Body Mass Index Trajectories of Indigenous Australian Children and Relation to Screen Time, Diet, and Demographic Factors

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Objective: Limited cross-sectional data indicate elevated overweight/obesity prevalence among Indigenous versus non-Indigenous Australian children. This study aims to quantify body mass index (BMI) trajectories among Indigenous Australian children aged 3-6 and 6-9 years and to identify factors associated with the development of overweight/obesity.

Methods: Three-year BMI change was examined in up to 1,157 children in the national Longitudinal Study of Indigenous Children. BMI trajectories among children with normal baseline BMI ($n = 907/1,157$) were quantified using growth curve models.

Results: Baseline prevalences of overweight/obesity were 12.1% and 25.4% among children of mean age 3 and 6 years, respectively. Of children with normal baseline BMI, 31.9% had overweight/obesity 3 years later; BMI increased more rapidly for younger versus older (difference: 0.59 kg/m²/year; 95% CI: 0.50-0.69), female versus male (difference: 0.15 kg/m²/year; 95% CI: 0.07-0.23), and Torres Strait Islander versus Aboriginal (difference: 0.36 kg/m²/year; 95% CI: 0.17-0.55) children. Results were consistent with less rapid rates of BMI increase for children with lower sugar-sweetened beverage (including fruit juice) and high-fat food consumption. Children's BMI was lower in more disadvantaged areas.

Conclusions: Overweight/obesity is common, and increases rapidly, in early childhood. Interventions are required to reduce the overweight/obesity prevalence among Indigenous Australian children in the first 3 years of life and to slow the rapid overweight/obesity onset from age 3 to 9 years.

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Introduction

High body mass index (BMI) among children is an increasing problem globally (1). Childhood overweight/obesity can have severe short-term health consequences and is associated with an increased risk of obesity later in life, which has multiple comorbidities, including cardiovascular diseases, diabetes, and some cancers (2). Elevated rates of childhood overweight/obesity have been observed in Indigenous compared to non-Indigenous populations globally, including in Australia, Canada, New Zealand, and the United States (3). According to national cross-sectional data from 2011 to 2013, the combined prevalence of overweight/obesity among Australian Aboriginal and Torres Strait Islander (hereafter referred to as Indigenous) children is similar to that of non-Indigenous children at age 2-4 and 5-9 years (17.3% vs. 23.0% and

23.9% vs. 22.5%) but significantly higher at age 10-14 years (38.6% vs. 28.1%) and in most older age groups (4). Importantly, the prevalence of obesity is significantly higher among Indigenous compared to non-Indigenous Australians starting from age 5-9 years (4).

The underlying causes of obesity are complex, including interactions between individual, family, and community factors (5,6). Globally, low levels of physical activity, high levels of sedentary behavior, and energy-dense diets are understood to contribute to the high burden of obesity (5). Social inequality, dispossession, and the transition from traditional to Western diets—among other impacts of colonization—are understood to contribute to the elevated prevalence of overweight/obesity among Indigenous compared to non-Indigenous populations internationally (6,7).

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The traditional lifestyle of Indigenous Australians was characterized by high levels of physical activity and a well-balanced diet and associated with low rates of chronic diseases (8). Colonization resulted in the displacement of Indigenous people and dispossession of traditional lands. This disrupted traditional diet, lifestyle, and culture, resulting in reduced physical activity and a shift to a Western diet characterized by high intake of energy-dense foods (8,9). Colonization has also resulted in persisting socioeconomic disadvantage (8); for example, today, Indigenous compared to non-Indigenous Australians are more likely to be unemployed (10), experience food insecurity (11), have inadequate housing (12), live in disadvantaged neighborhoods (13), and have poorer health (4).

There is no robust quantitative evidence on trajectories of BMI across the childhood years in this population and insufficient evidence on the relationship between key factors and BMI. The existing longitudinal evidence on Indigenous Australian children's BMI is limited to two studies examining the persistence of high BMI among children with overweight/obesity at baseline (14) and a third study examining weight gain in 157 infants (15). Existing evidence on factors associated with BMI among Indigenous Australian children, based on cross-sectional data, suggests that the prevalence of overweight/obesity increases with age and is higher among females versus males and in urban versus remote areas (4,14).

Longitudinal analyses are necessary to identify critical periods for, and factors associated with, overweight/obesity development. Evidence specific to the Indigenous Australian population is required to enable efficient targeting of overweight/obesity prevention efforts. This paper aims to quantify BMI trajectories among Indigenous Australian children aged 3-6 and 6-9 years and to identify factors associated with the development of overweight/obesity. We hypothesize that the prevalence of overweight/obesity is high and increases from an early age and that child, family, and area-level factors are associated with childhood BMI trajectories (6).

Methods
Study population
This paper is based on data from the Longitudinal Study of Indigenous Children (LSIC), a national study managed by the Australian Government Department of Social Services. Indigenous children aged 0.5-2 years and 3.5-5 years were recruited from 11 diverse sites in 2008 through purposive sampling (see (16) for details).

Face-to-face surveys with the child and the primary caregiver (usually the mother) are conducted annually by Indigenous interviewers. Up to 1,759 children have participated across waves of the study, representing 5% to 10% of the Indigenous population in the designated age ranges. The primary caregiver reported all data utilized in the current study except children's height and weight (measured) and remoteness and area-level disadvantage (derived from participants' addresses).

In this study, we examined data from LSIC Wave 3-6 (collected 2010-2013), using Data Release 6.0.

Variables
Anthropometric measurements. LSIC interviewers measured children's weight (in light clothing) and height (without shoes) using Homedics model SC-305-AOU-4209 digital scales and Soehnle Professional Model 5003 stadiometers. Metric measurements were used to calculate children's BMI (kg/m^2).

A cleaning method based on WHO standards and protocols was applied to improve the validity of the data (17); in the current data release, measurements were also excluded if they were associated with an extreme change in BMI between consecutive waves (BMI z score change ≥ 4). We selected Wave 3 as our study baseline due to potentially reduced data reliability in earlier waves (17).

We examined BMI (kg/m^2) as a continuous, rather than categorical, outcome to maximize our ability to detect associations with BMI change. We examined raw BMI rather than BMI z scores because this is a more "stable" method for longitudinal studies (18); the within-child variability in BMI z score depends on the child's baseline BMI z score, whereas variability in BMI is not related to baseline BMI (18).

When BMI categories were examined, we classified children according to established BMI z score cutoff points, calculated using the WHO international reference. The cutoff points for overweight and obesity are more conservative for children ≤ 5 versus >5 to 18 years of age, given potentially different implications of "excess" weight at different growth stages (19). Thus, we have explicitly allowed for differences between these groups and present results separately where appropriate.

We examined BMI trajectories in a sample restricted to children with normal BMI at baseline ($n = 907/1,155$) to facilitate examination of factors related to the development of high BMI and to reduce the likelihood that exposures at baseline were caused by high baseline BMI (reverse causation). We examined baseline (rather than time-varying) measures of all exposures to minimize the impact of reciprocal causation (20).

Other variables. *A priori* variables included the child's age group at baseline (≤ 5 vs. >5 years), sex, Indigenous identification (Aboriginal, Torres Strait Islander, or both), screen time, and dietary indicators. Children were categorized as having lower versus higher screen time if they met versus exceeded the amount of screen time recommended for their age based on caregiver-reported hours spent watching TV, DVDs, or videos on a typical weekday. Children were categorized as lower versus higher consumers of sugar-sweetened beverages and high-fat foods, respectively, if they consumed these foods at <2 versus ≥ 2 occasions the preceding day, according to caregiver report.

Additional potential confounding factors included the child's and caregiver's general physical health; caregiver's social and emotional well-being, highest qualification, and employment status; family financial strain, food insecurity, and number of adults in household; and remoteness, area-level disadvantage, and caregiver perception of community safety and availability of places for children to play.

See Supporting Information File S1 for additional information on exposures.

Statistical analysis
We examined the distribution of children's BMI category across waves of the study by age group. We modeled BMI trajectories from Wave 3-6 among children with normal baseline BMI, using a

multilevel growth model (20,21). To account for the correlation structure inherent to the LSIC survey design, repeated BMI measurements were nested within children, and children were nested within their geographic area. We assumed that individual trajectories were linear but included a random effect for age (in months, centered at the mean age across waves) to allow children to differ in BMI intercept and slope (change in BMI over time). An autoregressive residual structure (AR-1) was used (see Supporting Information File S1 for details).

We examined the association between exposures at baseline and children's BMI trajectories. We included age group, sex, Indigenous identification, screen time, sugar-sweetened beverage consumption, and high-fat food consumption as *a priori* variables in our model and added to this preliminary model each factor that demonstrated an association with BMI change in unadjusted analysis. We included an interaction term between each exposure and age to test whether the exposure explained variation in children's rate of BMI change.

We conducted sensitivity analyses to examine whether we observed similar associations when using a categorical (versus continuous) measure of BMI change and to explore the potential impact of remoteness, definition of sugar-sweetened beverages, children's disability, regression dilution, and missing BMI data on findings (Supporting Information File S2). Analyses were conducted in Stata® 14.

Ethics

The LSIC survey was conducted with ethical approval from the Departmental Ethics Committee of the Australian Commonwealth Department of Health and from Ethics Committees in each state and territory, including relevant Aboriginal and Torres Strait Islander organizations. The current analysis was approved by the Australian National University's Human Research Ethics Committee (Protocol No. 2011/510).
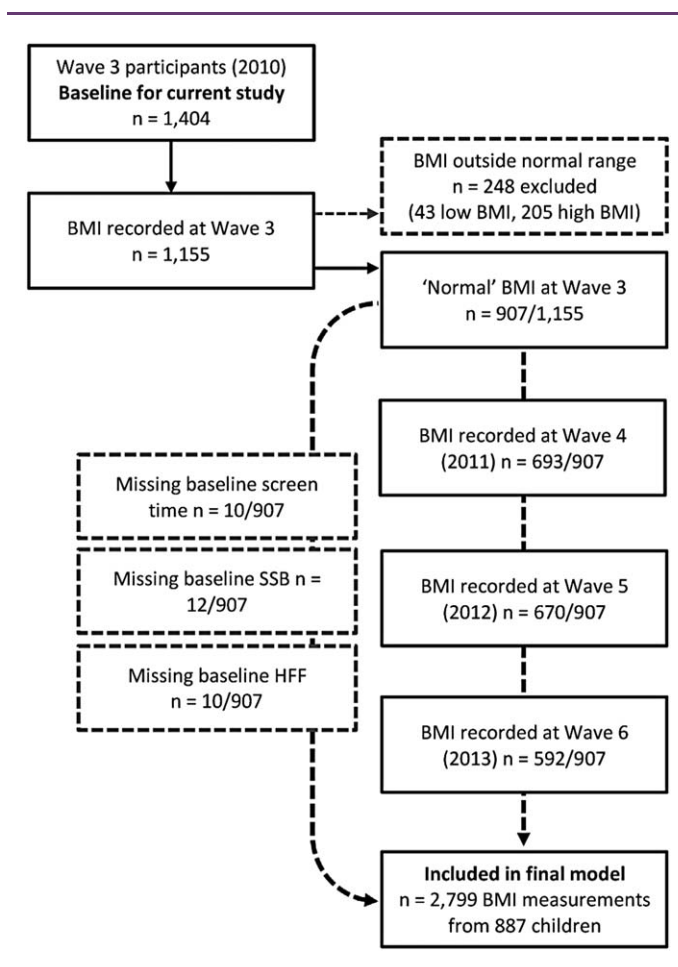

Figure 1 Flow diagram for participants in LSIC Waves 3-6 and in the current study. These figures refer to interviews with the primary caregiver.

Results

Change in BMI category over time

BMI data were available for 1,155 of the 1,404 children participating in Wave 3 (our study baseline; Figure 1). Among younger children (mean age 3 years at baseline), the prevalence of overweight and obesity, respectively, was 9.0% ($n = 60/667$) and 3.1% ($n = 21/667$) at baseline, 7.3% ($n = 37/504$) and 3.6% ($n = 18/504$) at Wave 4, 12.0% ($n = 59/491$) and 6.3% ($n = 30/491$) at Wave 5, and 25.6% ($n = 111/433$) and 13.5% ($n = 59/433$) at Wave 6 (Figure 2A). Corresponding figures for the older children (mean age 6 years at baseline) were 16.0% ($n = 78/488$) and 9.4% ($n = 46/488$), 15.2% ($n = 57/375$) and 11.7% ($n = 44/375$), 16.1% ($n = 58/361$) and 15.0% ($n = 54/361$), and 23.6% ($n = 74/313$) and 18.2% ($n = 57/313$) across successive waves. Less than 5% of children were underweight across waves.

Our trajectory analysis was restricted to children with normal baseline BMI ($n = 907/1,155$) (Figure 1). Within the younger group, 4.4% ($n = 19/429$) had become overweight and 1.0% ($n = 4/429$) had developed obesity 1 year later (at Wave 4); this increased to 10.4% ($n = 43/413$) and 3.4% ($n = 14/413$) at Wave 5 and 25.5% ($n = 93/365$) and 8.8% ($n = 32/365$) at Wave 6 (Figure 2B). Corresponding figures for the older group were: 9.1% ($n = 24/264$) and 1.5% ($n = 4/264$) at Wave 4, 12.5% ($n = 32/257$) and 3.9% ($n = 10/257$) at

22.9% ($n = 52/227$) and 5.3% ($n = 12/227$) at Wave 6. At Wave 4, 2.8% ($n = 12/429$) of younger children and 4.2% ($n = 11/264$) of older children had become underweight, 1.9% ($n = 8/413$) and 4.7% ($n = 12/257$) at Wave 5, and ≤0.8% ($n ≤ 3/365$) and 1.8% ($n = 4/227$) at Wave 6.

Association between factors and BMI over time

In addition to the variables identified for inclusion *a priori*, remoteness and disadvantage were included in our final model as they demonstrated an association with BMI change in unadjusted analysis (Table 1). We tested for differences between the younger and older group in the association between each exposure and BMI change, but these interactions were not included in the final model as there was no evidence of differential relationships by age group.

The associations of exposures to BMI intercept and annual BMI change (slope) in the final model are presented in Figure 3. Based on the final model, the mean BMI intercept was 16.46 kg/m^2 (95% CI: 16.14 to 16.76) for children in the younger group and 15.07 kg/m^2 (95% CI: 14.72 to 15.42) for children in the older group. The mean BMI intercept was significantly lower for children with low versus high sugar-sweetened beverage consumption (difference: −0.20 kg/m^2; 95% CI: −0.39 to −0.01) and those living in the
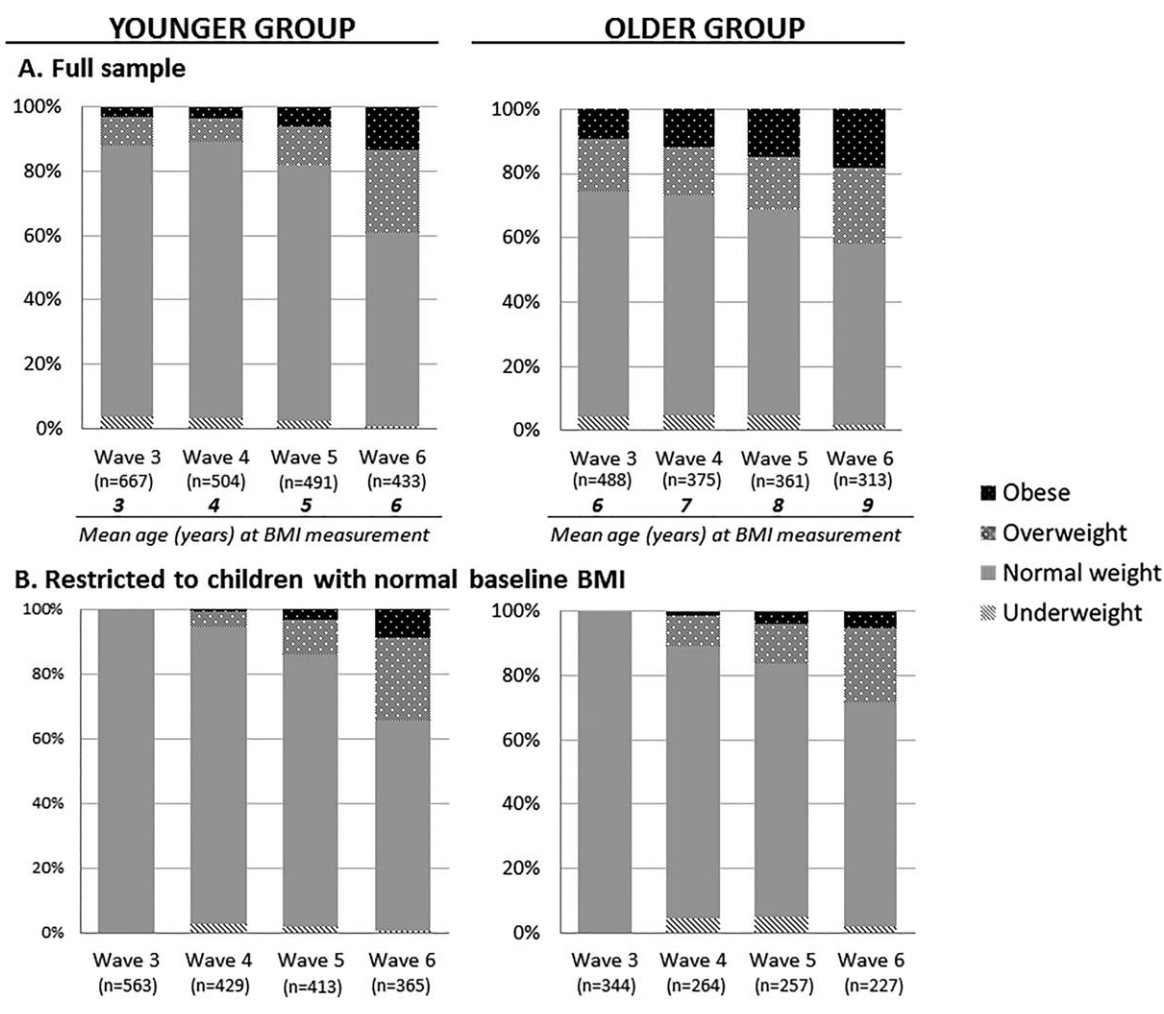
Figure 2 Distribution of BMI categories across waves of LSIC, by age group, among (**A**) the full sample and (**B**) children with normal BMI at baseline.

most disadvantaged versus most advantaged areas (difference: -0.52 kg/m^2; 95% CI: -0.91 to -0.13). We did not observe a significant difference in BMI intercept by screen time, sex, high-fat food consumption, or remoteness.

On average, children in the younger group children increased in BMI by 0.08 kg/m^2 per year (95% CI: -0.06 to 0.22), and children in the older group by 0.67 kg/m^2 per year (95% CI: 0.52 to 0.82). The rate of BMI increase was significantly higher for females versus males (difference: 0.15 kg/m^2/year; 95% CI: 0.07 to 0.23) and Torres Strait Islander versus Aboriginal children (difference: 0.29 kg/m^2/year; 95% CI: 0.11 to 0.46) (Figure 3). Low versus high consumers of high-fat foods increased in BMI at a lower rate (difference: -0.08 kg/m^2/year; 95% CI: -0.17 to 0.00), with the difference approaching significance ($P = 0.06$). We did not observe a significant difference in BMI change by screen time, sugar-sweetened beverage consumption, remoteness, or area-level disadvantage.

Figure 4 presents these results graphically to depict how key variables related to trajectories of BMI across the study period. Given the faster rate of BMI increase for female versus male and Torres Strait

Islander versus Aboriginal children, we observed higher mean BMI for these groups by age 9 years (difference approaching significance for sex; significant for Indigenous identification). Mean BMI was lower across waves for children living in the most disadvantaged versus most advantaged areas, but these differences were only significant at some waves. Predicted BMI was lower across waves for children with lower sugar-sweetened beverage and high-fat food consumption, but these differences did not reach significance.

Results of our sensitivity analyses were consistent with our primary analysis (Supporting Information File S2). The relationship between sugar-sweetened beverage consumption and BMI slope approached significance *($P = 0.05$)* when the variable definition was expanded to include fruit juice.

Discussion

We observed a high, and increasing, prevalence of overweight and obesity in this sample of Indigenous Australian children. At baseline

TABLE 1 Children's BMI at baseline and Wave 6, and BMI change, by child and family characteristics at baseline (2010)

	Wave 3 (baseline)		Wave 6		Change (Δ) in BMI from Wave 3 to Wave 6[b]				
	n	Mean BMI (95% CI)	*n*	Mean BMI (95% CI)	*n*	Mean Δ BMI (95% CI)	% ↓ in BMI z score	% ↔ BMI z score	% ↑ BMI z score
Total	907	15.6 (15.5 to 15.7)	592	16.7 (16.5 to 16.9)	592	1.0 (0.8 to 1.2)	11.5	59.6	28.9
Child factors									
Screen time									
Higher screen time	704	15.7 (15.7 to 15.8)	457	16.7 (16.6 to 16.9)	457	0.9 (0.7 to 1.1)	10.7	60.8	28.5
Lower screen time	193	15.2 (15.0 to 15.3)	129	16.5 (16.1 to 16.9)	129	1.3 (0.9 to 1.6)	14.0	55.8	30.2
Sugar-sweetened beverages[a]									
≥2	272	15.7 (15.6 to 15.9)	173	16.9 (16.6 to 17.2)	173	1.2 (0.9 to 1.5)	6.9	60.7	32.4
<2	623	15.6 (15.5 to 15.7)	409	16.6 (16.4 to 16.8)	409	0.9 (0.7 to 1.1)	13.7	58.9	27.4
High-fat foods									
≥2	277	15.7 (15.5 to 15.8)	180	17.1 (16.8 to 17.4)	180	1.3 (1.0 to 1.7)	9.4	57.2	33.3
<2	620	15.6 (15.5 to 15.7)	405	16.5 (16.3 to 16.7)	405	0.9 (0.7 to 1.0)	12.6	60.5	26.9
Sex									
Male	452	15.8 (15.7 to 15.9)	290	16.5 (16.3 to 16.7)	290	0.7 (0.5 to 0.9)	12.1	62.4	25.5
Female	455	15.5 (15.4 to 15.6)	302	16.9 (16.6 to 17.1)	302	1.3 (1.1 to 1.6)	10.9	57.0	32.1
Age group at baseline[a]									
≤5 years	563	16.0 (15.9 to 16.1)	365	16.4 (16.2 to 16.6)	365	0.4 (0.2 to 0.5)	14.3	61.4	24.4
>5 years	344	15.0 (14.9 to 15.2)	227	17.2 (16.9 to 17.5)	227	2.1 (1.8 to 2.3)	7.1	56.8	36.1
Indigenous identification									
Aboriginal	802	15.6 (15.5 to 15.7)	526	16.7 (16.5 to 16.8)	526	1.0 (0.8 to 1.1)	11.4	59.9	28.7
Torres Strait Islander	59	15.6 (15.3 to 16.0)	40	17.3 (16.4 to 18.2)	40	1.7 (0.7 to 2.6)	15.0	50.0	35.0
Both	46	15.7 (15.3 to 16.0)	26	16.3 (15.5 to 17.2)	26	0.8 (0.0 to 1.6)	7.7	69.2	23.1
General physical health									
Poor, fair, or good	216	15.5 (15.3 to 15.6)	133	16.7 (16.3 to 17.1)	133	1.1 (0.8 to 1.4)	18.7	50.4	30.9
Very good or excellent	687	15.7 (15.6 to 15.8)	456	16.7 (16.5 to 16.9)	456	1.0 (0.8 to 1.2)	9.5	62.0	28.5
Caregiver and family factors									
Caregiver's general physical health									
Poor, fair, or good	494	15.6 (15.5 to 15.7)	316	16.8 (16.5 to 17.0)	316	1.1 (0.8 to 1.3)	11.1	59.5	29.4
Very good or excellent	408	15.7 (15.5 to 15.8)	272	16.6 (16.4 to 16.8)	272	0.9 (0.7 to 1.2)	12.1	59.6	28.3
Caregiver's social and emotional well-being									
High distress	134	15.5 (15.3 to 15.8)	86	16.8 (16.4 to 17.2)	86	1.2 (0.8 to 1.7)	9.3	60.5	30.2
Low distress	732	15.7 (15.6 to 15.7)	485	16.7 (16.5 to 16.9)	485	1.0 (0.8 to 1.2)	12.2	58.4	29.5
Financial strain									
Run out of money	114	15.7 (15.5 to 15.9)	75	16.7 (16.3 to 17.2)	75	1.0 (0.6 to 1.5)	8.0	61.3	30.7
Just enough money	446	15.7 (15.6 to 15.8)	281	16.8 (16.5 to 17.0)	281	1.0 (0.7 to 1.2)	14.6	55.9	29.5
Can save money	337	15.5 (15.4 to 15.7)	230	16.6 (16.3 to 16.8)	230	1.0 (0.7 to 1.3)	9.1	63.5	27.4
Went without meals in past year									
Yes	74	15.1 (14.8 to 15.4)	39	15.7 (15.1 to 16.3)	39	0.5 (−0.1 to 1.1)	18.0	59.0	23.1
No	820	15.7 (15.6 to 15.8)	543	16.8 (16.6 to 16.9)	543	1.0 (0.9 to 1.2)	11.1	59.3	29.7
Primary caregiver employment									
Not employed	622	15.6 (15.5 to 15.7)	400	16.6 (16.4 to 16.8)	400	1.0 (0.8 to 1.2)	11.0	60.3	28.8
Employed part time	147	15.8 (15.6 to 16.0)	107	16.9 (16.5 to 17.3)	107	1.0 (0.7 to 1.4)	10.3	61.7	28.0
Employed full time	125	15.6 (15.4 to 15.8)	77	16.7 (16.2 to 17.2)	77	0.9 (0.4 to 1.5)	16.9	53.3	29.9
Primary caregiver education									
Less than Year 12	493	15.5 (15.4 to 15.6)	310	16.5 (16.3 to 16.8)	310	0.9 (0.7 to 1.2)	11.9	60.0	28.1
Year 12 or beyond	346	15.8 (15.6 to 15.9)	243	16.9 (16.6 to 17.2)	243	1.1 (0.8 to 1.4)	11.5	59.7	28.8
Number of adults in household									
1	253	15.6 (15.4 to 15.8)	155	16.7 (16.4 to 17.0)	155	1.1 (0.8 to 1.4)	10.3	56.8	32.9
2	437	15.7 (15.6 to 15.8)	288	16.7 (16.4 to 16.9)	288	0.9 (0.7 to 1.1)	11.5	61.5	27.1
≥3	91	15.3 (15.1 to 15.6)	57	16.9 (16.3 to 17.6)	57	1.4 (0.9 to 2.0)	7.0	64.9	28.1

TABLE 1. (continued).

	Wave 3 (baseline)		Wave 6		Change (Δ) in BMI from Wave 3 to Wave 6[b]				
	n	Mean BMI (95% CI)	*n*	Mean BMI (95% CI)	*n*	Mean Δ BMI (95% CI)	% ↓ in BMI z score	% ↔ BMI z score	% ↑ BMI z score
Area-level factors									
Remoteness[a]									
None	241	15.8 (15.6 to 16.0)	174	16.7 (16.4 to 17.0)	174	0.9 (0.6 to 1.2)	13.2	60.9	25.9
Low	450	15.7 (15.5 to 15.8)	285	16.9 (16.7 to 17.2)	285	1.2 (1.0 to 1.5)	7.7	60.4	31.9
Moderate	125	15.4 (15.1 to 15.6)	82	16.2 (15.7 to 16.6)	82	0.6 (0.2 to 1.0)	17.1	61.0	22.0
High/extreme	91	15.3 (15.0 to 15.6)	51	16.3 (15.7 to 16.9)	51	1.0 (0.4 to 1.6)	17.7	49.0	33.3
Area-level disadvantage[a]									
Most advantaged	168	15.8 (15.6 to 16.0)	122	16.8 (16.4 to 17.1)	122	0.9 (0.6 to 1.3)	13.1	59.8	27.1
Middle advantage	554	15.7 (15.6 to 15.8)	362	16.9 (16.7 to 17.1)	362	1.2 (1.0 to 1.4)	9.4	59.1	31.5
Most disadvantaged	185	15.2 (15.0 to 15.4)	108	15.8 (15.5 to 16.2)	108	0.5 (0.1 to 0.8)	16.7	61.1	22.2
Safe community									
No (not so safe or really bad)	123	15.5 (15.3 to 15.8)	72	16.7 (16.2 to 17.2)	72	1.1 (0.6 to 1.6)	11.1	58.3	30.6
Yes (okay, safe, or very safe)	743	15.6 (15.6 to 15.7)	495	16.7 (16.5 to 16.9)	495	1.0 (0.8 to 1.2)	11.7	59.4	28.9
Places to play									
No (not many or none)	241	15.5 (15.4 to 15.7)	139	16.7 (16.3 to 17.0)	139	1.0 (0.6 to 1.4)	9.4	64.8	25.9
Yes (some, a few, or lots)	625	15.7 (15.6 to 15.8)	426	16.7 (16.5 to 16.9)	426	1.0 (0.8 to 1.2)	12.2	57.5	30.3

The sample includes children with a BMI in the normal range at baseline. Total number may vary across exposure categories due to missing data.
[a]Association between exposure and category of BMI change (*P* value for Pearson $\chi^2 < 0.20$) and/or significant difference in mean BMI change across exposure categories.
[b]Change in BMI assessed based on BMI *z* scores to account for the expected differences in BMI change over time for children of different ages. Research has demonstrated improved health outcomes and decreased fat mass for children with obesity who experience decreases in BMI *z* score greater than 0.5 and 0.6 units per year, respectively, but there is no standard definition for a "significant" increase in BMI *z* score for children. Thus, we have conservatively defined BMI decrease (average annual BMI *z* score change of ≤-0.3), BMI maintenance (average annual BMI *z* score change between −0.3 and 0.3), and BMI increase (average annual BMI *z* score change of ≥0.3).

(2010), the prevalence of combined overweight/obesity was 12% and 25% among children of mean age 3 and 6 years, respectively. These figures increased to 39% and 42% 3 years later, when children were a mean age of 6 and 9 years, respectively. It is difficult to directly compare these prevalence estimates with national estimates from 2011-2013 due to differences in sampling approaches, definitions of overweight/obesity, and age categorization, but findings are consistent with a high (12%-22%) overweight/obesity prevalence among Indigenous children aged 2 to 4 years, increasing to around 40% at age 10 years (4). Almost one-third of children with normal baseline BMI had developed overweight/obesity 3 years later. We are unaware of other longitudinal data about Indigenous Australian children for comparison, but in a non-Indigenous Australian sample, just over 20% of children with normal BMI at age 4 to 5 years had developed overweight/obesity 6 years later (22). Our findings indicate that interventions are required both to reduce the prevalence of overweight/obesity in the first 3 years of life and to slow the rapid onset of overweight/obesity from age 3 to 9 years.

The faster rate of annual BMI increase observed for children aged 6-9 versus 3-6 years is consistent with the average BMI observed at these ages in the WHO Reference data. The higher mean BMI at baseline and higher 3-year incidence of overweight/obesity in the younger versus older group may be an artifact of the different BMI cutoff points used (Supporting Information File S3).

When comparing the two groups of children when they are each around 6 years of age (younger children at Wave 6, older children at Wave 3), in the whole sample—not restricted to those with normal baseline BMI—we observed a higher overweight/obesity prevalence in the younger group (39.3% vs. 25.4%). Given that both groups are the same age and therefore subject to the same cutoff points, the prevalence difference may reflect differences between the two cohorts or a temporal trend toward an increasing prevalence of overweight/obesity. Bearing in mind differences in methodology, the existing national data are consistent with an increase in the prevalence of overweight/obesity among Indigenous Australian children since 1994 (4,23), in line with international trends (1).

Consistent with cross-sectional evidence of an elevated obesity prevalence (4,14,24), we identified a faster rate of BMI increase for female versus male and for Torres Strait Islander versus Aboriginal children, suggesting that these groups may be at increased risk of developing overweight/obesity from an early age. Previous research identified similar BMI trajectories for male and female children in the general Australian population (25), but this has not previously been explored among Indigenous Australian children; differences between groups in unmeasured factors related to BMI (e.g., physical activity) may partially explain our findings. Given the observed tracking of weight status across the life course (2), females may face an increased risk of overweight/obesity during the childbearing years, incurring an increased risk of overweight/obesity among their offspring (26). Therefore, promoting healthy BMI among female Indigenous children from early childhood should be a priority to prevent spiraling increases in overweight/obesity prevalence.

Mean intercept and slope*	Younger group	Older group
Mean intercept (BMI) [95%CI]	16.46 [16.14,16.76]	15.07 [14.72,15.42]
Mean slope (ΔBMI/year) [95%CI]	0.08 [-0.06,0.22]	0.67 [0.52,0.82]

Difference in intercept and slope coefficients, by exposure categories

	n	Difference in intercept* from referent [95%CI]	Difference in slope coefficient from referent [95%CI]
Screen-time			
Higher screen-time	695	0 [ref]	0 [ref]
Lower screen-time	192	-0.20 [-0.42,0.03]	0.03 [-0.04,0.17]
Sugar-sweetened beverages†			
≥2	270	0 [ref]	0 [ref]
<2	617	-0.20 [-0.39,-0.01]	-0.05 [-0.14,0.03]
High-fat foods			
≥2	274	0 [ref]	0 [ref]
<2	613	-0.15 [-0.34,0.04]	-0.08 [-0.17,0.00]
Sex‡			
Male	439	0 [ref]	0 [ref]
Female	448	-0.02 [-0.19,0.15]	0.15 [0.07,0.23]
Indigenous identification‡			
Aboriginal	782	0 [ref]	0 [ref]
Torres Strait Islander	59	0.29 [-0.11,0.70]	0.29 [0.11,0.46]
Both	46	-0.07 [-0.48,0.33]	-0.02 [-0.20,0.16]
Level of Relative Isolation			
None	234	0 [ref]	0 [ref]
Low	438	-0.10 [-0.37,0.16]	0.11 [0.01,0.22]
Moderate	125	-0.31 [-0.70,0.07]	-0.02 [-0.18,0.14]
High/Extreme	90	-0.14 [-0.60,0.32]	-0.03 [-0.22,0.16]
Area-level disadvantage†			
Most advantaged	165	0 [ref]	0 [ref]
Middle advantage	539	-0.03 [-0.31,0.25]	-0.06 [-0.18,0.05]
Most disadvantaged	183	-0.52 [-0.91,-0.13]	-0.12 [-0.28,0.04]

	Estimate [95%CI]
Variance between clusters	0.05 [0.01,0.21]
Variance between individuals' slopes	0.00 [0.00,0.00]
Variance between individuals' intercepts	0.87 [0.70,1.08]
Correlation between measurements w/in individuals	0.88 [0.83,0.91]
Residual variance	1.29 [1.12,1.49]

Figure 3 Mean BMI intercept and slope for children by age group and association with exposures. Based on the final model, which was adjusted for all variables shown and age group; repeated BMI measurements were nested within children, and children were nested within their geographic area. This includes 887 children, providing 2,799 observations of BMI: 553, 421, 406, and 358 BMI measurements of children in the younger group across waves and 334, 257, 250, and 220 measurements of children in the older group across waves. *We estimated the mean BMI intercept across exposure categories for children in the younger and older age group. BMI intercept was calculated at the mean age of the sample across waves (centered age = 0) and all other covariates at the value of the referent group: exceeded recommended hours of screen time, at least two sugar-sweetened beverages, at least two high-fat foods, male, Aboriginal, no remoteness, most advantaged area. We estimated the mean BMI slope (annual change in BMI) across exposure categories for children in the younger and older age group, with BMI calculated at the mean age of younger/older children at baseline and all other covariates at the value of the referent group. †Significant association between exposure variable and BMI intercept (P value for Wald test <0.05). ‡Significant association between exposure variable and BMI slope (P value for Wald test <0.05).

Consumption of unhealthy foods was common; around 30% of children reportedly consumed sugar-sweetened beverages or high-fat foods on at least two occasions on the day preceding interview. This fits with contemporary national data estimating that unhealthy foods constitute 38.4% of Indigenous children's total energy intake and that sugar-sweetened beverage consumption is high from age 2 to 3 years (11). Although they did not reach statistical significance, our findings are consistent with a lower annual BMI increase for children who consumed fewer sugar-sweetened beverages (P = 0.05 when including fruit juice) and high-fat foods (P = 0.06) at baseline. The magnitude of the association observed between sugar-sweetened beverage consumption and BMI is consistent with findings of a meta-analysis of prospective studies (27). Findings for other dietary indicators (such as high-fat or snack foods) have been less consistent; this has been attributed to variation in the foods examined and limitations of standard methods of assessing dietary intake (28).

Within this relatively disadvantaged population, children in more disadvantaged versus advantaged areas tended to have lower BMI. This is consistent with previous findings from LSIC (29) and with the socioeconomic patterning of child obesity in low- and middle-income countries (30), but it contrasts the inverse relationship between BMI and socioeconomic advantage established in the general Australian population and in other developed countries (31,32). In our sample, the prevalence of underweight was elevated among children in more disadvantaged versus advantaged areas, alongside a relatively lower prevalence of overweight/obesity (Supporting Information File S4). Our findings support a persisting "double burden of malnutrition" (i.e., the co-occurrence of underweight and
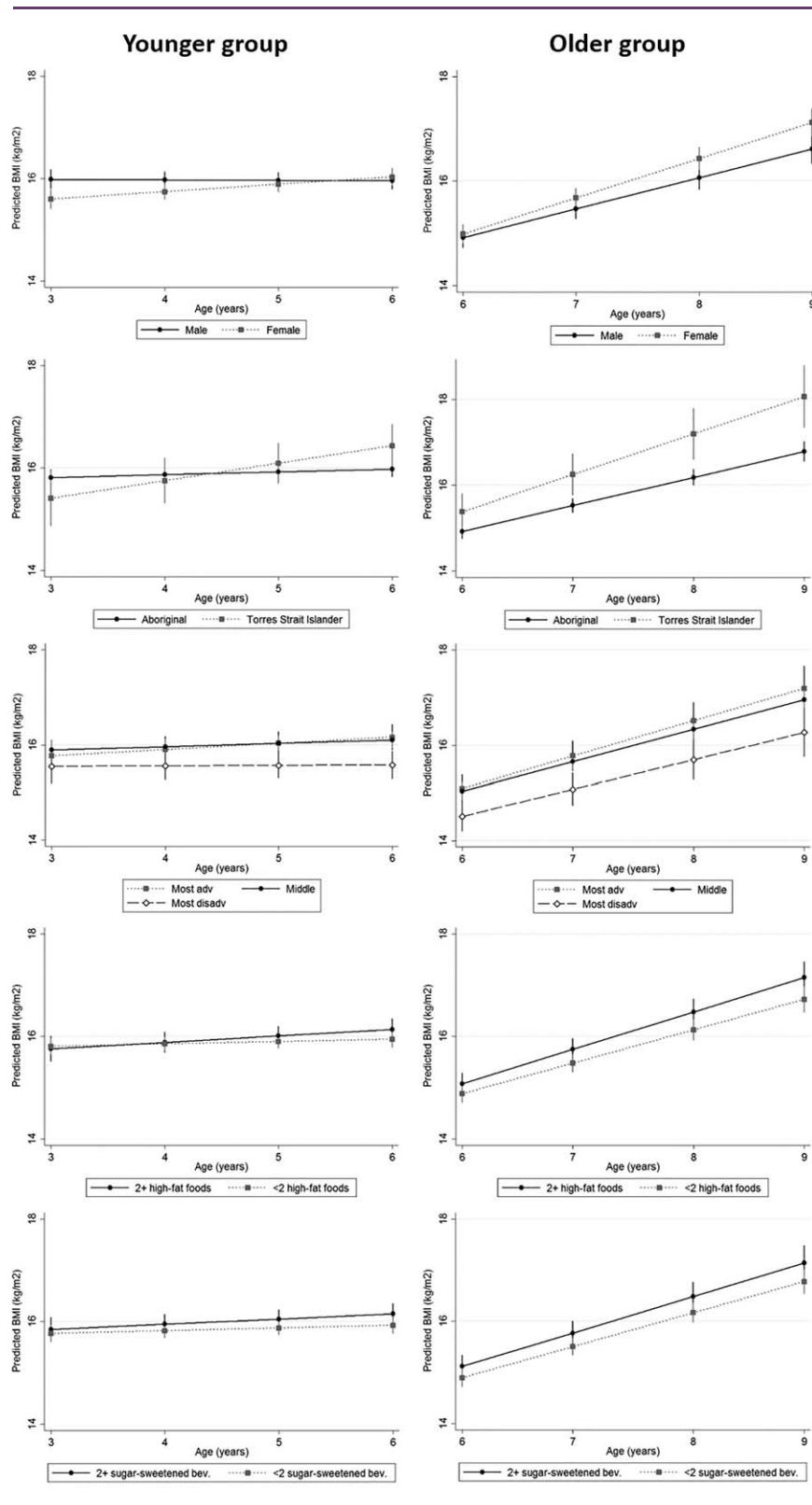
Figure 4 Predicted BMI across waves by children's sex, Indigenous identification, and baseline area-level disadvantage, high-fat food consumption, and sugar-sweetened beverage consumption, for children in the younger and older age group. Based on the final model, which was adjusted for screen time, sugar-sweetened beverage consumption, high-fat food consumption, age, sex, Indigenous identification, remoteness, and area-level disadvantage. It includes BMI measurements for 553, 421, 406, and 358 children in the younger group across waves and 334, 257, 250, and 220 children in the older group across waves. Population-averaged estimates of BMI were calculated (using the margins and marginsplot commands in Stata) at the mean age of younger/older children at each wave and based on the distribution of all other covariates within the study population.

overweight/obesity within families and/or communities), which has been observed in Indigenous populations internationally (7); efforts to promote healthy BMI need to consider undernutrition and overnutrition together (33).

We did not observe differences in BMI trajectories by remoteness, contrasting findings from cross-sectional research (14,24). Previous findings were not adjusted for area-level disadvantage, which is strongly linked to remoteness; our findings may indicate that the relationship between remoteness and children's BMI is largely explained by area-level disadvantage. Although the overall association between remoteness and BMI change was not significant, we did observe a significantly faster rate of BMI increase in areas with "low" versus "no" remoteness. We did not have any *a priori* hypotheses about differences in BMI change between these two settings, and thus results are hard to interpret.

Only 21.5% of children in the sample met screen time recommendations, with reported hours of screen time exceeding figures previously published for Indigenous children (34). Our analyses did not provide evidence that higher, versus lower, screen time at baseline was associated with greater increases in BMI over time. There is strong international evidence from cross-sectional data that people with obesity have increased screen time (35,36), but there is not conclusive evidence that increased screen time leads to increased BMI over time (28,37). Based on our findings, we can exclude a large prospective effect of baseline screen time on BMI change in this sample, consistent with previous research employing longitudinal methods (35,37,38).

We may have underestimated the association between screen time and BMI in our sample because of the short duration of our study (36,37), the limited sample size, imprecise measurement of screen time, and regression dilution (see Supporting Information File S2). The accuracy of our measure of screen time may be reduced due to reliance on caregiver report, although most of the current evidence is based on indirect measures (35), and because it did not include hours spent playing electronic games. However, electronic game use is estimated to constitute a small proportion of total screen time by Indigenous Australian children, so the resulting underestimation of screen time is likely to be small (34).

The imprecise measurement of dietary intake may have resulted in the underestimation of associations with BMI or in residual confounding (27,37). Reported intake according to 24-hour recall does not always reflect usual intake, particularly for items consumed infrequently (28,39). Furthermore, intake is based on caregiver report of the number of occasions the child consumed these foods, with no information on portion size, which might have resulted in misclassification. However, the available data can provide a crude representation of consumption patterns; data collected using this method have been utilized in other studies (28,39), and we have used similar variable definitions when possible for consistency. A substantial limitation of our study, as in similar studies (28,39), is that we could not adjust for children's physical activity or energy expenditure, as these data were not collected in LSIC.

A potential limitation of this study is the extent of missing data on children's BMI across waves of the study; however, we did not observe a significant difference in dropout across key exposure variables, suggesting that unbalanced missing data were not biasing our estimation of BMI slope.

Conclusion

The use of robust longitudinal methods enabled the first quantification of BMI trajectories for Indigenous Australian children and the first quantification of variation by key factors. More than 10% of children already had overweight/obesity by age 3 years, and we observed a rapid onset of overweight/obesity between age 3-6 and 6-9 years. This indicates the need for interventions to reduce the prevalence of overweight/obesity in the first 3 years of life and to slow the rapid onset of overweight and obesity from age 3 to 9 years. Efforts to promote healthy BMI trajectories among female children are of particular priority.

There is limited evidence on what works to improve the weight status of Aboriginal and Torres Strait Islander children. Our findings suggest that reducing consumption of sugar-sweetened beverages and high-fat foods from an early age could have a beneficial impact on children's BMI trajectories; despite the small magnitude of observed effects, reducing consumption could have a substantial impact at the population level given the high level of current consumption (36). However, it is imperative that programs and policy are developed in partnership with Indigenous communities, address the broader sociocultural and environmental context in which health behaviors occur (40), and are sustainable.O

Acknowledgments

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