

Markers of chronic disease risk in a cohort of Aboriginal children: findings from the Study of Environment on Aboriginal Resilience and Child Health (SEARCH)

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While there have been dramatic declines in cardiovascular disease (CVD) mortality in the Aboriginal and Torres Strait Islander population in recent decades,¹ chronic disease is still responsible for the substantial majority of the burden of disease.² Some of the most prevalent chronic conditions and risk factors observed among Aboriginal and Torres Strait Islander adults include overweight and obesity, renal disease, diabetes and CVD.³ Aboriginal and Torres Strait Islander people have an earlier onset of chronic disease compared to Non-Indigenous people, with the incidence of these diseases increasing over time among young people, including a rise in diabetes.⁴ For example, heart/circulatory and endocrine diseases were among the most common health conditions affecting Aboriginal and Torres Strait Islander people aged 10-24 years in 2012-13.⁵

Existing research within the Aboriginal and Torres Strait Islander and other populations indicates that a high BMI increases the risk of chronic disease. Recent evidence indicates that around a third of Aboriginal and Torres Strait Islander children aged 2-17 years of age are affected by overweight or obesity and this has increased over the past decade.^{6,7} Overweight and obesity has been found to increase rapidly particularly in younger children.⁸ A high BMI in childhood is associated with increased risks of chronic

Abstract

Objective: This study investigated chronic disease risk markers among a cohort of Aboriginal children in New South Wales.

Methods: Distributions of body mass index (BMI), blood lipids and haemoglobin A1c (HbA1c) among Aboriginal children aged 5-<19 years were investigated. Prevalence ratios (PR) were calculated for borderline/high total cholesterol, low-density lipoprotein (LDL) cholesterol and HbA1c, and low high-density lipoprotein (HDL) cholesterol, by age group, sex and BMI.

Results: Almost half (46.8%) of the cohort, had a normal BMI and 53.3% had overweight or obesity. Prevalence of chronic disease risk markers was low, with no individuals having high total cholesterol (0.0%) and few having high LDL (3.0%) or borderline/high HbA1c (2.6%); 85.5% of the cohort had normal HDL. There was no significant variation in the prevalence of chronic disease risk markers by age group or sex. The prevalence of borderline total cholesterol was 28% higher (PR 1.28, 95%CI 1.06-1.54), and the prevalence of low HDL was double (2.00, 1.19-3.35) for participants with obesity versus normal BMI.

Conclusions: Dyslipidaemia and elevated HbA1c prevalence was low in the cohort, increasing with high BMI. Overweight and obesity were common, which increase the risk of developing chronic disease later in life.

Implications for public health: Findings indicate few Aboriginal children have dyslipidaemia and hyperglycaemia, supporting screening for chronic disease risk factors from 18 years of age. Opportunities to reduce overweight and obesity among children should be considered to decrease the future risk of chronic disease.

Key words: chronic disease, Aboriginal, children, body mass index, cholesterol

disease and disease markers, including high blood pressure, diabetes and elevated cholesterol levels, as well as an increased risk of having a high adult BMI.^{4,9-11}

Recently updated guidelines suggest screening Aboriginal and Torres Strait Islander peoples for CVD risk factors from the age of

18 years and to perform absolute CVD risk assessment from the age of 30 years.¹² It is suggested that diabetes screening should also begin from the age of 18 years unless individuals are affected by overweight or obesity, as this can increase the risk of chronic disease.¹³ While it is clear that some Aboriginal and Torres Strait Islander children

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experience chronic disease, there is limited evidence quantifying the extent of chronic disease risk markers in this population.¹⁴ There is also limited evidence on how risk profiles vary by sex or by age, and are related to BMI.

As the Study of Environment on Aboriginal Resilience and Child Health (SEARCH) is a New South Wales (NSW) based project, the preferred term, Aboriginal, is respectfully used hereafter in this paper.¹⁵ This study analysed the distribution of blood lipids and haemoglobin A1c (HbA1c) in a cohort of Aboriginal children in relation to age, sex and BMI, to assist in assessing the risk of chronic disease, such as CVD and diabetes, in the population. The objective was to determine the prevalence of those at low and high risk of developing chronic disease and to contribute to improving early detection and prevention by informing screening principles in Aboriginal children. To our knowledge, this is the first Australian study to quantify the distribution of chronic disease risk markers in Aboriginal children in relation to BMI, age and sex.

Methods

Ethics and engagement

Ethics approval for the present analysis was provided by the Australian National University's Human Research Ethics Committee (2013/162), and the Aboriginal Health and Medical Research Council (892/12). Engagement and feedback was sought from the Tharawal Aboriginal Corporation, Riverina Medical and Dental Aboriginal Corporation, and Awabakal Ltd to gain input on the study design and interpretation of results.

The Study of Environment on Aboriginal Resilience and Child Health (SEARCH)

SEARCH is a longitudinal cohort study of more than 1,600 Aboriginal children and 642 caregivers from urban and regional areas of NSW.¹⁶ Baseline data (Phase 1) were collected between 2008–2012 through four Aboriginal Community Controlled Health Services (ACCHSs) within NSW. Data were collected by Aboriginal Research Officers who were employed by the local ACCHS. The Research Officers conducted face-to-face surveys with the child and their caregiver, and conducted a clinical assessment including weight, height,

waist circumference and blood pressure measurements.

In Phase 2, families were followed up from 2014–2020. Phase 2 included surveys and clinical assessment with an additional chronic disease component in which biomarkers, including a blood lipid panel and HbA1c testing, were collected for analysis. This study used data from Phase 2.

Study sample

This study was restricted to children 5–<19 years of age who had a plausible BMI z-score recorded.¹⁷ Of these children, those who had total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL) and HbA1c recorded were included in further analysis based on these outcomes.

Study variables

The primary outcome variables were blood lipids and HbA1c. Blood lipids can be a marker for CVD and HbA1c can be a marker for diabetes.³ The thresholds used to define risk markers used in this study were based on cut-offs used for adults, given the lack of validated thresholds established for Aboriginal children.¹³ This requires additional investigation and was beyond the scope of this paper.

Cholesterol measures included in this study were total cholesterol, HDL cholesterol ('good' cholesterol) and LDL cholesterol ('bad' cholesterol). The *National Guide to a preventative health assessment for Aboriginal and Torres Strait Islander people* was used to specify the cut-offs.¹³ For total cholesterol <4.0 mmol/L is normal, 4.0–7.5 mmol/L is borderline and >7.5 mmol/L is high. For LDL cholesterol <2.0 mmol/L is normal, 2.0–3.5 mmol/L is borderline, and >3.5 mmol/L is high.¹³ For HDL cholesterol <1.0 mmol/L is low and ≥1.0 mmol/L is normal, with higher HDL being protective against CVD.¹³

HbA1c tests were used to diagnose diabetes and monitor management by analysing the levels of glucose in the blood.⁵ Since 2012, HbA1c has been accepted as the primary method for diagnosing diabetes.¹³ The *National Guide to a preventative health assessment for Aboriginal and Torres Strait Islander people* was also used to specify relevant HbA1c cut-offs. HbA1c of <5.5% (<42mmol/mol) is normal, 5.5–6.4% (42–47 mmol/mol) is borderline (prediabetic and requires further investigation), and ≥ 6.5% (≥48 mmol/mol) is diagnostically high for

diabetes.¹³ Borderline and high HbA1c were grouped together to avoid small numbers.

BMI was both an exposure and a secondary outcome. BMI and BMI z-scores were calculated for each participating child based on measured height and weight. BMI is a proxy measure of total body fat. BMI can be standardised in relation to age and sex by calculating BMI z-scores. This allows comparison of BMI across different age groups and sexes. The World Health Organization (WHO) cut-offs were used to categorise BMI in this study. This study was restricted to children aged 5 to 19 years so that all participants were subject to the same BMI z-score cut-offs.¹⁷ The WHO guidelines define underweight as a BMI z-score of <-2, normal weight as -2 to 1, overweight as 1–2, and obesity as >2 in those aged 5–19 years.¹⁷ Underweight and normal weight were grouped together to avoid small numbers. The other exposure variables were age and sex. Age was calculated based on date of birth and date at survey. Age was grouped into three categories: 5–<10 years, 10–<15 years and 15–<19 years. Sex was a binary variable coded as male and female.

Statistical analysis

The first step of the analysis was to quantify the distribution of blood lipids (total cholesterol, HDL cholesterol and LDL cholesterol), HbA1c and BMI z-scores in the cohort overall, using histograms, means and 95% confidence interval (CI) for each outcome, overall and in relation to age, sex and BMI. All table cells <5 were confidentialised.

Regression analyses were undertaken to calculate prevalence ratios (PR) for the categories outlined above and to quantify the association between each outcome and age, sex and BMI. First, univariate analyses were undertaken. Next, analyses were undertaken with additional adjustment for age and sex, where appropriate. BMI was not adjusted for when quantifying the association between age or sex and the outcomes, as BMI is potentially along the causal pathway between age or sex and the outcome of interest. An alpha level of 0.05 was the threshold for statistical significance. All statistical analyses were performed using Stata 16.

Results

This study included 507 children with a plausible BMI z-score recorded; 51.9% were female and 81.7% were aged 5-<15 years. Almost half (46.8%) were in the normal weight category, with 22.5% overweight, and 30.8% with obesity (Figure 1). The mean BMI z-score was 1.11 (95%CI 0.98-1.23); BMI z-score and obesity prevalence was higher for older age groups. The distribution of BMI was relatively similar in males and females (Table 1).

Total cholesterol

Of the 442 children who had total cholesterol recorded, 43.9% had normal, 56.1% had borderline and no individuals had high total cholesterol (Figure 1). The mean total cholesterol was 4.19 mmol/L (95%CI 4.12-4.26), with relatively similar distribution across age groups and sex. The prevalence of borderline total cholesterol increased with increasing age and was higher in those with a higher BMI (Table 1). However, there was no significant difference in the prevalence of borderline (versus normal) total cholesterol

by age group or sex. There was a 28% increase in the prevalence of having borderline (versus normal) total cholesterol for those with obesity compared to those with a normal weight (1.28, 95%CI 1.07-1.53). Results were similar after adjusting for age and sex (PR 1.28, 95%CI 1.06-1.54) (Table 2).

HDL cholesterol ('good cholesterol')

Of the 440 children who had HDL recorded, 14.6% had low, and 85.5% had normal HDL cholesterol (Figure 1). The mean HDL was 1.41 mmol/L (95%CI 1.38-1.45) with relatively

Figure 1: BMI z-scores, blood lipids and HbA1c distribution in the cohort.

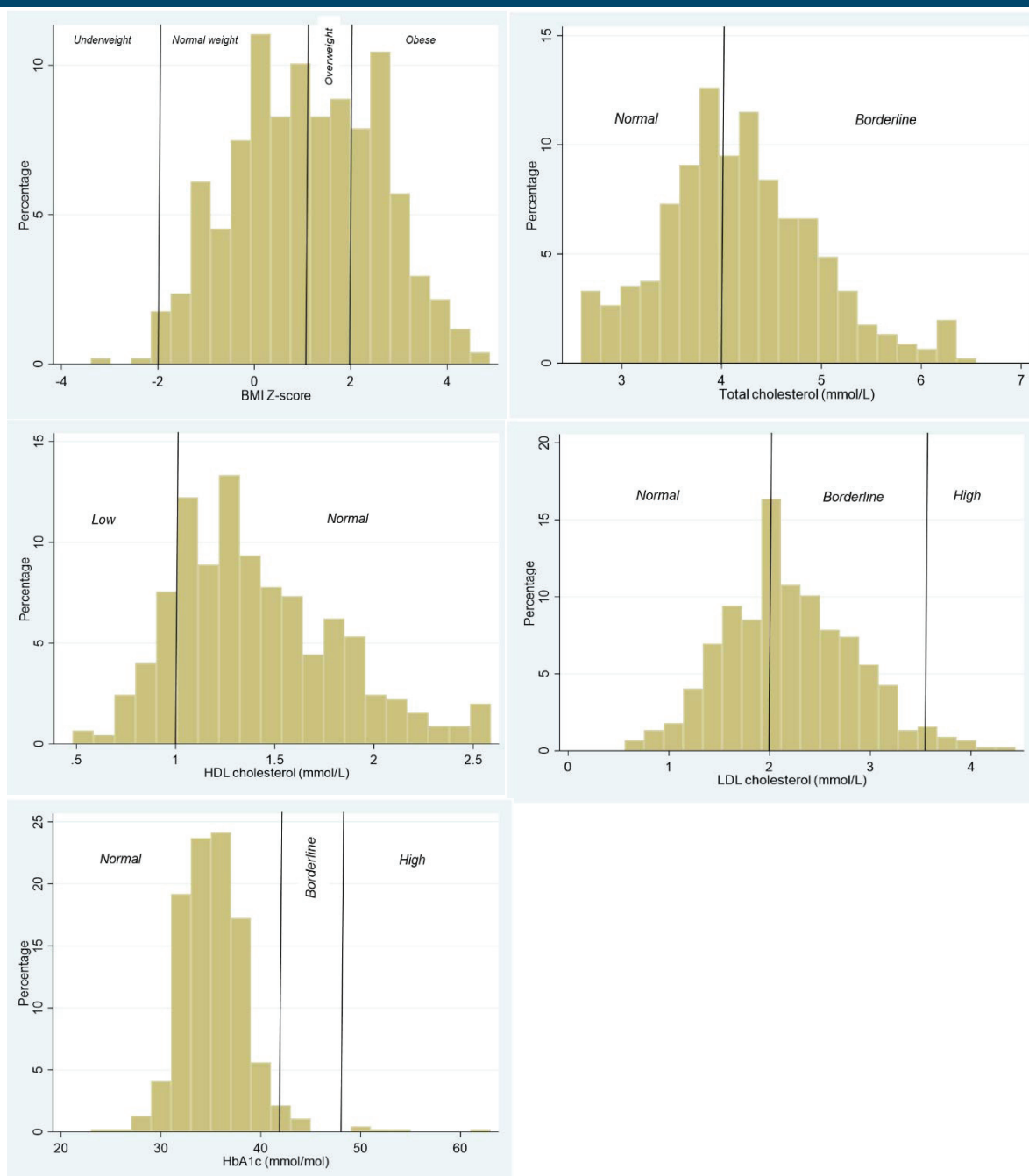


Table 1: Chronic disease risk markers distribution overall and by age group and sex.

BMI z-scores					
	Mean (95% CI)	Normal weight % (n)	Overweight % (n)	Obese % (n)	Total
Total	1.11 (0.98–1.23)	46.8 (237)	22.5 (114)	30.8 (156)	507
Age group					
5-<10	0.78 (0.58–0.99)	58.7 (115)	18.9 (37)	22.5 (44)	196
10-<15	1.22 (1.03–1.42)	41.7 (91)	23.4 (51)	34.9 (76)	218
15-<19	1.50 (1.21–1.80)	33.3 (31)	28.0 (26)	38.7 (36)	93
Sex					
Male	1.04 (0.85–1.24)	47.5 (116)	20.9 (51)	31.6 (77)	244
Female	1.16 (0.99–1.33)	46.0 (121)	24.0 (63)	30.0 (79)	263
Total cholesterol					
	Mean (95% CI) mmol/L	Normal % (n)	Borderline % (n)	High % (n)	Total
Total	4.19 (4.12–4.26)	43.9 (194)	56.1 (248)	0.0 (0)	442
Age group					
5-<10	4.18 (4.06–4.29)	45.4 (78)	54.7 (94)	0.0 (0)	172
10-<15	4.17 (4.06–4.29)	43.9 (82)	56.2 (105)	0.0 (0)	187
15-<19	4.25 (4.08–4.41)	41.0 (34)	59.0 (49)	0.0 (0)	83
Sex					
Male	4.18 (4.08–4.29)	43.3 (93)	56.7 (122)	0.0 (0)	215
Female	4.20 (4.10–4.30)	44.5 (101)	55.5 (126)	0.0 (0)	227
BMI					
Normal	4.11 (4.02–4.21)	49.0 (99)	51.0 (103)	0.0 (0)	202
Overweight	4.19 (4.03–4.34)	46.5 (46)	53.5 (53)	0.0 (0)	99
Obese	4.30 (4.16–4.44)	34.8 (49)	65.3 (92)	0.0 (0)	141
HDL cholesterol					
	Mean (95% CI) mmol/L	Low % (n)	Normal % (n)		Total
Total	1.41 (1.38–1.45)	14.6 (64)	85.5 (376)		440
Age group					
5-<10	1.49 (1.42–1.55)	11.7 (20)	88.3 (151)		171
10-<15	1.41 (1.35–1.47)	15.0 (28)	85.0 (159)		187
15-<19	1.27 (1.21–1.34)	19.5 (16)	80.5 (66)		82
Sex					
Male	1.47 (1.41–1.52)	11.2 (24)	88.8 (190)		214
Female	1.36 (1.31–1.42)	17.7 (40)	82.3 (186)		226
BMI					
Normal	1.49 (1.44–1.56)	10.5 (21)	89.6 (180)		201
Overweight	1.43 (1.35–1.52)	12.1 (12)	87.9 (87)		99
Obese	1.28 (1.22–1.34)	22.1 (31)	77.9 (109)		140
LDL cholesterol					
	Mean (95% CI) mmol/L	Normal % (n)	Borderline % (n)	High % (n)	Total
Total	2.20 (2.14–2.26)	38.3 (167)	58.7 (256)	3.0 (13)	436
Age group					
5-<10	2.19 (2.10–2.28)	41.4 (70)	57.4 (97)	<2.9 (<5)	<172
10-<15	2.16 (2.06–2.25)	37.8 (70)	58.9 (109)	3.2 (6)	185
15-<19	2.33 (2.18–2.49)	32.9 (27)	61.0 (50)	<7.2 (<6)	<83
Sex					
Male	2.15 (2.06–2.24)	39.7 (85)	56.5 (121)	3.7 (8)	214
Female	2.25 (2.17–2.34)	36.9 (82)	60.8 (135)	2.3 (5)	222
BMI					
Normal	2.12 (2.04–2.21)	42.4 (84)	55.6 (110)	<2.5 (<5)	<199
Overweight	2.20 (2.07–2.33)	36.7 (36)	61.2 (60)	<5.0 (<5)	<101
Obese	2.31 (2.19–2.43)	33.6 (47)	61.4 (86)	5.0 (7)	140
HbA1c					
	Mean (95% CI) mmol/mol	Normal % (n)	Borderline/high % (n)		Total
Total	34.87 (34.54–35.20)	97.4 (442)	2.6 (12)		454
Age group					
5-<10	34.77 (34.32–35.23)	97.8 (176)	<2.8 (<5)		<181
10-<15	35.18 (34.64–35.72)	97.4 (187)	2.6 (5)		192
15-<19	34.35 (33.46–35.24)	96.3 (79)	<6.0 (<5)		<84
Sex					
Male	34.78 (34.36–35.21)	97.4 (221)	2.6 (6)		227
Female	34.96 (34.44–35.47)	97.4 (221)	2.6 (6)		227
BMI					
Normal	34.38 (33.88–34.88)	98.1 (207)	<2.4 (<5)		<212
Overweight	34.52 (33.96–35.07)	99.0 (102)	<4.7 (<5)		<107
Obese	35.87 (35.24–36.49)	95.0 (133)	5.0 (7)		140

similar distribution across age groups and sex. The prevalence of low HDL increased with increasing age and was higher in those with a higher BMI (Table 1). However, there was no significant difference in the prevalence of low (versus normal) HDL by age group or sex. The prevalence of having low (versus normal) HDL more than doubled for those with obesity compared to those with a normal weight (PR 2.12, 95%CI 1.27–3.53); the age- and sex-adjusted PR was 2.00 (95%CI 1.19–3.35) (Table 2).

LDL cholesterol ('bad cholesterol')

Of the 436 children who had LDL recorded, 38.3% had normal, 58.7% had borderline, and 3.0% had high levels of LDL cholesterol (Figure 1). The mean LDL was 2.20 mmol/L (95%CI 2.14–2.26), with relatively similar distribution across age groups and sex. The prevalence of borderline and high LDL increased with increasing age and was higher in those with a higher BMI (Table 1). However, there was no significant difference in the prevalence of borderline/high (versus normal) LDL by age group, sex or BMI (Table 2).

HbA1c

Of the 454 children who had HbA1c recorded, 97.4% had normal and 2.6% had borderline or high HbA1c (Figure 1). The mean HbA1c was 34.87 mmol/mol (95%CI 34.54–35.20), with similar distribution across age groups and sex. The prevalence of borderline/high HbA1c increased with increasing age and was higher for those with a higher BMI (Table 1). However, there was no significant difference in the prevalence of borderline/high (versus normal) HbA1c by age group, sex or BMI (Table 2).

Discussion

The majority of children in this cohort study of urban Aboriginal children had normal cholesterol and HbA1c levels. However, there was some evidence of early chronic disease risk markers. More than half the cohort was affected by overweight or obesity and this increased in prevalence with increasing age. The prevalence of borderline total cholesterol and low HDL was significantly higher for those with obesity compared to those with normal weight. This suggests that those with obesity are likely to be at a higher risk of developing chronic disease than those in the normal weight range, even from an early age. In general, the prevalence of chronic disease

markers (total cholesterol, HDL cholesterol, LDL cholesterol and HbA1c) increased with age and BMI.

The results showed an increase in mean BMI with increasing age: 0.78 (95%CI 0.58-0.99) at 5-<10 years 1.22 (1.03-1.42) at 10-<15 and 1.50 (1.21-1.80) at 15-<19 years. Close to half the cohort had a BMI of normal weight (46.8%), and 53.3% had overweight or obesity. This was higher than the 2018-19 national average, which found 38% of Aboriginal and Torres Strait Islander children aged 2-17 years had overweight or obesity.⁷ Other studies have also found that BMI increases with increasing age and that children who are overweight are more likely to become overweight adults, leading to an increased risk of chronic disease in adulthood.⁹

Overall, a majority of the cohort had healthy cholesterol levels; there were no individuals with high total cholesterol (0.0%), few individuals with high LDL ('bad') cholesterol (3.0%), and the majority of individuals had normal HDL ('good') cholesterol (85.5%). However, cholesterol results should be interpreted with caution when analysing them in isolation as these need to be considered in the context of absolute CVD risk.¹⁸ Absolute CVD risk assessment includes multiple factors such as smoking status, blood pressure, blood lipid levels, diabetes status, age and sex to quantify the absolute risk of CVD.¹⁸ For this study, this assessment was not carried out as it was beyond the scope of the study. Given that we analysed these individual risk measures in isolation rather than in the context of an individual's absolute CVD risk, results should be interpreted with caution.¹⁸

There was no significant difference in the prevalence of chronic disease risk markers, other than BMI, between age groups or between males and females. The risk of diabetes associated with HbA1c was also found to be low in this cohort; the majority of the cohort had a normal HbA1c (97.4%) and only a few individuals had a borderline or high HbA1c (2.6%). While type 1 diabetes commonly occurs during childhood and type 2 is more common in adulthood, type 2 is still a concern for young people.⁵ In this study, individuals with obesity had higher levels of total cholesterol and lower levels of HDL compared to individuals with normal weight, both of which can increase the risk of CVD.¹³ There was a 28% (PR 1.28, 95%CI 1.06-1.54) increase in the prevalence of

Table 2: Prevalence ratios (PR) and 95% CI for blood lipids and HbA1c, by age group, sex, and BMI.

Borderline/high total cholesterol						
	Unadjusted			Adjusted for age and sex		
	PR	95% CI	P value	PR	95% CI	P value
Age group						
5-<10	1	-	-	1	-	-
10-<15	1.03	0.85 – 1.24	0.776	1.03	0.86 – 1.24	0.761
15-<19	1.08	0.86 – 1.35	0.501	1.09	0.87 – 1.36	0.472
Sex						
Male	1	-	-	1	-	-
Female	0.98	0.83 – 1.15	0.793	0.97	0.82 – 1.15	0.723
BMI						
Normal	1	-	-	1	-	-
Overweight	1.05	0.84 – 1.32	0.675	1.04	0.82 – 1.31	0.751
Obese	1.28	1.07 – 1.53	0.008	1.28	1.06 – 1.54	0.009
Low HDL cholesterol						
Age group						
5-<10	1	-	-	1	-	-
10-<15	1.28	0.75 – 2.19	0.366	1.28	0.75 – 2.19	0.357
15-<19	1.67	0.91 – 3.05	0.096	1.60	0.88 – 2.92	0.127
Sex						
Male	1	-	-	1	-	-
Female	1.58	0.99 – 2.53	0.057	1.54	0.97 – 2.47	0.070
BMI						
Normal	1	-	-	1	-	-
Overweight	1.16	0.60 – 2.26	0.663	1.07	0.55 – 2.10	0.835
Obese	2.12	1.27 – 3.53	0.004	2.00	1.19 – 3.35	0.009
Borderline/high LDL cholesterol						
Age group						
5-<10	1	-	-	1	-	-
10-<15	1.06	0.90 – 1.26	0.492	1.06	0.89 – 1.25	0.525
15-<19	1.15	0.94 – 1.40	0.179	1.14	0.93 – 1.39	0.201
Sex						
Male	1	-	-	1	-	-
Female	1.05	0.90 – 1.21	0.551	1.03	0.89 – 1.20	0.680
BMI						
Normal	1	-	-	1	-	-
Overweight	1.10	0.91 – 1.33	0.337	1.07	0.88 – 1.30	0.521
Obese	1.15	0.98 – 1.37	0.095	1.14	0.96 – 1.35	0.127
Borderline/high HbA1c						
Age group						
5-<10	1	-	-	1	-	-
10-<15	1.17	0.32 – 4.30	0.811	1.17	0.32 – 4.30	0.812
15-<19	1.65	0.38 – 7.19	0.507	1.65	0.38 – 7.21	0.507
Sex						
Male	1	-	-	1	-	-
Female	1.00	0.33 – 3.05	1.000	0.99	0.32 – 3.02	0.981
BMI						
Normal	1	-	-	1	-	-
Overweight	0.51	0.06 – 4.52	0.547	0.49	0.06 – 4.37	0.523
Obese	2.64	0.79 – 8.84	0.116	2.52	0.73 – 8.63	0.143

borderline total cholesterol for those who had obesity compared to those with in the normal weight range. The prevalence of low HDL also doubled (2.00, 1.19-3.35) for those with obesity compared to those in the normal weight range.

This is one of the first studies to analyse the distribution of chronic disease risk markers within a population of Aboriginal children. The results may contribute to improving prevention of chronic disease in the Aboriginal and Torres Strait Islander population through identification of low

and high risk groups. Current guidelines suggest screening Aboriginal and Torres Strait Islander people for CVD risk from the age of 18 years,¹² and similarly diabetes from 18 years of age, unless individuals are affected by overweight or obesity.¹³ This study reinforces that screening for chronic disease risk from this age is appropriate, particularly for those affected by chronic disease risk factors, as we found low prevalence of chronic disease risk markers at earlier ages. Due to the high prevalence of overweight and obesity within the cohort, opportunities to promote health

factors in childhood that decrease the risk of chronic disease should be further considered.

Limitations of this study included the use of adult cut-offs to assess chronic disease risk, as cut-offs for children were not available.¹³ We included underweight participants in the normal weight group due to small numbers; this might bias our results because those classified as underweight might have a different risk profile to those with normal weight. However, there were fewer than five underweight participants; therefore, this is unlikely to significantly affect our results. SEARCH involves Aboriginal children that live in urban areas and are attending Community Controlled Health Services, and may have differing health parameters than those who do not. Therefore, the results from this study may not be strictly generalisable to the Aboriginal and Torres Strait Islander population. We had limited power to detect differences in outcomes, due to the small sample size and some outcomes being uncommon in the cohort; therefore, any null results should not be over-interpreted.

Future studies could explore how social and environmental factors relate to the risk of chronic disease, to identify protective factors associated with healthy trajectories. Research has shown that caregivers experience a large number of stressful events in their life, with the most common cause being the poor health of close family members, and this has the potential to affect the health of children.¹⁹ Therefore, caregivers' health factors and lived experiences relating to their families' health could be further investigated in order to understand the relationships between the health of caregivers and children, and how this impacts on the wellbeing of families. It would also be helpful to investigate participants' self-reported health conditions and health status in the surveys in relation to the chronic disease risk markers, to assess how many of the cohort are aware of their chronic disease risk profile or have already been diagnosed with a chronic disease. As SEARCH continues, it would be valuable to repeat this research and assess the levels of total cholesterol, HDL, LDL and HbA1c in an ageing cohort in order to identify the critical ages at which risk markers emerge and affect chronic disease risk.

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