased number of WTRF + at baseline observed in women but not in men could be attributed to several factors, including biological differences, lifestyle factors, and potential disparities in healthcare utilisation, differential competing mortality, and management between genders. Hormonal differences, such as the protective effects of oestrogen in women, could also play a role in these observed differences [28]. Further research is needed to fully understand the underlying mechanisms and to determine if these findings can be generalised across different populations.

The socioeconomic disparities highlighted by the HLE free of CVD, cancer, or dementia differences at age 50 years between the most deprived and the least deprived socioeconomic groups in New Zealand underscore the profound impact of socioeconomic status on health outcomes. The most deprived group had an HLE of 7 years compared to 11.7 years in the least deprived,

highlighting the profound role of socioeconomic factors. However, the convergence of HLE across socioeconomic groups with multiple WTRF + at baseline underscores the potential of lifestyle intervention and optimal clinical management in mitigating these disparities. Population health strategies such as improved primary care and targeted public health initiatives may help to enhance HLE and quality of life across different socioeconomic groups [29, 30].

Our research stands out as the most comprehensive study involving a diverse group of people with T2D in New Zealand, highlighting significant health outcomes over two and a half decades. The linear trend analysis for HLE with increasing WTRF + at baseline generally supported a linear relationship, suggesting that managing more WTRF + at baseline consistently contributes to increased HLE. However, the non-significant linear relationship observed in several subgroups, such as men, most IMD categories, and the NZE population, indicates that the marginal benefits of additional WTRF + at baseline may vary. This finding underscores the need for targeted interventions that consider the unique responses of different demographic groups to risk factor management. This study uniquely incorporates every patient from participating general practices and employs extensive, nationally representative data sources for tracking all new health outcomes, including specific causes of death and hospital admissions. While primary ICD codes in detailed registration databases ensure high accuracy in clinical documentation and disease categorisation [10], there are limitations such as potential underreporting or delayed diagnosis, particularly for early-stage dementia. Furthermore, the accuracy and timeliness of dementia diagnosis may have changed over time due to advancements in diagnostic criteria, increased awareness, and improved recording practices. These changes could affect the reliability and completeness of dementia data in different periods, potentially influencing our findings. Future research should consider using population survey cohorts to address these limitations and provide a more comprehensive understanding of dementia prevalence. Moreover, the analysis of the specific impact of individual risk factors or their combinations on health outcomes is complex and requires separate detailed analysis. This will be an important focus for future research to understand the relative significance of different risk factors, such as the immediate impact of BP control versus long-term glycaemic management on CVD outcomes.

However, the study faced challenges with the varied ethnic makeup of New Zealand's population, potentially affecting the classification accuracy of self-reported data and the representativeness of the sample. Despite this, the DCSS database with the linkage to national databases captured a considerable portion of the Pacific, Māori, and possibly lower socioeconomic European populations, offering insights into diabetes-related complications [13]. A notable observation was the higher rate of ESRD hospital admissions among younger NZE patients, indicating a possible inclusion of individuals with type 1 diabetes [10]. This misclassification issue, common in primary care settings, may have moderated the differences observed across ethnic groups [31]. While adjustment with covariates was processed, there might still be some misclassification in the covariates, suggesting the need for future studies to adjust for this risk of misclassification. Previous studies have examined the impact of self-reported lifestyle factors, such as smoking, diet, and physical activity, on overall life expectancy using survey data in the general population [25, 26]. These lifestyle risk factors, however, are not routinely recorded in electronic health records, limiting dynamic surveillance. Our study investigates the effect of routinely recorded WTRF + at baseline on HLE free of CVD, cancer, and dementia among people with T2D. This approach enables ongoing surveillance of changes in HLE free of major chronic conditions, identifying high-risk groups for targeted integrated care strategies to improve their prognosis. While our primary focus is on estimating years lived free of chronic conditions, the multistate life table approach also incorporates mortality risk as a competing event, ensuring that our life expectancy estimates reflect both quality of life (QOL) and the likelihood of mortality. This dual consideration provides valuable insights into both QOL and mortality risk. It is important to clarify that our study primarily aims to facilitate improvements in QOL by understanding how within-target risk factors influence healthy life expectancy (HLE). Although direct assessments of mortality could be performed using other methodologies, our multistate model allows for the simultaneous consideration of nonfatal outcomes, thereby offering a comprehensive picture of health dynamics in T2D management. Although the period effect, equivalent to temporal or seasonal effect, has been adjusted for in the modelling process, there might be potential information bias due to the longitudinal nature of the data (like coding and population composition change over time). This suggests that further external validation studies are warranted to confirm our findings. In this study, we focused on CVD, dementia, and cancer due to their high prevalence, significant impact on health-related QoL and life expectancy in individuals with T2D, and because these conditions are well recorded in the routine data. Although conditions such as advanced kidney disease (CKD/ESRD) also present substantial health and economic burdens, they often contribute to mortality through CVD pathways. Future research should include

CKD/ESRD to provide a more comprehensive understanding of the health burden in this population.

Obesity or overweight is a significant risk factor of overall life expectancy in the general population. In our study, the prevalence of obesity in individuals with T2D exceeded 60%, while prior research indicates that the prevalence of obesity is stable within this demographic [10, 19]. Effective clinical interventions for obesity, such as GLP-1 receptor agonists, were not available to our study cohort by the end of follow-up. However, regulatory and administrative changes in New Zealand have now improved access to GLP-1 receptor agonists and SGLT-2 inhibitors, especially for high-risk ethnic groups. Setting non-obesity or non-overweight as a within-target risk factor would have significantly reduced the size of the subgroup with multiple WTRF + at baseline, hindering further modelling efforts and producing uncertain estimates that offer limited guidance for policymakers. Future research could focus on populations with a lower prevalence of obesity to explore these associations further, although most people with T2D are either obese or overweight at the time of diagnosis and continue to be so thereafter [18]. Neighbourhood deprivation measurement using NZDep2013 might not be prospective for some participants, leading to potential misclassification bias. However, this measurement has been proven consistently stable over time [10]. While analysing the exact combinations of WTRF + at baseline could provide detailed insights, this approach would result in numerous combinations with small subgroups, limiting the statistical power. Therefore, we focused on the number of WTRF + at baseline rather than specific combinations. Our findings also indicate consistent positive effects across different strata, rather than statistical interactions between the number of WTRF + at baseline and these factors. Future studies with larger sample sizes are warranted to estimate HLE based on specific combinations of WTRF + at baseline and the interaction effects on the estimations. In our study, the number of WTRF + at baseline was assessed at baseline. It is important to note that while the relationships between the covariates and the number of WTRF + at baseline are cross-sectional, these factors are likely to interact and evolve over time. Future studies could benefit from a longitudinal approach to explore how changes in WTRF + at baseline correlate with variations in other demographic and clinical characteristics. This could provide deeper insights into the progression and management of T2D and its complications.

Previous research has explored various management goals, including stricter targets for glycaemic control, lipids, and blood pressure, in relation to overall life expectancy [9]. Unlike these studies, our research focussed on HLE free from CVD, cancer, or dementia. This narrower focus, combined with our inclusion of prevalent cases with generally stable and higher clinical measurements, means that setting overly strict goals would have led to small sample sizes and uncertain estimates due to fewer outcomes. Therefore, future research should consider populations with stricter clinical measurements to further investigate these associations, while also evaluating the potential risks and benefits, as stricter goals/cutoffs can be harmful in certain settings.

Conclusions

Based on the comprehensive insights gathered from the DCSS dataset linked with national databases in NZ, our study highlights the pivotal role of managing WTRF + at baseline in significantly extending HLE free from major conditions such as CVD, cancer, and dementia among individuals with T2D. This positive impact is consistent across various demographics, including sex, ethnicity, and socioeconomic status, illustrating the universal benefit of implementing clinical targets. Our findings underscore the necessity of incorporating lifestyle and clinical interventions into the management of T2D to prevent associated chronic conditions, emphasising a multidisciplinary approach to healthcare that can adapt to the needs of diverse populations. This research contributes valuable knowledge to the field, advocating for targeted public health strategies and personalised patient care to improve quality of life and HLE for those living with T2D, by demonstrating the potential impact of maintaining WTRF + at baseline.

Abbreviations

BMI	Body mass index
CKD	Chronic kidney disease
CVD	Cardiovascular diseases
DCSS	Diabetes Care Support Service
eGFR	Estimated glomerular filtration rate
ESRD	End-stage renal disease
HLE	Healthy life expectancy
HR	Hazard ratio
IMD	Index of multiple deprivation
NZE	New Zealand European
LDL	Low-density lipoprotein
NZ	New Zealand
SBP	Systolic blood pressure
TC	Total cholesterol
T2D	Type 2 diabetes

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12916-024-03743-y

Supplementary Material 1.

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Authors' contributions
D.Y., C.W., Z.Z, and D.S. designed the research. D.Y., H.F., Y.C., conducted statistical analysis. D.Y., Z.W., C.W., and D.S. led the writing of the manuscript. D.Y., Z.Z., K.P., J.B., R.C., B.J.OW., G.S., H.F., C.W., and D.S. contributed to the interpretation of the results and critical revision of the manuscript for important intellectual content and approved the final version of the manuscript. D.Y. and D.S. are the guarantors of this work and, as such, had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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Data availability
The datasets analysed in the current study are not publicly available because of agreements with the primary care organisations and Ministry of Health who provided the data but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The North Health Ethics Committee approved the DCSS for research purposes in 1992, and then as an ongoing audit in 1996 (92/006). Approval for waiver of individual informed consent and approval for study was provided by the New Zealand Health Disability Ethics Committee on March 25, 2019. Anonymised data were used for this analysis and all methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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