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A feasibility study investigating the risk of prediabetes among children in New Zealand

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Prediabetes is a non-communicable disease (NCD) that is common in New Zealand (NZ), and it can lead to poor health. The aim of this study was to identify whether there is an increased risk of developing prediabetes among 11–13-year-olds, outside an organised screening programme. Consenting school aged children and their parents completed a series of screening questionnaires including dietary patterns, anthropometrics and socio-economic characteristics. Adapted Australasian Paediatric Endocrinology Guidelines (APEG) criterion was used to identify children at risk of developing prediabetes or have new onset prediabetes. Of the 276 participants, significant differences between Pacific, Māori and non- Māori non-Pacific children were evident among those who: were obese (BMI > 95th percentile); lived in overcrowded homes and in deprived areas. In our study, a large proportion of children (35%) were at risk of developing prediabetes. From our dietary analyses, we identified two distinct dietary patterns from among the children: (1) a diverse diet that included a wide range of foods, but was particularly high in sweet and savoury snacks, takeaway foods, and sugary drinks; and (2) a predominantly vegetarian diet rich in legumes. The study prevalence of prediabetes risk is *indicative* of childhood lifestyles, and we recommend early screening and better resourcing for promotion of healthy nutrition as preventative measures.

Background

Non-communicable diseases (NCDs) that are predominantly diet-related, such as obesity, prediabetes, and Type 2 diabetes (T2DM) have an extensive impact on the New Zealand (NZ) health system. These NCDs collectively cost the health system over $3 billion per year[1–6]. Globally, NCDs are the leading cause of mortality and account for approximately 90% of deaths[7]. Of these NCDs, prediabetes and the risk factors of developing this condition are growing at an alarming rate, and urgent actions are needed to change the epidemic trajectory[8]. Furthermore, all of these conditions are the leading drivers for health inequalities, particularly among Pacific people living in NZ, as they experience the highest inequities with these NCDs[9]. A major independent risk factor for prediabetes and T2DM is obesity, which in NZ, 1 in 10 children (2–14 years) are considered obese (9.4%), based on children's height and weight at, or above, the 95th percentile for age and sex adjusted body mass index (BMI). The prevalence of obesity among Pacific (29%) and Māori (13%) children is disproportionately higher than for European/Other (7%) and Asian (3%) children[10]. It has been well established that childhood obesity is a life-course predictor of being overweight in young adulthood[11], and it is associated with obesity related co-morbidities (e.g., T2DM and cardiovascular disease) in adulthood[12]. Further, it has been reported that persistent obesity is established in early childhood, before 11-years-old[13]. Wardle et al. (2006) analysed data from over

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5,800 English school children aged 11–12 years and found that obesity prevalence at the time of the study was approximately 25%. In their five-year follow-up, the obesity prevalence rose by approximately 7%, and for the most part this was because children who were overweight became obese. More recently, childhood obesity rates for children younger than 5 years of age has significantly reduced, but the increased rates for children older than this age group continue to remain high[14]. Regardless, it had been suggested that 'obesity' and 'overweight' are associated with a chronic low-grade inflammatory state[15], which may increase the risk of cardiovascular events, later in life. Therefore, a focus on early prevention in childhood will be critical for the prevention of poor health outcomes and other related risk factors in adulthood.

Prediabetes defined as having a blood glucose level that is above normal, but below the threshold diagnostic criteria for T2DM. In NZ, the diagnostic criteria for prediabetes is based on having a haemoglobin A1C (HbA1c) between 41-49mmol/mol (5.8–6.7%), and no formal diagnosis of T2DM[16]. The prevalence of prediabetes is high among NZ adults (25%)[16], and it is elevated in Pacific adolescents and young adults. Among youth aged 15–24 years, 13% of Pacific youth have prediabetes (vs. 7% in NZ Europeans), and the prevalence increases with each life-course age group, with older adults (65–74 years) having the highest prevalence 56% (vs. 44% in NZ Europeans)[16]. A recent study of 451 Auckland school aged children, reported an overall prediabetes prevalence of 16% among their sample of 8–11-year-olds. Children of South-East Asian ethnicity had the highest prevalence (30%) and among Pacific Island children the prevalence was 27%[17]. With such a high study prevalence rate, the authors recommended the need for early identification of prediabetes and timely intervention at childhood, rather than adulthood.

Methods

This paper provides recent findings from a feasible cross-sectional study undertaken in the wider Wellington region, NZ (2021–2022) to establish whether the risk of developing prediabetes was prevalent, when screening for prediabetes amongst young adolescents (aged 11–13-year-olds), using a two-phased process. This study involved recruiting 11–13-year-olds from primary schools in the Wellington, NZ, who fit the criteria (see below) for targeted screening for prediabetes risk. The targeted age group was selected because these children were due for their routine dental health check-ups which had been conducted yearly since the age of 2 years old until the end of their middle-school (year 8). This dental examination timeframe offered an ideal window-frame to undertake opportunistic screening for children at risk of developing prediabetes. Full ethical approval was obtained from the NZ national health ethics committee and all methods and processes were performed in accordance with the relevant guidelines and regulations of the Health and Disability Ethical Guidelines (HDEC: 2022-FULL-12212).

Phase 1: participant recruitment and enrolment

Thirty-three out of the 36 schools that were approached and invited to collaborate, had agreed to participate in the study. The three schools that had declined to participate in our study were already heavily involved in other research, and the school Principals did not want to overcommit their families and students to new projects. Prior to each child's dental exam at each school, all parents were sent a study pack, inviting them and their child to participate by completing a consent form and screening questionnaire (phase 1). The study packs were distributed by the school office and the consent form sought parental/guardian permission to access their child's health dental data, obtain an HbA1c blood test (if the child qualified for phase 2), and to access their family general practitioner (GP) if the research programme identified a new-onset of prediabetes or T2DM (i.e., provide a letter of referral outlining the test results). Of note, a child assent form was also provided to each child (written in age-appropriate level) to provide an informed consent in parallel with their parents/guardians. To complete participation in phase 1, all completed (and non-completed) packs were returned to the school after a 7 day period (collected by our team). Within the study pack, parents and their child were to complete the ***Screening questionnaire.*** The questionnaire was composed of validated questions taken from the NZ national health survey[18] and other questions previously used in our research projects[19,20], such as: full name and address; ethnicity (self-identified); age of the child and family members living under the same household; parents' perception of whether their child is overweight or obese; measured current weight (kilograms, kg) and height (centimeters, cm); maternal history of T2DM and or gestational diabetes of the mother whilst pregnant with the child; first degree relatives with T2DM and other symptoms of T2DM (e.g. dark patches in body folds/creases); medications for mental health conditions (depression and anxiety); ever treated for other conditions (asthma, high blood pressure, heart trouble, diabetes, stroke, thyroid and sleep problems, and others); sleep health (snoring and apnoeas whilst sleeping); parents knowledge of the child's dental status and history of treatment; how active the child is each week; and a 7-day record of food groups consumed (yes/no).

We used a previously adapted ***dietary diversity*** questionnaire[20] to explore the individual consumption of foods and food groups in childrens' home environment. The individual food groups included both nutritious and descretionary food items. . The data consisted of 26 food groups groups (15 nutritious and 11 discretionary). However, we had combined or omitted food groups that were not consumed to 12 groups. This helped to determine the total percentage of items consumed within a food grouping, per person. The groupings were: **Group 1**: meats, poultry, fish diversity; **Group 2**: dairy products diversity; **Group 3**: bread, cereals and starchy vegetable diversity; **Group 4**: legumes and nut diversity; **Group 5**: fruit diversity; **Group 6**: vegetable diversity; **Group 7**: oil and fat diversity; **Group 8**: drinks diversity; **Group 9**: alcohol diversity (omitted); **Group 10**: sauces, spreads and flavouring diversity; **Group 11**: sweets and sweet snacks diversity; **Group 12**: savoury snacks diversity; and **Group 13**: take way food diversity.

For research purposes, in phase 1 of the initial screening, we used the the Australasian Pediatric Endocrinology Guidelines (APEG)[21] criterion, which endorsed screening among younger children aged 10 years and older, and among Indigenous peoples including Pacific peoples and Māori. Children who fit the APEG criterion (i.e.,

identified as being at risk of developing prediabetes), and returned a signed parental consent and child assent forms with the screening questionnaire were eligible and therefore invited directly by the research team to participate in Phase 2.

Phase 2: targeted screening for prediabetes

Following their consent and eligibility, the child and their parents received a confirmed appointment time and location for testing (usually at the child's school) by the research team. Clinical data were collected by research assistants and nurses included: *anthropometric measurements*: body weight (kg) and height (cm) were measured from which BMI z-scores and percentiles were calculated using the NZ Ministry of Health's BMI calculator for New Zealanders; waist and hip circumferences were measured to obtain the waist-to-hip ratio (WtHR) as an indicator of abdominal fatness; blood pressure; and a non-fasting blood glucose test (HbA1c) was taken specifically by a trained nurse. The HbA1c tests were delivered to a large pathology contracted provider in the region on the same day of the testing to analyse the samples. The results were emailed to the research lead and summary results were forwarded to parents within 24 h of test interpretation. Initially, we used HbA1c cut-off points of 37-39mmol/mol plus established risk factors, as optimal cut-off points for screening undiagnosed prediabetes[21,22].

Results

In phase 1 of the study, a total number of 654 study packs were delivered across 36 schools. Of that, 322 children (49%) returned their study packs in response to the study invitation. We retracted the following data from 46 participants, defined here as, non-respondents who provided questionnaires that were returned and designated as *'declined*, *not eligible*, *or refused'*. Also, six respondents had provided consent, but their screening questionnaires were not received, and they were uncontactable. Therefore, the remaining 276 (45%) participants of the eligible 608 participants (after removing those who declined/uncontactable) were retained as 'participants' in phase 1.

Table 1 highlights the characteristics of phase 1 participants, which includes the main risk factors for prediabetes, by ethnicity. Gender distribution was significantly different ($p = 0.034$) between ethnic groups with 56% males among Māori and nMnP children and 38% males among Pacific children. A higher proportion of Pacific (38%) and Māori (26%) participants reported a history of first-degree relatives with T2DM, compared to19% of nMnP participants. Of the 216 parents that self-reported observations of their child as being obese, a high proportion 71.8% (155/216) of parents 'did not' perceive their child as being obese. This proportion was much higher among nMnP participants (90%) compared with Māori participants (49%) and Pacific participants (46%). For the BMI categories, using the childhood percentile rankings for young New Zealanders, 53.6% (30/56) of Pacific, followed by 51.4% (18/35) of Māori children were classified in the obese (BMI ≥ 95th percentile) range. This was more than twice the proportion of 10.4% (13/125) nMnP children. Further, 35.9% (97/270) of children reported 'ever' receiving treatment for co-existing conditions (e.g., asthma (23.3%), Attention Deficit Disorder and Autism (11.9%), sleep problems like snoring (5.2%), and psychological conditions (3.7%)) and this proportion was particularly high among Māori children (55%). Regarding living conditions at home, 11.2% (30/267) of children lived in crowded houses, whereby the number of people exceeds the number of bedrooms with higher proportions among Māori and Pacific children (10% and 32%, respectively) compared with nMnP children (4%). A quarter of the children were in the most deprived quintile, as measured by the NZ Index of Multiple Deprivation (2018) scale[23], which measures the level of deprivation for people in a small area based on nine Census variables (measured in quintiles) whereby 1 is the least deprived and 5 is the most deprived areas. There was a significant difference ($p < 0.0001$) between ethnic groups with 49% of Māori children and 45% of Pacific children who live in the most deprived quintile, compared with 11% among nMnP children. Finally, based on the phase 1 screening questionnaire, the team identified 97/179 or 35% of children as being at moderate to high risk of developing prediabetes. The majority of these children were Pacific (48/97 or 49.4%), followed by Māori (30/97 or 30.9%), and non-Māori-non-Pacific (19/97).These children were invited to the second phase of the study (see Table 3).

The study also examined the dietary patterns for children in this study in Table 2. Principal Axis Factor (PAF) method and Oblimin rotation was used for analysis of the 12 food data groups (described above). We excluded data that were identified as being major outliers ($n = 5$ participants), and thus the analyses were based on 264 observations. Each dietary pattern was allocated weights for each food group, which were used to calculate a standardised mean score for each dietary pattern. Each factor was rotated and compared to observe the cross-loading to identify and improve interpretability of each dietary factor. Parallel analyses and scree plots were also used to check for data interpretability. Each of the dietary pattern scores was standardised to have a mean of zero and a variance of one. Each participant was assigned a score for each dietary pattern, since a typical person's diet may include characteristics of more than one pattern. Thus, the dietary pattern scores are a constant measure of how closely the participant's diet matches each type of diet. Based on the PAF approach, of all the participants' dietary intake, standardised scores above 0.40 (i.e., threshold) for any given food grouping indicated a strong propensity matched to a particular dietary pattern. Thus, we have identified two distinctive dietary pattern groups (from 65 potential dietary groups). Our selection of the two-factor loadings was confirmed by parallel analyses. **Dietary factor** 1 consisted of a very diverse range of food groups, but particularly high in: takeaways, sweets and savoury snack foods, drinks, meats and poultry and breads and cereals. **Dietary factor 2** composed primarily of very high diversity of vegetarian and legume-based foods, and to a more limited extent sauces, spreads and flavourings. Additionally, the food groups 11–13 had negative standardised scores indicating that the children who consumed more food items under Dietary factor 2 ate a healthier range of food items within the food groups.

Out of the 97 children selected for phase 2, more than half were females ($n = 54$) and 43 were males. In Table 3 (below), based on the median scores (Wilcoxon two-sample p-value test due to non-parametric data), across the

	Māori	Pacific	nMnP	Total
	n = 41 (14.9%)	n = 66 (23.9%)	n = 169 (61.2%)	n = 276 (100%)
Age-groups				
10 years old	5 (12.2)	8 (12.1)	11 (6.5)	24 (8.7)
11 years old	23 (56.1)	32 (48.5)	79 (46.8)	137 (48.6)
12 years old	13 (31.7)	22 (33.3)	66 (39.1)	101 (36.6)
13 years old	0 (0)	4 (6.1)	13 (7.7)	17 (6.2)
p-value				*0.330*
Gender				
Male	23 (56.1)	25 (37.9)	95 (56.2)	143 (51.8)
Female	18 (43.9)	41 (62.1)	74 (43.8)	133 (48.2)
p-value				***0.034***
Maternal history of T2DM, gestational diabetes				
Yes	5 (13.2)	4 (6.3)	14 (8.4)	23 (8.6)
No	33 (86.8)	60 (93.3)	153 (91.6)	246 (91.5)
Missing				7
p-value				*0.479*
History of first-degree relatives with T2DM				
Yes	10 (26.3)	25 (38.5)	32 (19.2)	67 (24.8)
No	28 (74.8)	40 (61.5)	135 (80.8)	203 (75.2)
Missing				6
p-value				***0.009***
Parent perception of child overweight/obese				
Overweight				
No	29 (82.9)	45 (80.4)	111 (88.8)	155 (71.8)
Yes	6 (17.1)	11 (19.6)	14 (11.2)	61 (14.4)
Missing				60
p-value				0.285
Obese				
No	17 (48.6)	26 (46.4)	112 (89.6)	155 (71.8)
Yes	18 (51.4)	30 (53.6)	13 (10.4	61 (28.2)
Missing				60
p-value				***< 0.0001***
BMI categories				
Normal (BMI < 85th% ile)	11 (31.4)	15 (26.8)	98 (78.4)	124 (57.4)
Overweight (BMI ≥ 85th %ile and BMI ≤ 95th %ile)	6 (17.2)	11 (19.6)	14 (11.2)	31 (14.4)
Obese (BMI ≥ 95th %ile)	18 (51.4)	30 (53.6)	13 (10.4)	61 (28.2)
Missing				60
p-value				***< 0.0001***
Treatment for co-existing conditions (ever)				
Yes	22 (55.0)	18 (28.1)	57 (34.3)	97 (35.9)
No	18 (45.0)	46 (71.9)	109 (65.7)	173 (64.1)
Missing				6
p-value				***0.017***
Live in crowded housing				
No	36 (90.0)	43 (68.2)	158 (96.3)	237 (88.8)
Yes	4 (10.0)	20 (31.8)	6 (3.7)	30 (11.2)
Missing				9
p-value				***< 0.0001***
Deprivation (quintiles)				
1 = Least deprived	6 (14.6)	7 (10.9)	58 (34.7)	71 (26.1)
2	4 (9.8)	6 (9.4)	37 (22.2)	47 (17.3)
3	3 (7.3)	11 (17.2)	33 (19.8)	47 (17.3)
4	8 (19.5)	11 (17.2)	20 (12.0)	39 (14.3)
5 = Most deprived	20 (48.8)	29 (45.3)	19 (11.4)	68 (25.0)
Missing				4
p-value				***< 0.0001***
Continued				

	Māori	Pacific	nMnP	Total
	n = 41 (14.9%)	n = 66 (23.9%)	n = 169 (61.2%)	n = 276 (100%)
Identified as being at moderate-high risk of prediabetes				
No	11 (26.8)	18 (27.3)	150 (88.8)	179 (64.9)
Yes	30 (73.2)	48 (72.7)	19 (11.2)	97 (35.1)
p-value				***< 0.0001***

Table 1. Distribution frequency of characteristics of children with undiagnosed prediabetes and risk factors. Key: nMnP = non-Māori-non-Pacific; %ile = percentile; deprivation = NZ IMD deprivation scale (2018). Note: not all numbers totalled to 276 due to missing data. Significant values are in bold and italic.

Group	Group items	Dietary Factor1	Dietary Factor2
G01	Meat, poultry, fish diversity	0.66	0.08
G02	Dairy products diversity	0.63	0.16
G03	Bread, cereals and starchy vegetable diversity	0.66	0.30
G04	Legume and nut diversity	0.00	0.66
G06	Vegetable diversity	0.03	0.78
G07	Oil and fat diversity	0.42	0.27
G08	Drinks diversity	0.77	0.02
G10	Sauces, spreads and flavouring diversity	0.53	0.42
G11	Sweets and sweet snacks diversity	0.81	-0.10
G12	Savoury snacks diversity	0.73	-0.02
G13	Take away food diversity	0.89	-0.19

Table 2. Dietary diversity patterns (7-days) for Pacific children. Groups excluded: Fruit diversity (Group 5) and alcohol (Group 9).

three ethnic groups, Pacific children's risk factor profile for prediabetes based on anthropometric measurements were significantly higher in: weight, hip circumference, BMI percentile rank, and blood glucose test results were significantly different, than for non-Māori-non-Pacific children.

Out of the 97 children selected for phase 2, more than half were females ($n = 54$) and 43 were males. In Table 3 , based on the median scores (Wilcoxon two-sample p-value test due to non-parametric data), across the three ethnic groups, Pacific children's risk factor profile for prediabetes based on anthropometric measurements were significantly higher in: weight, hip circumference, BMI percentile rank, and blood glucose test results were significantly different, than for non-Māori-non-Pacific children.

Table 4 provides multivariate regression analyses for each food group, by age, gender, ethnicity, BMI category, deprivation, and being at risk of prediabetes. Median scores and the interquartile variance were used because the data was non-parametric, and the Wilcoxon two sample test was used for examining statistical relationships between co-variates. Of note, there were clear significant differences between gender groups for all food groups but not for food groups 4, 5, 6 and 11 (legume/nuts, fruits, vegetables, sweets and sweet snacks). Children who were obese scored higher than their peers in the normal BMI percentile range across all food groups, but especially for groups 1, 8, 10,12, and 13 (meats/poultry/fish, drinks, sauces/spreads/ flavouring, savoury snacks, and take-away food). Furthermore, consumption of various food groups was significantly documented among those children living in the most 'deprived' areas for all groups, but not for food items listed under food groups: 2, 4, 5, 6, and 12 (dairy products, breads/cereals and starchy vegetables, legume/nuts, fruits, vegetables, and savour snacks). Pacific children reportedly ate greater amounts of food items in groups 3, 7, 10, 11 and 12, compared to their ethnic counter-parts. The factor scores (FS1, FS2) are the standardised mean scores for each dietary factor loading (as defined above), for that group. Positive standardised score counts indicate the number of deviations above or below the standardised mean score threshold (0.4). In our study, children who were of Pacific ethnicity, live in the most deprived area, and were in the highest BMI percentile (> 95th), all scored above the mean score threshold, indicating a greater propensity for dietary factor 1, which as previously described was especially high in food groups 1, 3, 12 and 13. Also, children who were in the 12 year old age group score positively higher than the mean score for dietary factor 2, which consisted primarily of vegetarian and legume-based food.

Discussion

There are two major findings from this cross-sectional study. First, is the social-cultural-environmental determinants of health that highlight the long-term trajectory for poor health outcomes among children and those that are most vulnerable. Second, that children in their young adolescent years (aged 11–13 years old) have the high propensity to develop risk factors for prediabetes, which supports previous international studies[11,21,22], particularly for Indigenous population groups[24–27], and our study findings demonstrate the

	Māori (n = 30)	Pacific (n = 48)	nMnP (n = 19)
Age (years)	12.0	12.0	12.1
	95% CI 11.8–12.2	95% CI 11.8–12.2	95% CI 11.8–12.4
p-value	*0.630*	*0.385*	*Ref*
Height (cm)	158	160.7	157.3
	95% CI 154.6–161.4	95% CI 157.9–163.4	95% CI 152.9–163.4
p-value	*0.766*	***0.0124***	*Ref*
Weight (kg)	60.7	70.5	55.7
	95% CI 55.0–66.3	95% CI 63.6–77.3	95% CI 48.9–62.5
p-value	*0.172*	***0.020***	*Ref*
Waist (cm)	80.6	84.4	78.3
	95% CI 75.7–85.5	95% CI 80.1–88.7	95% CI 72.7–84.0
p-value	*0.538*	*0.142*	*Ref*
Hip (cm)	94.9	101.6	91.8
	95% CI 90.5–99.3	95% CI 97.3–105.8	95% CI 86.9–96.8
p-value	*0.361*	***0.012***	*Ref*
BP mmHg (systolic)	117.1	117.7	112.3
	95% CI 112.3–121.9	95% CI 113.2–122.2	95% CI 105.6–119.0
p-value	*0.406*	*0.280*	*Ref*
BP mmHg (diastolic)	76.5	76.2	72.4
	95% CI 71.4–81.7	95% CI 72.7–79.8	95% CI 68.5–76.2
p-value	*0.291*	*0.293*	*Ref*
BMI percentile rank	88.6	91.2	78.1
	95% CI 81.6–95.5	95% CI 86.7–95.7	95% CI 66.0–90.3
p-value	*0.090*	***0.010***	*Ref*
HbA1c test results (mmol/mol)	34.3	35.2	32.9
	95% CI 32.9–35.6	95% CI 34.5–36.0	95% CI 30.6–35.3
p-value	*0.193*	***0.022***	*Ref*

Table 3. Participants who completed phase 2. nMnP = Non-Māori-non-Pacific; BMI Percentile: Normal (BMI < 85th percentile), Overweight (BMI ≥ 85th– < 95th percentile), Obese (BMI ≥ 95th percentile), Severely Obese (BMI ≥ 99th percentile); HbA1c test: blood sugar levels. Significant values are in bold and italic.

unique opportunistic window-frame for screening NZ children for prediabetes, a strategy recommended by international agencies[21,28,29].

The first major finding implicates that known risk factors for prediabetes are strongly established at an earlier age group, and that it is particularly strong for Pacific children. Social issues play an important role in partially explaining these risk factors, particularly given that a quarter of the participants reside in the *'most highly' deprived areas (and another 25% live in the least deprived (more privileged) areas)*, and added to this variable, are those children living in overcrowded households. This latter variable was most prominent for Pacific children, and research has shown clear relationships between communicable diseases[30] and living in overcrowded homes among children, and its impact on social and health wellbeing (e.g., higher rates of asthma among Pacific children due to cold and damp homes)[31,32]. However, the social issues highlights a wide gap in living conditions across this study group, and therefore it emphasises that prediabetes risk factors are not only starting at a young age, but are specially pronounced in Pacific children, with risks closely linked to issues like poverty and overcrowded housing. In reality, where and how children live are closely linked to how it affects their health, even from an early age. Moreover, as prediabetes is a diet-related condition, the dietary diversity findings from the current study yielded two interesting dietary patterns. One that was predominantly diverse but high in savoury snacks and takeaway food (dietary factor 1), and the other being almost entirely vegetarian and legume based (dietary factor 2). The children in our study, particularly those living in the most deprived areas, classified as obese (above the 95th percentile), and of Pacific ethnicity, had shown to consume foods that align closely with dietary pattern 1. This is consistent with existing evidence[20,33,34] showing that similar dietary habits and risk profiles are linked to higher rates of type 2 diabetes and prediabetes among both youth and adults. Yet, effective and large-scale prevention programmes have not enabled better health outcomes for children (and the wider population) who are characteristic of type 2 diabetes and prediabetes risk, which from previous research[35,36] cite known and established barriers (e.g., language, cultural appropriateness, financial support and transport) as inhibiting success and compliancy.

Furthermore, it is possible that food groups and habits that are being consumed today are far more different compared to dietary patterns of the last two to three generations, because globalisation and technology has advanced food availability, costs and ultimately the impact from climate change. The latter is an interesting rationale for the dietary patterns, because dietary patterns have shown to contribute to the rising burden of diet related NCDs. For example, high consumption of processed food can assist to increase environmental

	G01 m (IQR)	G02 m (IQR)	G03 m (IQR)	G04 m (IQR)	G05 m (IQR)	G06 m (IQR)	G07 m (IQR)	G08 m (IQR)	G10 m (IQR)	G11 m (IQR)	G12 m (IQR)	G13 m (IQR)	FS 1	FS2
Age-groups														
10 Years	27.8 (16.7)	27.8 (16.7)	34.4 (12.5)	11.1 (33.3)	26.7 (11.7)	32.4 (18.9)	33.3 (22.2)	26.7 (13.3)	31.8 (18.2)	37.5 (33.3)	50.0 (10.0)	21.4 (32.1)	− 0.3 (1.4)	− 0.1 (1.0)
11 Years	25.9 (11.1)	27.8 (16.7)	34.4 (12.5)	22.2 (22.2)	23.3 (16.7)	32.4 (21.6)	33.3 (22.2)	26.7 (20.0)	31.8 (18.2)	41.7 (25.0)	40.0 (30.0)	21.4 (21.4)	− 0.2 (0.9)	− 0.1 (1.3)
12 Years	25.9 (18.5)	27.8 (16.7)	34.4 (15.6)	22.2 (33.3)	23.3 (23.3)	35.1 (24.3)	33.3 (22.2)	26.7 (13.3)	31.8 (22.7)	41.7 (33.3)	40.0 (20.0)	21.4 (28.6)	− 0.3 (0.9)	− 0.1 (1.5)
13 Years	25.9 (18.5)	27.8 (11.1)	37.5 (9.4)	27.8 (33.3)	20.0 (10.0)	35.1 (13.5)	22.2 (11.1)	26.7 (13.3)	36.4 (13.6)	41.7 (25.0)	40.0 (20.0)	21.4 (21.4)	− 0.3 (0.9)	0.1 (1.1)
p-value	*0.876*	*0.607*	*0.402*	*0.244*	*0.770*	*0.513*	*0.589*	*0.900*	*0.601*	*0.542*	*0.801*	*0.662*	*0.967*	*0.554*
Gender														
Male	25.9 (14.8)	27.8 (16.7)	31.3 (12.5)	11.1 (27.8)	23.3 (20.0)	32.4 (21.6)	33.3 (22.2)	23.3 (20.0)	31.8 (18.2)	41.7 (33.3)	40.0 (20.0)	21.4 (21.4)	− 0.4 (0.9)	− 0.2 (1.3)
Female	29.6 (14.8)	33.3 (16.7)	37.5 (12.5)	22.2 (33.3)	26.7 (20.0)	32.4 (18.9)	33.3 (22.2)	26.7 (20.0)	31.8 (18.2)	50.0 (25.0)	45.0 (30.0)	21.4 (28.6)	− 0.1 (1.1)	0.0 (1.5)
p-value	***0.034***	***0.008***	***0.004***	*0.079*	*0.063*	0.119	***0.008***	***0.033***	***0.042***	***0.010***	***0.007***	*0.074*	***0.002***	***0.035***
Ethnicity (3 groups)														
Māori	33.3 (11.1)	27.8 (16.7)	31.3 (18.8)	11.1 (33.3)	23.3 (23.3)	32.4 (24.3)	33.3 (22.2)	26.7 (20.0)	31.8 (27.3)	41.7 (25.0)	40.0 (20.0)	28.6 (35.7)	− 0.1 (1.0)	− 0.3 (1.5)
Pacific	33.3 (18.5)	33.3 (16.7)	37.5 (17.2)	22.2 (33.3)	26.7 (20.0)	29.7 (27.0)	44.4 (33.3)	33.3 (20.0)	31.8 (18.2)	50.0 (33.3)	50.0 (30.0)	35.7 (35.7)	0.3 (1.8)	− 0.0 (1.2)
Non-Maori non-Pacific	25.9 (13.0)	27.8 (11.1)	34.4 (12.5)	22.2 (27.8)	23.3 (16.7)	32.4 (21.6)	33.3 (22.2)	23.3 (13.3)	31.8 (18.2)	41.7 (33.3)	40.0 (20.0)	21.4 (21.4)	− 0.4 (0.7)	− 0.1 (1.3)
p-value	***< 0.000***	***0.025***	***0.049***	***0.042***	*0.150*	*0.573*	***< 0.000***	***0.000***	*0.114*	***0.006***	***0.0009***	***< 0.000***	***< 0.000***	*0.416*
BMI category														
Normal (BMI < 85th %tile)	25.9 (14.8)	27.8 (16.7)	34.4 (12.5)	22.2 (22.2)	25.0 (16.7)	32.4 (24.3)	33.3 (22.2)	26.7 (20.0)	31.8 (18.2)	41.7 (25.0)	40.0 (20.0)	21.4 (21.4)	− 0.4 (0.8)	− 0.0 (1.3)
Overweight (BMI ≥ 85th and BMI < 95th ile%	25.9 (11.1)	30.6 (11.1)	34.4 (9.4)	11.1 (33.3)	16.7 (13.3)	28.4 (24.3)	33.3 (22.2)	20.0 (13.3)	31.8 (13.6)	41.7 (16.7)	40.0 (20.0)	21.4 (21.4)	− 0.3 (0.5)	− 0.1 (1.2)
Obese (BMI ≥ 95th percentile)	33.3 (18.5)	27.8 (16.7)	37.5 (21.9)	22.2 (44.4)	26.7 (23.3)	32.4 (23.0)	38.9 (22.2)	33.3 (20.0)	36.4 (22.7)	50.0 (37.5)	50.0 (40.0)	39.3 (32.1)	0.3 (1.6)	− 0.1 (1.7)
p-value	***< 0.0001***	*0.860*	*0.391*	*0.289*	***0.025***	*0.154*	*0.101*	***0.0005***	***0.009***	*0.069*	***0.034***	***< 0.0001***	***0.002***	*0.646*
Deprivation quintile														
1 = Least deprived	25.9 (14.8)	27.8 (16.7)	34.4 (12.5)	22.2 (22.2)	23.3 (13.3)	32.4 (18.9)	22.2 (22.2)	20.0 (13.3)	31.8 (13.6)	41.7 (25.0)	40.0 (20.0)	14.3 (14.3)	− 0.5 (0.7)	− 0.1 (1.2)
2	25.9 (14.8)	27.8 (16.7)	34.4 (12.5)	33.3 (33.3)	23.3 (20.0)	35.1 (24.3)	33.3 (22.2)	23.3 (13.3)	36.4 (18.2)	41.7 (25.0)	40.0 (20.0)	21.4 (21.4)	− 0.1 (0.6)	0.3 (1.4)
3	25.9 (14.8)	27.8 (11.1)	31.3 (15.6)	11.1 (22.2)	23.3 (20.0)	32.4 (24.3)	33.3 (22.2)	26.7 (13.3)	31.8 (18.2)	41.7 (25.0)	40.0 (20.0)	21.4 (21.4)	− 0.4 (0.9)	− 0.3 (1.0)
4	29.6 (18.5)	30.6 (16.7)	34.4 (15.6)	22.2 (33.3)	21.7 (16.7)	27.0 (16.2)	33.3 (22.2)	26.7 (20.0)	36.4 (22.7)	58.3 (33.3)	40.0 (20.0)	28.6 (21.4)	− 0.1 (1.1)	− 0.2 (1.3)
5 = Most deprived	33.3 (20.4)	33.3 (22.2)	37.5 (21.9)	22.2 (44.4)	26.7 (26.7)	32.4 (25.7)	44.4 (22.2)	33.3 (20.0)	31.8 (27.3)	50.0 (37.5)	50.0 (35.0)	35.7 (32.1)	0.1 (1.8)	0.0 (1.6)
p-value	***< 0.0001***	*0.125*	*0.059*	*0.152*	*0.586*	*0.092*	***0.041***	***0.006***	***0.029***	***0.004***	*0.083*	***< 0.0001***	***< 0.0001***	***0.020***
Identified as being at moderate-high risk of prediabetes														
No	25.9 (14.8)	27.8 (11.1)	34.4 (12.5)	22.2 (22.2)	23.3 (16.7)	32.4 (21.6)	33.3 (22.2)	20.0 (13.3)	31.8 (18.2)	41.7 (33.3)	40.0 (20.0)	21.4 (21.4)	− 0.4 (0.8)	− 0.1 (1.2)
Yes	33.3 (14.8)	27.8 (16.7)	34.4 (15.6)	22.2 (33.3)	23.3 (20.0)	32.4 (24.3)	44.4 (22.2)	26.7 (20.0)	36.4 (22.7)	50.0 (33.3)	40.0 (30.0)	28.6 (28.6)	0.1 (1.4)	− 0.1 (1.4)
p-value	***< 0.0001***	*0.090*	*0.266*	*0.427*	*0.247*	*0.638*	***< 0.0001***	***0.0005***	***0.0005***	***< 0.0001***	***0.041***	***< 0.0001***	***< 0.0001***	*0.763*

Table 4. Multivariate analyses of dietary habits and socio-demographic covariates. m = median scores, IQR = interquartile range variance, FS1 and FS2 = standardised factor scores. Significant values are in bold and italic.

sustainability by reducing food waste and extending shelf-life, but not reduce the NCD rates. In particular, new issues are emerging, where 45% of young people surveyed around the world report that climate change has negatively impacting on their daily functioning including eating, school, sleep health and relationships[37]. In the current study, we cannot show that climate change has had a direct impact on the dietary patterns illustrated, however, it is an important consideration for dietary behaviours, because as climate change advances, diet-related NCDs will also be exacerbated, and much of this will be determined by the habits and behaviours of dietary patterns in relation to the environment[38]. However, children living in high-deprivation areas in NZ, particularly Pacific children, face disproportionately high rates of obesity due in part to unhealthy food environments. These environments are characterised by the high availability and marketing of energy-dense, nutrient-poor foods, often concentrated in low-income neighbourhoods[39]. Pacific families are especially affected, with many residing in areas saturated with fast food outlets, contributing to elevated obesity risk[40]. Research has shown that individual-level interventions are insufficient in these contexts, and urgent public health policies are needed to improve food environments, including restricting unhealthy food marketing and improving access to healthy food[41,42]. Without systemic changes, existing inequities in childhood health, especially among Pacific children are likely to persist.

The second major finding from our study demonstrate the unique opportunistic window-frame for screening NZ children for prediabetes, a strategy recommended by international agencies[21,28,29].

Phase 2 of the study further highlighted the need for early screening for Indigenous children at risk of prediabetes. With more than a third (35%) of the total study population being identified as being at risk of prediabetes given the high presence of risk factors ($p < 0.0001$), particularly for obesity (≥ 99th percentile) where Pacific children had two times the proportion, compared to non-Māori-non-Pacific children ($p < 0.0001$), and the blood glucose test results had demonstrated that on average, Pacific children had borderline moderate blood sugar levels that were higher than for non-Māori-non-Pacific children ($p = 0.02$). Invariably, researchers will recognise that the blood test results in the current study were not sufficiently high enough for *defined* prediabetes, we believe, our phase 1 screening questionnaire, followed by a lower HbA1c cutoff point of 35-39mmol/mol *plus* established risk factors, could be adequate to identify a potential risk of prediabetes. Several studies have identified that progression from prediabetes to T2DM in young adolescents can occur at an accelerated rate, compared to adults[43,44], and it is also important to note, that relying on HbA1c alone and using the cutoff points suggested by the ADA and APEG are based on an adult population. There are many studies that endorse the need for more research to be undertaken for the purpose of targeting 'at-risk' population groups as a preventive approach to establishing defined prediabetes and prevent the development of T2DM[45], particularly among more ethnically diverse children[46], and this is what our study findings endorses. Due to the small sample size and design of our study, we were not able to establish the actual prevalence of prediabetes in this age group, but our work does highlight a significant gap in screening and optimising on a lower HbA1c cutoff points, that requires further exploration. However, there is a concerning number of childhood participants with obesity and other risk factors that may be indicative that more research needs to be undertaken to establish a current baseline since it is difficult to approximate the number of undiagnosed children. Current national proportions estimate approximately 13.5% of children (ages 2–14) were considered obese, indicating an increase over the last five years of 1.9%[27]. What we understand from previous research is that there can be difficulty detecting prediabetes in children where the cutoff rates for various glucose tests differ across organisations, but are also derived from studies that contained non-representative samples as described earlier[47]. This finding emphasises the need to act sooner with early interventions to mitigate the onset of prediabetes and prevent increasing rates of T2DM development in adolescents. Interventions of this calibre need to incorporate lifestyle changes as they are most effective when combined with pharmacological intervention[48]. It is also relevant to focus on other environmental and structural factors that may be barriers to access sustainable lifestyle changes[49] as highlighted from the first major finding, or an approach that includes health promotion and education programmes that raise awareness and can be embedded in the school or home environment.

Conclusions

This study highlights that key risk factors for childhood prediabetes are firmly established before children reach youthhood (i.e., aged 15–24 years old), and other factors (social-cultural-environmental) that are known to perpetuate these risk factors are not new findings when tackling diet related NCDs. However, a tailored approach to addressing the health inequalities faced by vulnerable population groups such as Pacific and Māori children are needed now more than ever. The need for early prevention methods to curb unhealthy eating habits and behaviours that contribute to the childhood obesity epidemic which increases the risk of prediabetes and T2DM is needed. More than likely, these programmes should be culturally appropriate and tailored, particularly in the current study where Pacific and Māori children were overrepresented in poor health outcome statistics. If early and holistic programmes can be employed to mitigate the risks of developing prediabetes amongst adolescents, we may see a shift in health and wellbeing amongst this age group, which could safeguard their wellbeing later in life and avoid early onset of adult NCDs. Acknowledging the risk of T2DM by focusing on prediabetes risk was also a major outcome of this study, alongside the need to explore opportunistic screening window-frames and a lower cutoff point for HbA1c that are age and culturally representative. Our study reported a high prediabetes risk proportion (35%), and we believe that this is sufficient for health researchers and policymakers to discuss the potentiality of early intervention, through screening for prediabetes risk among children from age 10 years old, with a focus on those children in high-risk population groups.

Limitations

We have discussed above about the limitations of a small sample size. However, this study was conducted post-covid-19 pandemic lockdown, which placed a strained the education system, thus impacting effective recruitment

strategies. The polarisation of the pandemic also meant distrust between families and health researchers, and the competing nature of other research projects targeting the same schools for their own research meant there was high attrition of schools, children and their families consenting to participate in our study. Moreover, the restrictions of the pandemic, funding period and the time allocated to recruit participants to carry out phases 1 and 2, and conduct data analyses, placed an enormous strain on our research team's capability to complete this work within a 12-month timeline. Thus, the final study sample and findings may not be representative of the general population of NZ children of similar age and ethnicity. The timeline and funding resources also limited our ability to collect more accurate diagnostic measures, as a single point-in-time measurement of HbA1c was conducted, with no repeat testing (preferably in 6 months).

Future implications

- Implement a scaled-up national-based cross-sectional study that involves a larger sample size that is more reflective of the children aged 10–15 years old (based on the APEG guidelines) with better resources, will improve our understanding of the magnitude of prediabetes risk among NZ children.
- Further research is needed to optimise on lower HbA1c cutoff points (plus established risk factors) that represent children aged 10–15 years old and is inclusive of cultural diversity.
- Health promotional programmes that are inclusive of the social-cultural-environmental determinants of health, and that are culturally tailored to address the prediabetes risk for children and their families, be more widely available to vulnerable groups that need it the most.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

RF wrote the paper and assisted by HD. SC conducted the main analyses aided by MC, and assisted with data interpretations. All authors read the manuscript and provided input where necessary.

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Declarations

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

This project received full ethical approval from the Southern Health and Disability Ethics Committee: HDEC 2022 FULL 12212.

Consent for publication

Written informed consent was obtained from participants and their legal guardians.

Additional information

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