Original Article

Risk factors for excess body fatness in New Zealand children

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Objective: To identify demographic and lifestyle risk factors for excess body fatness in a multiethnic sample of New Zealand children. **Design:** Cross-sectional study. **Participants:** A total of 1229 European, Polynesian, Asian, and 'Other' children aged 5-11 y (603 male, 626 female) living in New Zealand. **Measurements:** Percentage body fat (%BF) was measured using hand-to-foot bioelectrical impedance analysis, and overfat participants were defined as those with a %BF greater than 25% (boys) and 30% (girls). A parent proxy questionnaire was developed for assessing demographic and lifestyle factors, and multiday memory pedometers were used to estimate physical activity levels over five days. **Results:** After controlling for differences in sex, age, and socioeconomic status (SES), Asian children were more likely to have excess body fat than European children. The adjusted odds of overfat also increased with age and decreased with SES. Three lifestyle risk factors related to fat status were identified: low physical activity, skipping breakfast, and insufficient sleep on weekdays. Clustering of these risk factors resulted in a cumulative increase in the prevalence of overfat. Active transport, sports participation, lunch bought at school, fast food consumption, sugary drink consumption, and weekend sleep duration were not associated with fat status after adjustment for the selected demographic variables. **Conclusion:** The findings from this study enhance our understanding of the risk factors for excess body fatness in New Zealand children, and highlight key demographic and lifestyle priorities for future interventions.

Key Words: obesity, ethnicity, diet, physical activity, sleep

INTRODUCTION

The prevalence of overweight and obesity in children has reached epidemic proportions in many countries. Given the long-term economic and public health consequences associated with childhood obesity, the development of preventative strategies for reducing the accretion of excess fat in young people is essential. However, obesity is a complex disorder that is modulated by interactions between environmental and behavioral factors, and consequently isolating the key predictors of obesity in young people can be challenging.

Potential risk factors for obesity are commonly categorized as either demographic (non-modifiable) or lifestyle (modifiable) factors. An understanding of the demographic risk factors related to obesity can help prioritize the population groups to be targeted by public health initiatives. For example, there is evidence that the prevalence of childhood obesity in developed countries is relatively high among certain ethnic minorities[1-3] and in those from underprivileged backgrounds.[4-6] Nonetheless, the interplay between socioeconomic status (SES) and ethnicity and their effects on obesity remain unclear. It has been hypothesized that the high occurrence of obesity in some ethnic minorities is due to their overrepresentation in low socioeconomic regions.[7] Subsequent research suggests that SES is not the only contributor to ethnic differences in childhood obesity, but that other ethnic-specific variables (e.g., body composition, culture, maturation) may have important roles.[8-10]

While demographic risk factors are useful for isolating the population groups most susceptible to obesity, lifestyle factors reflect the underlying behaviors that promote excessive fat accumulation. A wide range of potential lifestyle risk factors for childhood obesity have been investigated with variable results.[11] The most consistent predictors of obesity in children are low levels of physical activity,[12-15] unhealthy dietary patterns,[16-20] and insufficient sleep.[4,21-23] However, there is limited information describing the interactions among multiple risk factors and their cumulative (or confounding) effects on children's adiposity. The only study to address this issue investigated overweight and obesity in German children aged 5-7 years with various combinations of three demographic risk factors (parental overweight, low SES, and high birth weight).[5] On average, children that had two risk factors were more likely to be overweight or obese

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than those with single risk factors, with the highest prevalence values observed when all three risk factors were combined. The potential effects of clustering modifiable risk factors have yet to be assessed.

Further complications arise when the different methods used to classify obesity in children are considered. Most previous studies have used age- and sex-specific body mass index (BMI) percentiles to define obesity. This is despite evidence that BMI may not provide an equivalent estimate of body fatness across different ethnic groups due to its inability to distinguish between fat and fat-free mass.[24,25] Furthermore, the relationship between the BMI percentiles used to define childhood obesity and negative health outcomes is uncertain. As such, risk factors for BMI-determined obesity may not necessarily correspond to a higher risk of morbidity. Several studies have proposed sex-specific percentage body fat (%BF) limits ranging from 20% to 30% that coincide with elevated risk of health complications in children.[26-28] These health-related %BF criteria may provide more relevant reference points for determining obesity risk factors than existing BMI standards.

It is clear that an awareness of the factors associated with excessive fat accrual in children is essential to counteract the current obesity trends. Despite evidence that the average BMI of young New Zealanders is rapidly increasing,[29] there is no information describing the predictors of body fatness in this population. Thus, the primary objective of this study was to identify key demographic and lifestyle factors associated with increased risk of excess adiposity in a multiethnic sample of New Zealand children. A secondary objective was to examine the effects of risk factor clustering on the prevalence of overfat in children.

METHODS

Participants

A total of 1 229 children (603 boys, 626 girls) aged 5-11 years were randomly selected from 27 primary (elementary) schools in Auckland, New Zealand. Participating schools were purposively sampled to replicate the geographic and socioeconomic distribution of primary schools in the Auckland district. The ethnic composition of the sample was 46.8% European (283 boys, 292 girls), 33.1% Polynesian (201 boys, 206 girls), 15.9% Asian (99 boys, 97 girls), and 4.1% from other ethnicities (20 boys, 31 girls). The Polynesian ethnic group composed of Pacific Island (58.2%) and Maori (41.8%) children, and the Asian group composed of Indian (38.3%), Chinese (21.9%), Korean (13.8%), Filipino (9.7%), Sri Lankan (4.1%), and Other Asian (12.2%) children. SES was estimated using the Ministry of Education decile classification system for New Zealand primary schools. For the purposes of this study, participants from schools with a decile rating between 1 and 3 were categorized into the 'Low' SES group, and those from schools rated between 4 to 7 and between 8 to 10 were considered 'Middle' and 'High', respectively. Ethical approval for this study was obtained from the Auckland University of Technology ethics committee. Written informed consent was provided by each participant and his or her legal guardian.

Measurements and procedures

The height of each participant was measured to the nearest millimeter with a portable stadiometer (Design No. 1013522, Surgical and Medical Products, Seven Hills, Australia), and weight was assessed to the nearest 0.1 kg on a digital scale (Model Seca 770, Seca, Hamburg, Germany). Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Resistance (R) measurements were obtained at 50 kHz using a bioelectrical impedance analyzer (Model BIM4, Impedimed, Capalaba, Australia) with a tetrapolar arrangement of self-adhesive electrodes (Red Dot 2330, 3M Healthcare, St Paul, MN, USA). After swabbing the skin on the right hand and foot with alcohol, source electrodes were placed on the dorsal surface of the foot over the distal portion of the second metatarsal, and on the hand on the distal portion of the second metacarpal. Sensing electrodes were placed at the anterior ankle between the tibial and the fibular malleoli, and at the posterior wrist between the styloid processes of the radius and ulna. Testing was initiated after the participants emptied their bladder, and had been lying supine with their arms and legs abducted for at least 10 min. Testing was completed when repeated measurements of R were within 1 Ω of each other. Fat and fat-free mass were calculated from R, height, and weight using a prediction equation previously validated in New Zealand children.[25] %BF was derived as fat mass divided by weight and multiplied by 100. Boys and girls were classified as 'overfat' if their %BF exceeded 25% and 30% (respectively).[28]

Sealed multiday memory pedometers (Model NL-2000, New Lifestyles Inc, Lee's Summit, MO) were used to measure daily steps over three weekdays and two weekend days. An overall mean step count was obtained after scaling the averaged data by a ratio of five weekdays to two weekend days. Recent step count guidelines of 16 000 (boys) and 13 000 (girls) steps per day[30] were used to categorize participants into 'active' and 'inactive' groups. Data were excluded if participants did not wear their pedometer for more than one hour on a given day (as determined by a parent proxy questionnaire). Daily step counts below 1 000 or above 30 000 were regarded as outliers and were removed.[31]

A proxy questionnaire was administered to the parents of each participant to collect information about potential risk factors for obesity while avoiding the recall error associated with self-report surveys in children.[32] Parents were asked how their child usually traveled to and from school, with responses grouped into active (walking, cycling) or motorized transport (car, bus, train). Frequency of breakfast, fast food, and sugary drink consumption in the last full week was determined (servings per week), as were the number of weekdays the participants purchased lunch at school (zero to five days). Participation in organized sport outside of school was assessed for the last full week (zero to seven days). Parents were also asked what time their child usually goes to bed and gets up from bed on weekdays and weekends. The difference between the two times provided the total minutes of sleep for each participant.

Statistical analyses

Data were analyzed using SPSS version 14.0 for Windows (SPSS Inc., Chicago, IL). Differences in participant characteristics (age, height, weight, BMI, and %BF) between sexes and among ethnic groups were assessed by two-way ANOVA, with significant associations examined by pairwise comparisons using *t*-tests. Logistic regression analysis was used to investigate associations between excess body fatness and selected demographic and lifestyle variables. Odds ratios for each category were adjusted for the four demographic factors (sex, age, ethnicity, and SES), and then by all 13 factors

concurrently. Ethnic differences in the frequencies of sex, age, and SES categories were examined using the chi-squared test. Analysis of covariance was used to compare the mean %BF between children with and without selected risk factor clusters while adjusting for age, ethnicity, and SES. Differences in the prevalence of overfat were examined using chi-squared analysis. A p value less than 0.05 was used to indicate statistical significance.

RESULTS

The physical characteristics of the three major ethnicities in this study are presented in Table 1. No significant differences in age or height were observed between sexes or among ethnic groups. Polynesian boys and girls were heavier and had higher BMI values than their European and Asian counterparts. European children had less body fat than Polynesian and Asian children of the same sex, although the difference between European and Asian girls was not statistically significant. Boys averaged less body fat than girls across the whole sample and within each ethnic group.

Table 2 shows the unadjusted, partially adjusted (sex, age, ethnicity and SES), and fully adjusted (all factors) odds ratios for excess body fatness for each of the demographic and lifestyle variables assessed. Unadjusted analyses indicated significant associations between fat status and age, ethnicity, SES, physical activity, breakfast, bought lunch, fast food, sugary drinks, and weekday sleep. Adjusting for differences in sex, age, ethnicity, and SES negated associations between fat status and bought lunch, fast food, and sugary drink consumption. However, the partially adjusted odds ratios for three of the four demographic variables remained significant. The odds of overfat increased with age; 11-12-year-old children were 15.4 times more likely to be overfat than those aged 5-6 years. In addition, children from the low SES group were 1.6 and 2.7 times more likely to have excess body fatness than children in the middle and high groups, respectively. While Asian children were 1.8 times more likely to be overfat than European children, differences were not significant for the other two ethnic groups. In the fully adjusted model, bought lunch and sugary drink consumption were restored as significant variables, whereas ethnicity became non-significant.

It is clear that adjusting for demographic differences had a noticeable effect on the odds of overweight between ethnic groups, but not between sex, age, or SES categories. Further analyses revealed no significant variation in sex ($p = 0.539$) or age ($p = 0.561$) distributions among the ethnic groups. In contrast, the SES distribution differed significantly by ethnicity ($p < 0.001$): 73.2% of Polynesian children were in the low SES group and only 8.4% were in the high SES group, compared with 15.8% and 51.0% for European children, and 25.5% and 42.3% for Asian children. To investigate the interaction between SES and ethnicity on fat status, the effects of SES on the odds of overfat were determined for the three major ethnic groups in this study (Fig 1). European and Asian children with a low SES were 2.6 and 3.3 times more likely to be overfat (respectively) than their high SES counterparts (European, $p = 0.006$; Asian, $p = 0.004$). Although Polynesian children in the Low and Middle SES groups were 2.9 and 3.8 times more likely to be overfat (respectively) than those in the High SES group, the difference was significant for the Middle SES group only ($p = 0.044$). There were no significant differences in the odds of overfat between Middle and High SES groups for European ($p = 0.395$) and Asian ($p = 0.430$) ethnicities.

Physical activity, breakfast consumption, and weekday sleep hours were the only lifestyle factors significantly associated with fat status in both the partially and fully adjusted models (Table 2). Children who accumulated less than 16 000 (boys) and 13 000 (girls) steps/day were over two times more likely to be overfat than children who reached these targets. Similarly, children who had breakfast for 3-4 (both models) or 1-2 (partially adjusted model only) days in the preceding week had increased odds of overfat compared with those who had breakfast for five or more days. Of all the lifestyle risk factors, children that sleep for less than 12 hours on weekdays had the highest odds of overfat, rising from 3.4-3.9 in the 11-11.9 hour group to 5.3-7.0 in the < 10 hour group. Active transport, sports participation, and weekend sleep patterns were not associated with fat status either before or after adjustment for the other demographic and lifestyle variables.

The associations between body fatness, overfat prevalence, and various combinations of the lifestyle risk

Table 1. Participant characteristics; results are mean ± SD.[†]

	European		Polynesian		Asian		All	
	M ($N = 283$)	F ($N = 292$)	M ($N = 201$)	F ($N = 206$)	M ($N = 99$)	F ($N = 97$)	M ($N = 603$)	F ($N = 626$)
Age (yr)	8.2 ± 1.8	8.4 ± 1.7	8.4 ± 1.8	8.4 ± 1.8	8.6 ± 1.7	8.6 ± 1.8	8.4 ± 1.8	8.4 ± 1.8
Height (cm)	131 ± 12.1	131 ± 11.7	133 ± 12.8	133 ± 13.0	131 ± 10.3	130 ± 12.5	132 ± 12.0	131 ± 12.2
Weight (kg)	29.9 ± 8.6[‡]	30.4 ± 9.7[‡]	35.3 ± 13.0	35.7 ± 13.9	30.0 ± 8.3[‡]	29.3 ± 9.2[‡]	31.7 ± 10.5	31.9 ± 11.4
BMI ($kg \cdot m^{-2}$)	17.1 ± 2.4[‡]	17.3 ± 2.8[‡]	19.4 ± 4.3	19.5 ± 4.4	17.3 ± 2.8[‡]	16.9 ± 2.8[‡]	17.9 ± 3.4	18.0 ± 3.6
Body fat (%)	17.4 ± 6.0[§]	21.2 ± 6.4[‖]	20.4 ± 7.5[§¶]	23.1 ± 7.3	20.1 ± 6.8[§¶]	23.2 ± 7.5	18.9 ± 6.9[§]	22.2 ± 6.9

[†]M, male; F, female; BMI, body mass index. [‡]Significantly different from Polynesian of same sex ($p < 0.01$). [§]Significantly different from female of same ethnic group ($p < 0.01$). [‖]Significantly different from Polynesian of same sex ($p < 0.05$). [¶]Significantly different from European of same sex ($p < 0.01$).

Table 2. Correlates of overfat (%BF ≥ 25% [boys] or 30% [girls]) in New Zealand children aged 5-11 years.

	Number of Participants (%)		Unadjusted Odds Ratio (95% CI)	Partially Adjusted Odds Ratio (95% CI)†	Fully Adjusted Odds Ratio (95% CI)‡
	Non-overfat	Overfat			
Sex					
Male	500 (48.4%)	103 (52.6%)	1.00	1.00	1.00
Female	533 (51.6%)	93 (47.4%)	0.85 (0.62-1.15)	0.82 (0.59-1.13)	0.85 (0.56-1.27)
Age					
5-6 yr	217 (21.0%)	8 (4.1%)	1.00	1.00	1.00
7-8 yr	375 (36.3%)	37 (18.9%)	2.68 (1.22-5.85)*	2.76 (1.25-6.06)*	3.01 (1.10-8.25)*
9-10 yr	317 (30.7%)	87 (44.4%)	7.44 (3.54-15.7)**	7.84 (3.70-16.6)**	6.32 (2.38-16.8)**
11-12 yr	124 (12.0%)	64 (32.7%)	14.0 (6.50-30.2)**	15.4 (7.07-33.5)**	13.9 (5.06-38.2)**
Ethnicity					
European	514 (49.8%)	61 (31.1%)	1.00	1.00	1.00
Polynesian	318 (30.8%)	89 (45.4%)	2.36 (1.65-3.36)**	1.48 (0.96-2.28)	1.38 (0.81-2.36)
Asian	157 (15.2%)	39 (19.9%)	2.09 (1.35-3.25)**	1.78 (1.12-2.83)*	1.52 (0.86-2.70)
Other	44 (4.3%)	7 (3.6%)	1.34 (0.58-3.11)	0.97 (0.40-2.36)	0.38 (0.11-1.29)
SES					
High	384 (37.2%)	41 (20.9%)	1.00	1.00	1.00
Middle	295 (28.6%)	50 (25.5%)	1.59 (1.02-2.47)*	1.59 (1.00-2.54)*	1.28 (0.74-2.23)
Low	354 (34.3%)	105 (53.6%)	2.78 (1.88-4.10)**	2.73 (1.70-4.38)**	2.09 (1.36-3.20)**
Physical Activity					
Active	431 (51.2%)	51 (30.7%)	1.00	1.00	1.00
Inactive	410 (48.8%)	115 (69.3%)	2.37 (1.66-3.39)**	2.26 (1.54-3.34)**	2.09 (1.36-3.20)**
Active Transport					
None	555 (54.4%)	92 (47.4%)	1.00	1.00	1.00
To or From School	133 (13.0%)	26 (13.4%)	1.18 (0.73-1.90)	1.12 (0.67-1.86)	1.40 (0.78-2.52)
To and From School	333 (32.6%)	76 (39.2%)	1.38 (0.99-1.92)	0.98 (0.69-1.41)	1.12 (0.72-1.75)
Sports Participation					
None	312 (31.0%)	54 (28.0%)	1.00	1.00	1.00
1-2 days/wk	414 (41.1%)	89 (46.1%)	1.24 (0.86-1.80)	1.29 (0.87-1.92)	1.41 (0.88-2.27)
3-4 days/wk	209 (20.7%)	30 (15.5%)	0.83 (0.51-1.34)	0.72 (0.43-1.20)	0.88 (0.48-1.62)
5+ days/wk	73 (7.2%)	20 (10.4%)	1.58 (0.89-2.81)	1.14 (0.62-2.12)	0.91 (0.39-2.11)
Breakfast					
None	13 (1.3%)	5 (2.6%)	2.38 (0.84-6.76)	1.46 (0.47-4.55)	1.38 (0.34-5.56)
1-2 days/wk	43 (4.2%)	20 (10.3%)	2.87 (1.64-5.02)**	1.88 (1.02-3.47)*	1.47 (0.73-2.98)
3-4 days/wk	53 (5.2%)	22 (11.3%)	2.56 (1.51-4.34)**	1.82 (1.02-3.26)*	2.24 (1.08-4.64)*
5+ days/wk	908 (89.3%)	147 (75.8%)	1.00	1.00	1.00
Bought Lunch					
None	603 (59.2%)	107 (55.2%)	1.00	1.00	1.00
1-2 days/wk	341 (33.5%)	65 (33.5%)	1.07 (0.77-1.50)	0.83 (0.58-1.20)	0.69 (0.44-1.08)
3-4 days/wk	33 (3.2%)	13 (6.7%)	2.22 (1.13-4.36)*	1.53 (0.73-3.20)	2.67 (1.03-6.90)*
5+ days/wk	42 (4.1%)	9 (4.6%)	1.21 (0.57-2.55)	0.71 (0.32-1.56)	0.41 (0.15-1.13)
Fast Food					
None	267 (26.3%)	39 (20.1%)	1.00	1.00	1.00
1-2 servings/wk	693 (68.3%)	141 (72.7%)	1.39 (0.95-2.04)	1.42 (0.94-2.15)	1.35 (0.83-2.21)
3-4 servings/wk	47 (4.6%)	9 (4.6%)	1.31 (0.60-2.88)	0.94 (0.41-2.18)	0.67 (0.22-2.07)
5+ servings/wk	7 (0.7%)	5 (2.6%)	4.89 (1.48-16.2)**	3.46 (0.94-12.7)	2.38 (0.48-11.8)
Sugary Drink					
None	159 (15.6%)	22 (11.4%)	1.00	1.00	1.00
1-2 servings/wk	465 (45.5%)	72 (37.3%)	1.12 (0.67-1.86)	1.09 (0.63-1.88)	1.63 (0.80-3.34)
3-4 servings/wk	189 (18.5%)	46 (23.8%)	1.76 (1.02-3.05)*	1.61 (0.90-2.90)	2.26 (1.06-4.84)*
5+ servings/wk	208 (20.4%)	53 (27.5%)	1.84 (1.08-3.16)*	1.56 (0.87-2.79)	2.37 (1.11-5.05)*
Weekday Sleep					
≥ 12 hr	92 (9.2%)	4 (2.2%)	1.00	1.00	1.00
11-11.9 hr	482 (48.1%)	70 (37.6%)	3.34 (1.19-9.38)*	3.36 (1.16-9.76)*	3.92 (1.07-14.4)*
10-10.9 hr	363 (36.2%)	89 (47.8%)	5.64 (2.02-15.8)**	3.96 (1.36-11.5)*	4.23 (1.13-15.8)*
< 10 hr	66 (6.6%)	23 (12.4%)	8.02 (2.65-24.2)**	5.25 (1.62-16.9)**	7.03 (1.63-30.4)**
Weekend Sleep					
≥ 12 hr	138 (14.0%)	20 (11.2%)	1.00	1.00	1.00
11-11.9 hr	362 (36.8%)	50 (28.1%)	0.95 (0.55-1.66)	1.01 (0.56-1.84)	0.70 (0.35-1.43)
10-10.9 hr	358 (36.4%)	78 (43.8%)	1.50 (0.89-2.55)	1.31 (0.74-2.33)	1.02 (0.52-1.99)
< 10 hr	125 (12.7%)	30 (16.9%)	1.66 (0.90-3.06)	1.05 (0.54-2.06)	0.85 (0.38-1.89)

†Adjusted for demographic factors (sex, age, ethnicity, and SES). ‡Adjusted for all other factors. *Significantly different from reference group ($p < 0.05$). **Significantly different from reference group ($p < 0.01$).

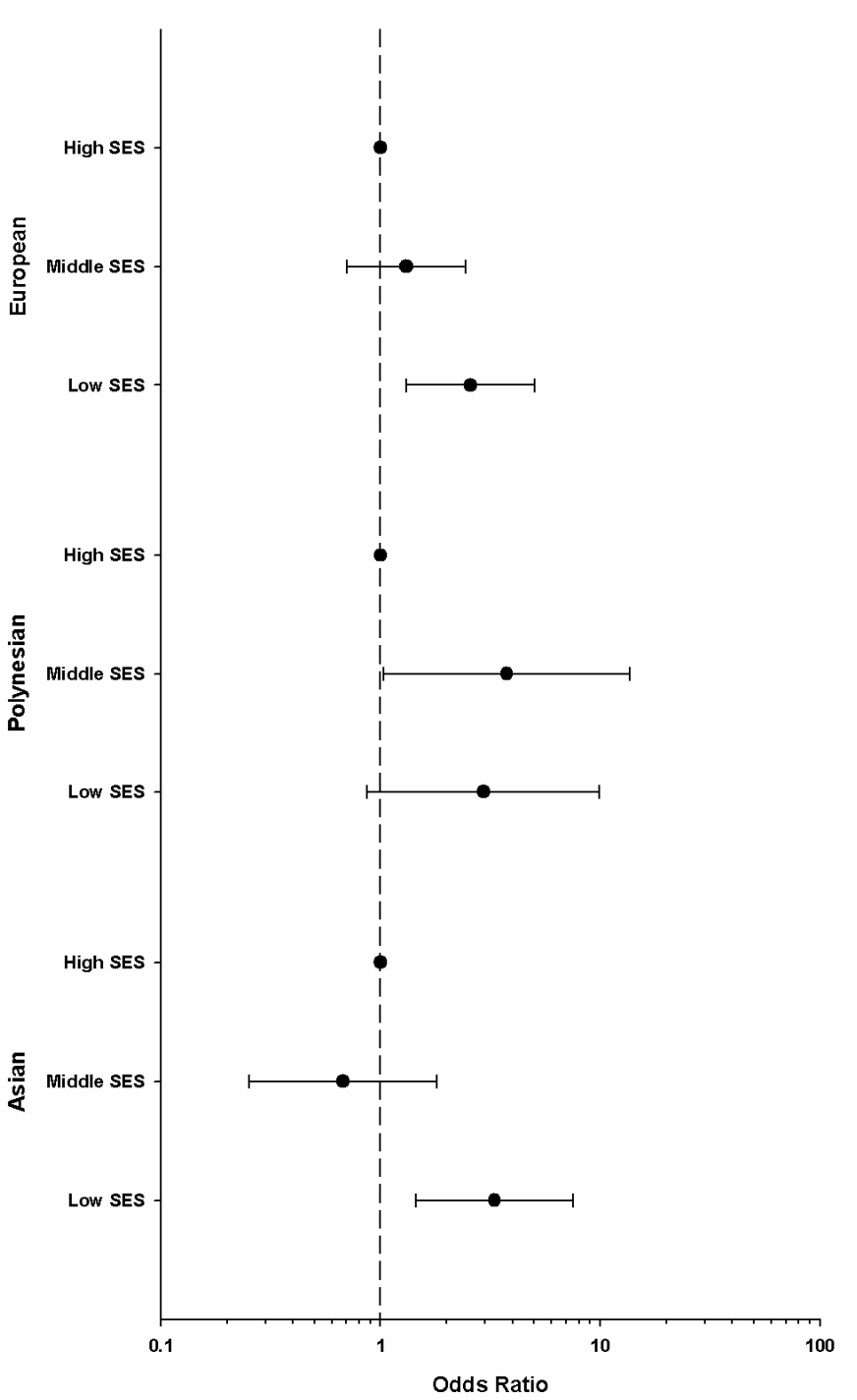

Figure 1. Relationship between the odds of overfat (%BF ≥ 25% [boys] or 30% [girls]) and socioeconomic status in European, Polynesian, and Asian children.

factors identified in all three models (inactivity, skipping breakfast, and low weekday sleep) were assessed separately for boys and girls while adjusting for differences in age, ethnicity, and SES (Table 3). Overall, larger differences in %BF were detected with clusters of two and three risk factors when compared with the single risk factor categories. Although children with lifestyle risk factors showed a greater prevalence of overfat than those without the risk factor(s), not all of the differences between cluster levels were statistically significant. For both boys and girls, chi-squared analysis revealed that the overfat prevalence in the inactive + low breakfast group was significantly higher than in the inactive group (boys, $p = 0.003$; girls, $p = 0.046$), but not in the low breakfast group (boys, $p = 0.286$; girls, $p = 0.266$). Similarly, combining the low breakfast and low weekday sleep groups resulted in a significant increase in overfat when compared with the low weekday sleep group (boys, $p = 0.001$; girls, $p = 0.017$), but not with the low breakfast group (boys, $p = 0.240$; girls, $p = 0.238$). No significant differences were detected between the inactive + low weekday sleep group and each individual risk factor. These findings suggest that skipping breakfast has the greatest effect on the prevalence of excess fatness in this sample. Indeed, the increase in overfat when combining all three risk factors was only significant (boys, $p = 0.004$; girls, $p = 0.042$) when compared to the two-factor cluster that did not already include the low breakfast group (inactive + low weekday sleep).

Table 3. Body fat and prevalence of overfat **(%BF ≥ 25% [boys] and 30% [girls])** in New Zealand children aged 5-11 years with three key correlates of overfat

Risk Factor(s)	Risk Factor(s) Absent			Risk Factor(s) Present		
	N	Body fat (%)	Overfat (%)	*N*	Body fat (%)	Overfat (%)
Boys						
Inactive (< 16 000 steps/day)	185	17.7 ± 0.9[†]	11.4	269	20.3 ± 0.8**	23.0**
Low Breakfast (< 5 days)	537	18.6 ± 0.5	14.7	59	21.7 ± 1.7**	37.3**
Low Weekday Sleep (< 11 hours)	297	18.3 ± 0.7	10.8	288	19.5 ± 0.8*	22.1**
Inactive + Low Breakfast	578	18.7 ± 0.5	15.7	24	24.6 ± 2.5**	50.0**
Low Breakfast + Low Weekday Sleep	571	18.7 ± 0.5	15.2	32	23.9 ± 2.2**	50.0**
Inactive + Low Weekday Sleep	466	18.3 ± 0.6	13.3	136	21.3 ± 1.1**	29.9**
Inactive + Low Breakfast + Low Weekday Sleep	588	18.7 ± 0.5	15.8	15	26.5 ± 3.2**	66.7**
Girls						
Inactive (< 13 000 steps/day)	279	21.4 ± 0.8	10.8	236	23.3 ± 0.8**	21.2**
Low Breakfast (< 5 days)	517	21.8 ± 0.6	13.2	97	24.6 ± 1.3**	25.8**
Low Weekday Sleep (< 11 hours)	351	22.3 ± 0.7	12.0	252	22.0 ± 0.9	19.0**
Inactive + Low Breakfast	580	21.9 ± 0.5	13.3	46	25.4 ± 1.9**	34.8**
Low Breakfast + Low Weekday Sleep	587	22.0 ± 0.5	13.5	39	24.4 ± 2.1*	35.9**
Inactive + Low Weekday Sleep	526	22.0 ± 0.6	12.5	100	23.2 ± 1.4	27.0**
Inactive + Low Breakfast + Low Weekday Sleep	606	22.1 ± 0.5	13.7	20	26.0 ± 2.9**	50.0**

[†]Mean ± 95% CI; adjusted for age, ethnicity, and socioeconomic status. *Significantly different from Risk Factor(s) Absent group ($p < 0.05$). **Significantly different from Risk Factor(s) Absent group ($p < 0.01$).

DISCUSSION

In this study, age, ethnicity, SES, physical activity, breakfast consumption, and weekday sleep duration were identified as important predictors of overfat in New Zealand children. While these findings are generally consistent with earlier research, specific risk factors reported by individual studies vary widely.[11] The discrepancies among studies may reflect fundamental differences between samples, such that observations made in one population may not apply to children from another. Inaccuracies in the techniques used to define childhood obesity may also contribute to the variation. The majority of previous studies have used BMI to estimate fat status. However, the relationship between existing BMI-based definitions of obesity and negative health outcomes in children is not known. Moreover, the use of BMI to classify obesity in multiethnic populations can be problematic.[24,25] The present study used a %BF-based definition of obesity to provide the first description of the correlates of excess fatness in New Zealand children.

Of the four demographic variables assessed in this study, age showed the strongest association with body fat status. This is not surprising given that children tend to accumulate body fat until they reach puberty as part of their normal physiological development.[33] When using a single %BF cut-off point for defining overfat, a natural increase in overfat participants would be expected with age. On the other hand, older children may have been exposed to obesogenic factors for a longer period than younger children. It is also possible that age-related differences in the morbidity associated with body fat could confound the use of a single %BF cut-off point. Although the use of %BF percentiles would account for developmental increases in fat mass by assuming a constant level of %BF for each year of age, there are currently no %BF percentiles related to health outcomes for children. Clearly, more research is needed to establish an internationally applicable definition of excess adiposity for predicting health risk in children of all ages.

The prevalence of excess adiposity was also affected by the deprivation level of the children: those in the low SES group showed greater odds of overfat than those in the high SES group. A variety of explanations have been proposed to explain the relationship between SES and obesity. For example, individuals from lower socioeconomic regions tend to have greater access to unhealthy foods and less knowledge of healthy dietary practices than those from higher socioeconomic regions.[34] Furthermore, physical activity participation in underprivileged communities can be hindered by perceptions of unsafe neighborhoods, limited access to gyms or sports clubs, and higher rates of sedentary activities.[35] While a number of previous studies have shown that socioeconomic factors play a role in the development of childhood obesity,[4-6] their impact may vary across different populations. Wang et al.[6] found that while American children with a low SES were more likely to be overweight than those with a high SES, the inverse relationship was observed in Brazilian children. Although ethnic variation in the relationship between SES and overfat was detected in the present sample, the general trend in all three ethnic groups was similar to that observed in other developed countries.[6]

Our initial analyses showed that Polynesian children were 2.4 times more likely to be overfat than European children. This is consistent with the relatively high prevalence of BMI-determined obesity previously reported in this ethnic grouping.[36,37] After adjusting for sex, age, and SES, differences in fat status between European and Polynesian children were negated. We suggest that the rates of obesity in Polynesian children may have been inflated by their overrepresentation in the 'at-risk' Low SES group. Thus, the traditional view that children of Polynesian descent are predisposed to obesity may largely reflect socioeconomic disadvantages rather than cultural or genetic influences. In contrast, Asian children maintained a significantly higher probability of overfat than European children after adjustment for differences in SES. Whether the increased risk of overfat in Asians is a consequence of fundamental differences in body composition or other sociocultural variables is undecided. However, the exclusion of ethnicity as a significant variable in the fully adjusted model suggests that behavioral factors may be involved.

Several lifestyle-related factors showed significant associations with fat status. In both the partially and fully

adjusted models, children who were not sufficiently active were more than twice as likely to be overfat than those who accumulated 16,000 (boys) or 13,000 (girls) steps per day.[30] These findings support the hypothesis that physical activity is inversely related to the risk of obesity in children,[12-15] and highlight the potential advantages of pedometer-based step count targets. Many existing strategies focus on increasing the amount of time children spend on moderate to vigorous activity each day. While beneficial, this approach is less practical and harder to assess than step-based initiatives. This study provides evidence that current step count recommendations are appropriate for reducing the risk of childhood obesity across a range of age, ethnic and SES groups.

Consumption of breakfast less than five days a week was the only dietary practice assessed which increased the odds of overfat in all models. Although several other cross-sectional studies have reported similar results in children and adolescents,[38-41] the mechanisms by which skipping breakfast contributes to the development of obesity remain unclear. Berkey et al.[42] provided evidence that the average number of calories consumed each day is greater in adolescents who regularly eat breakfast than in those who do not, suggesting that skipping breakfast does not promote fat gain through increased energy intake. It is possible that the tendency of overweight youth to skip breakfast is simply due to the high prevalence of dieting practices in this group.[38,43] In this case, reduced breakfast consumption would not be a determinant of obesity, but rather a result of the condition. To our knowledge, no studies have adjusted for differences in dieting behaviors when comparing breakfast patterns between obese and non-obese children.

The final lifestyle factor consistently associated with fat status in this sample was sleep duration on weeknights; children who sleep less than 10 hours each weeknight were over five times more likely to be overfat than those who sleep at least 12 hours. This finding is in line with earlier research suggesting that inadequate sleep is an important predictor of childhood obesity.[4,21-23] However, the mechanisms underlying the association between sleeping patterns and body composition have yet to be elucidated. Interestingly, we found that weekend sleep duration had little effect on the risk of overfat. One hypothesis is that insufficient sleep on weekdays is more strongly related to other obesogenic lifestyle practices than weekend sleep deprivation. Although other studies reported no interactions between sleep duration and other demographic and lifestyle variables,[4,21,22] none compared weekday and weekend sleep patterns. It is also possible that biological mechanisms are responsible for the effects of short sleeping times in children. For example, a lack of sleep is often associated with reduced leptin and elevated ghrelin in adults, two key opposing hormones in appetite regulation.[44,45] Similar hormonal changes in children may result in increased food intake and thus a greater risk of obesity. Nevertheless, this theory does not explain the lack of association between weekend sleep duration and overfat in our sample.

Frequent consumption of fast food, sugary drinks, and lunch bought at school have been reported as dietary risk factors for the development of obesity in children.[16-20] It should be noted, however, that the results presented in previous studies were not adjusted for differences in SES. Controlling for SES (in addition to age, sex, and ethnicity) eliminated the significant associations between fat status and fast food, sugary drinks, and bought lunch in our sample. These findings suggest that the three unhealthy dietary practices are closely related to SES, and may contribute to the elevated risk of overfat observed in underprivileged children.[4-6] Interestingly, the addition of all other lifestyle factors to the model restored the associations between overfat and both sugary drinks and bought lunch. This demonstrates that the relationships between the latter two variables and SES are affected by interactions with other lifestyle practices.

A secondary objective of this study was to investigate the effects of various combinations of lifestyle risk factors on children's body fatness. Knowledge of the risk factor clusters associated with the highest body fat levels would facilitate the development of interventions tailored to those most at-risk. Danielzik et al.[5] showed that combining three demographic risk factors for obesity (low SES, parental overweight, high birth weight) generated the highest risk of overweight in German children. Analyses of the key lifestyle factors in the present study (inactivity, low breakfast consumption, low weekday sleep) indicated similar patterns, with the three-factor combination resulting in a greater mean %BF and prevalence of overfat than the two- or single-factor groups. Although preventative initiatives that target multiple risk factors may offer cumulative benefits, the independent effects of modifiable factors in this study also support interventions that focus on only one risk factor. In our sample, a low frequency of breakfast consumption had the greatest influence on body fatness when compared to the other two lifestyle risk factors.

In summary, we identified several population subgroups at risk for excess fatness in New Zealand; namely older children, Asian children, and those from a low socioeconomic background. While the cross-sectional nature of this study precludes statements of cause-and-effect, our results also demonstrated a clear link between the risk of overfat and daily steps, breakfast consumption, and weekday sleep patterns, regardless of differences in sex, age, ethnicity, or SES. Furthermore, clustering of the major modifiable risk factors was associated with a cumulative increase in adiposity. Longitudinal research is required to determine the effects of these risk factors during development, including the potential implications for adult obesity. Understanding the determinants of excess fatness in young New Zealanders is essential to develop effective strategies for preventing obesity in this population.

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AUTHOR DISCLOSURES

James Scott Duncan, Grant Schofield, Elizabeth Karndu Duncan and Elaine Caroline Rush, no conflicts of interest.

REFERENCES

1. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. JAMA. 2004;291(23):2847-50.
2. Saxena S, Ambler G, Cole TJ, Majeed A. Ethnic group differences in overweight and obese children and young people in England: cross sectional survey. Arch Dis Child. 2004; 89(1):30-6.

3. Sorof JM, Lai D, Turner J, Poffenbarger T, Portman RJ. Overweight, ethnicity, and the prevalence of hypertension in school-aged children. Pediatrics. 2004;113(3 Pt 1):475-82.
4. Chaput JP, Brunet M, Tremblay A. Relationship between short sleeping hours and childhood overweight/obesity: results from the 'Quebec en Forme' Project. Int J Obes. 2006; 30(7):1080-5.
5. Danielzik S, Czerwinski-Mast M, Langnase K, Dilba B, Muller MJ. Parental overweight, socioeconomic status and high birth weight are the major determinants of overweight and obesity in 5-7 y-old children: baseline data of the Kiel Obesity Prevention Study (KOPS). Int J Obes Relat Metab Disord. 2004;28(11):1494-502.
6. Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. Am J Clin Nutr. 2002;75 (6):971-7.
7. Wang Y. Cross-national comparison of childhood obesity: the epidemic and the relationship between obesity and socioeconomic status. Int J Epidemiol. 2001;30(5):1129-36.
8. Gordon-Larsen P, Adair LS, Popkin BM. The relationship of ethnicity, socioeconomic factors, and overweight in US adolescents. Obes Res. 2003;11(1):121-9.
9. Whitaker RC, Orzol SM. Obesity among US urban preschool children: relationships to race, ethnicity, and socioeconomic status. Arch Pediatr Adolesc Med. 2006; 160(6): 578-84.
10. Goodman E, Adler NE, Daniels SR, Morrison JA, Slap GB, Dolan LM. Impact of objective and subjective social status on obesity in a biracial cohort of adolescents. Obes Res. 2003;11(8):1018-26.
11. Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. Int J Obes Relat Metab Disord. 1999;23 Suppl 8(S1-107.
12. Trost SG, Kerr LM, Ward DS, Pate RR. Physical activity and determinants of physical activity in obese and non-obese children. Int J Obes Relat Metab Disord. 2001;25(6): 822-9.
13. Janssen I, Katzmarzyk PT, Boyce WF, Vereecken C, Mulvihill C, Roberts C, Currie C, Pickett W. Comparison of overweight and obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. Obes Rev. 2005; 6(2):123-32.
14. Moore LL, Gao D, Bradlee ML, Cupples LA, Sundarajan-Ramamurti A, Proctor MH, Hood MY, Singer MR, Ellison RC. Does early physical activity predict body fat change throughout childhood? Prev Med. 2003;37(1):10-7.
15. Elgar FJ, Roberts C, Moore L, Tudor-Smith C. Sedentary behaviour, physical activity and weight problems in adolescents in Wales. Public Health. 2005;119(6):518-24.
16. Giammattei J, Blix G, Marshak HH, Wollitzer AO, Pettitt DJ. Television watching and soft drink consumption: associations with obesity in 11- to 13-year-old schoolchildren. Arch Pediatr Adolesc Med. 2003;157(9):882-6.
17. Gillis LJ, Bar-Or O. Food away from home, sugar-sweetened drink consumption and juvenile obesity. J Am Coll Nutr. 2003;22(6):539-45.
18. Thompson OM, Ballew C, Resnicow K, Must A, Bandini LG, Cyr H, Dietz WH. Food purchased away from home as a predictor of change in BMI z-score among girls. Int J Obes Relat Metab Disord. 2004;28(2):282-9.
19. Veugelers PJ, Fitzgerald AL. Prevalence of and risk factors for childhood overweight and obesity. CMAJ. 2005;173(6): 607-13.
20. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. Obes Res. 2004;12(5):778-88.
21. Sekine M, Yamagami T, Handa K, Saito T, Nanri S, Kawaminami K, Tokui N, Yoshida K, Kagamimori S. A dose-response relationship between short sleeping hours and childhood obesity: results of the Toyama Birth Cohort Study. Child Care Health Dev. 2002;28(2):163-70.
22. von Kries R, Toschke AM, Wurmser H, Sauerwald T, Koletzko B. Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep--a cross-sectional study. Int J Obes Relat Metab Disord. 2002;26(5):710-6.
23. Agras WS, Hammer LD, McNicholas F, Kraemer HC. Risk factors for childhood overweight: a prospective study from birth to 9.5 years. J Pediatr. 2004;145(1):20-5.
24. Deurenberg P, Deurenberg-Yap M, Foo LF, Schmidt G, Wang J. Differences in body composition between Singapore Chinese, Beijing Chinese and Dutch children. Eur J Clin Nutr. 2003;57(3):405-9.
25. Rush EC, Puniani K, Valencia ME, Davies PS, Plank LD. Estimation of body fatness from body mass index and bioelectrical impedance: comparison of New Zealand European, Maori and Pacific Island children. Eur J Clin Nutr. 2003;57 (11):1394-401.
26. Dwyer T, Blizzard CL. Defining obesity in children by biological endpoint rather than population distribution. Int J Obes Relat Metab Disord. 1996;20(5):472-80.
27. Washino K, Takada H, Nagashima M, Iwata H. Significance of the atherosclerogenic index and body fat in children as markers for future, potential coronary heart disease. Pediatr Int. 1999;41(3):260-5.
28. Williams DP, Going SB, Lohman TG, Harsha DW, Srinivasan SR, Webber LS, Berenson GS. Body fatness and risk for elevated blood pressure, total cholesterol, and serum lipoprotein ratios in children and adolescents. Am J Public Health. 1992;82(3):358-63.
29. Turnbull A, Barry D, Wickens K, Crane J. Changes in body mass index in 11-12-year-old children in Hawkes Bay, New Zealand (1989-2000). J Paediatr Child Health. 2004;40(1-2):33-7.
30. Duncan JS, Schofield G, Duncan EK. Step count recommendations for children based on body fat. Prev Med. 2007; 44(1):42-4.
31. Rowe DA, Mahar MT, Raedeke TD, Lore J. Measuring physical activity in children with pedometers: Reliability, reactivity, and replacement of missing data. Pediatric Exercise Science. 2004;16(4):343-54.
32. Sallis JF, Buono MJ, Roby JJ, Micale FG, Nelson JA. Seven-day recall and other physical activity self-reports in children and adolescents. Med Sci Sports Exerc. 1993;25 (1): 99-108.
33. McCarthy HD, Cole TJ, Fry T, Jebb SA, Prentice AM. Body fat reference curves for children. Int J Obes Relat Metab Disord. 2006;30(4):598-602.
34. James WP, Nelson M, Ralph A, Leather S. Socioeconomic determinants of health. The contribution of nutrition to inequalities in health. BMJ. 1997;314(7093):1545-9.
35. Wilson DK, Kirtland KA, Ainsworth BE, Addy CL. Socioeconomic status and perceptions of access and safety for physical activity. Ann Behav Med. 2004;28(1):20-8.
36. Ministry of Health. NZ Food NZ Children: Key Results of the 2002 National Children's Nutrition Survey. Ministry of Health: Wellington, 2003.
37. Tyrrell VJ, Richards GE, Hofman P, Gillies GF, Robinson E, Cutfield WS. Obesity in Auckland school children: a comparison of the body mass index and percentage body fat as the diagnostic criterion. Int J Obes Relat Metab Disord. 2001;25(2):164-9.

38. Boutelle K, Neumark-Sztainer D, Story M, Resnick M. Weight control behaviors among obese, overweight, and nonoverweight adolescents. J Pediatr Psychol. 2002; 27(6): 531-40.
39. O'Dea JA, Caputi P. Association between socioeconomic status, weight, age and gender, and the body image and weight control practices of 6- to 19-year-old children and adolescents. Health Educ Res. 2001;16(5):521-32.
40. Pastore DR, Fisher M, Friedman SB. Abnormalities in weight status, eating attitudes, and eating behaviors among urban high school students: correlations with self-esteem and anxiety. J Adolesc Health. 1996;18(5):312-9.
41. Vanelli M, Iovane B, Bernardini A, Chiari G, Errico MK, Gelmetti C, Corchia M, Ruggerini A, Volta E, Rossetti S. Breakfast habits of 1,202 northern Italian children admitted to a summer sport school. Breakfast skipping is associated with overweight and obesity. Acta Biomed. 2005;76(2):79-85.
42. Berkey CS, Rockett HR, Gillman MW, Field AE, Colditz GA. Longitudinal study of skipping breakfast and weight change in adolescents. Int J Obes Relat Metab Disord. 2003;27(10):1258-66.
43. Brugman E, Meulmeester JF, Spee-van der Wekke A, Beuker RJ, Zaadstra BM, Radder JJ, Verloove-Vanhorick PS. Dieting, weight and health in adolescents in The Netherlands. Int J Obes Relat Metab Disord. 1997;21(1):54-60.
44. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med. 2004;141(11):846-50.
45. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Medicine. 2004;1(3): e62.

Original Article

Risk factors for excess body fatness in New Zealand children

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紐西蘭兒童體脂肪過高的危險因子

目的：以多種族樣本，找出紐西蘭兒童體脂肪過高的人口學和生活型態危險因子。**設計**：橫斷性研究。**參與者**：共有 1229 位(603 位男孩，626 位女孩)5-11 歲居住在紐西蘭的歐洲、玻里尼西亞、亞洲及‘其他’種族的兒童。**測量方法**：以手-到-腳生物阻抗法測量體脂肪百分比(%BF)；過胖的定義為%BF 大於 25%(男童)和 30%(女童)。雙親代為回答問卷中人口學、生活型態因子，用多天記憶記步器來估計兒童 5 天的體能活動程度。**結果**：在控制性別、年齡和社經地位(SES)的差異後，亞洲兒童比較有可能比歐洲兒童有過高的體脂肪。校正過胖的勝算也隨著年齡的增加而增加及隨著 SES 增加而下降。三個生活型態的危險因子被找出來與肥胖狀態有關：低體能活動、不吃早餐及平日睡眠不足。這些危險因子的聚集導致過胖的盛行率持續上升。在校正被選出的人口學變項後，主動的交通方式、運動參與、在學校買的午餐、食用速食、含糖飲料飲用和週末睡眠時間長短與肥胖狀態沒有關聯。**結論**：本研究的發現提高我們對紐西蘭兒童過胖危險因子的瞭解，並指出未來介入計畫的關鍵人口學和生活型態變項的優先順序。

關鍵字：肥胖、種族淵源、飲食、體能活動、睡眠。