Childhood predictors of adult adiposity: findings from a longitudinal study

Geraldine FH McLeod, David M Fergusson, L John Horwood, Joseph M Boden, Frances A Carter

ABSTRACT

AIMS: The increasing prevalence of overweight and obesity has become a key challenge for New Zealand. The purpose of the present study was to examine childhood risk factors for adult adiposity in a longitudinal birth cohort.

METHODS: Data were gathered from the Christchurch Health and Development Study (CHDS), a birth cohort of 1,265 children born in Christchurch in 1977. Associations were examined between socio-demographic background, perinatal factors, infant and child characteristics, family functioning/child maltreatment and adiposity at ages 30 and 35 years. Adiposity was assessed using body mass index scores.

RESULTS: At ages 30 and 35, approximately one-third of cohort members were overweight and one-fifth were obese. Generalised estimating equation models showed that statistically significant ($p<.05$) predictors of later adiposity and overweight/obesity were: male gender, being born into a single-parent family, having parents with larger body size, higher early infant growth, limited or no breastfeeding, lower levels of cognitive ability and exposure to severe sexual abuse.

CONCLUSIONS: Overweight and obesity was associated with social and family background, biological endowment, cognitive ability and childhood adversity factors. These findings may assist in the development of structured adiposity intervention programmes in conjunction with established community organisations specialising in child and family health.

Concerns have been raised about the prevalence of obesity in New Zealand.[1,2] These concerns generally relate to the health, social and economic consequences that increasing rates of obesity cause New Zealanders and the health system.[1–4] Current estimates suggest that 31.6% of New Zealand adults meet standard criteria for obesity (BMI ≥30) with these rates being higher among Māori and Pasifika (47.1% and 66.9% respectively).[5,6]

The reduction of the population prevalence of obesity is a priority issue for the New Zealand Government[7] and *A Better Start Science Challenge* was developed to encourage research into tools to predict, prevent and treat childhood obesity, and to reduce obesity over the life course.[8] A key focus of this research is an ascertainment of the childhood predictors of later obesity. An extensive international literature has identified a number of childhood determinants of adult weight and obesity, including maternal factors, and child growth, diet and activity factors.[9–11] However, less is known about the role of these factors in contributing to adult obesity in the New Zealand context. Indeed, is has been argued that knowledge of local (within-population) factors is critical to developing approaches to solving the problem of obesity.[12] Further, prospective longitudinal studies are critical for understanding the causal role of early factors in determining adult obesity. Two longitudinal studies in New Zealand have data that allow examination of predictors of obesity across the life-course to adulthood. Research from the Christchurch Health and Development Study (CHDS) has shown that the association between breast feeding and BMI was mediated by early growth,[13] while research from the Dunedin Multidisciplinary

ISSN 1175-8716

Health and Development Study has shown that the development of adult obesity was associated with rapid early growth[14] and shorter duration of sleep.[15]

In order to examine further early predictors of adult obesity using New Zealand data, the present study examined a series of childhood risk factors for adiposity in adulthood using data from the Christchurch Health and Development Study (CHDS). The present study aimed to identify factors in the development and maintenance of high adiposity among individuals growing up in New Zealand.

Methods

Participants

Participants were members of the Christchurch Health and Development Study (CHDS) birth cohort. The CHDS is a longitudinal study of 1,265 children (630 females) born in the Christchurch (New Zealand) urban region over a four-month period during 1977. This cohort has been studied regularly from birth to age 35 using a combination of: interviews with parents and participants, standardised testing, teacher report and official record data.[16,17] The Appendix shows the sociodemographic characteristics of the CHDS cohort at birth, age 30 and age 35. All phases of the study were subject to ethical approval by the Regional Health and Disabilities Ethics Committee.

Measures

Body mass index

At ages 30 and 35, assessments of participants' height and weight were obtained. In 71% of cases, these estimates were recorded by trained staff using standardised measurements taken in respondents' homes, using Seca 214 portable stadiometers to measure height and Tanita HD-351 scales to measure body-weight. However, in a minority (29%) of cases, direct assessment of height/weight was not possible; for these participants, information was based on self-report data obtained via telephone or Skype interview. Overall, height and weight estimates were available for 99.0% (977/987) of those studied at age 30 and 95.9% (923/962) of those studied at age 35.

Using this information, body mass index (BMI) scores were calculated for respondents at ages 30 and 35. BMI was calculated as weight (kg)/height (m^2).

Validity of assessing BMI on the basis of self-report data was previously assessed on a subsample of the cohort at age 30. This showed a correlation of r=0.96 between assessments of BMI based on self-report and standardised measurement.[13]

Risk factors for obesity

A series of risk factors for overweight/obesity were gathered from the CHDS database based on previous research and theory.[10,13,18]

Socio-demographic background

Socio-demographic predictors included: maternal age at the child's birth; mother's educational qualifications; family type (child born into one-parent or two-parent family); family socioeconomic status;[19] averaged living standards (0–10 years) assessed on the basis of annual interviewer ratings; averaged family income (0–10 years).

Perinatal factors

At the birth interview, mothers were questioned about their pre-pregnancy height and weight, and that of their child's biological father (if known). From this information, measures of maternal and paternal BMI were calculated. Estimates of BMI were available for over 99% of biological mothers and fathers. Information was also gathered on: maternal smoking during pregnancy; infant feeding and growth over the child's first year (breastfeeding assessed at four months and one year; infant solid feeding assessed at four months; and infant weight gain (0–9 months) assessed from Plunket Book records).

Infant/child characteristics

Measures included gender; Māori/Pacific ethnicity; cognitive ability (7–8 years) using the Revised Wechsler Intelligence Scale for Children (WISC-R);[20] child attentional and conduct problems (7–9 years) using parent and teacher questionnaires;[21] and sleep problems (7–9 years).

Family functioning and childhood maltreatment

Measures of family functioning and child maltreatment were gathered on parental separations/divorce (0–16 years); and child maltreatment (childhood physical

punishment) (0–16 years); childhood sexual abuse (0–16 years), which was assessed by retrospective reports at 18 and 21 years.[22]

Additional predictors considered for inclusion but found not to be related to BMI in preliminary analysis included: birthweight; diversity of early solid food diet (number of different foods 0–4 months); birth order in family; number of hours sleep per night.

Statistical analysis

Bivariate associations between gender and adiposity

Repeated measures of BMI classified as <25 (normal), 25–29.9 (overweight) and 30+ (obese) at ages 30 and 35 years were examined by gender (Table 1). Gender and age-related differences in the distribution of BMI were tested for statistical significance using the chi-square test of independence.

Associations between childhood factors and BMI

Analysis of associations between adiposity and the risk factors summarised above was conducted in two stages. In the first stage, to facilitate data display each of the risk factors was dichotomised and related to an averaged measure of adiposity classified into BMI <25; 25–29.9; 30+ (Table 2). All associations were tested for statistical significance using the Mantel–Haenszel chi-square test of linearity. The strength of each association was summarised by the Pearson correlation (r) between BMI and the risk factor, with both measures scored in their natural (non-categorised) metrics.

In the second stage, a generalised estimating equation (GEE) modelling approach[23] was used to fit population-averaged regression models in which the repeated measures of BMI at ages 30, 35 were modelled as a function of age, gender and the significant risk factors identified above. Two models were fitted to predict (1) mean BMI and (2) percent overweight or obese (BMI≥25). The fitted models took the general form:

$$F(Yit) = B0 + B1\ AGEit + B2\ GENDERi + \sum Bj\ Xij$$

where Yit was the expected value of BMI, or the probability for being overweight/obese, for the ith participant at time t (t=30, 35 years); F was the appropriate link function (identity for continuous outcome (BMI); logistic for dichotomous outcome (percent overweight/obese)); AGEit was the age of individual i at time-period t; GENDERi represented the gender of the cohort member; and Xij were the set of childhood predictors. The repeated observations for each individual were permitted to be correlated over time. In fitting these models all predictors were scored in their natural metrics, and models were refined using methods of forward and backward elimination to identify a parsimonious and stable set of significant predictors of one or both outcomes.

To illustrate the net impact of each of the identified risk factors on the two outcomes (mean BMI, percent overweight/obese) the final regression models were re-run with all continuous predictors quantilised into either quartiles (duration of breastfeeding) or quintiles (early growth; parental BMI; cognitive ability). From the resulting models estimates of marginal adjusted mean BMI and percent overweight/obese were calculated for each level of each predictor pooled over the repeated measures at ages 30, 35 (Table 3).[24] Finally, models were then extended to test for multiplicative age by risk factor and gender by risk factor interactions.

Sample size and sample bias

The current analysis is based on a sample of 980 participants with data on BMI, of whom n=977 were assessed at age 30 and n=923 at age 35. These samples represented between 79.4% and 75.4% of the cohort members surviving to age 30 (n=1,231) and 35 years (n=1,223). The level of sample attrition raises issues regarding study validity. To examine this, all analyses were repeated using a two-stage data-weighting process to adjust for potential sample selection bias.[25] These analyses produced essentially identical conclusions to the reported analyses, suggesting findings were unlikely to have been influenced by selection bias.

Table 1: Distribution of body mass index (BMI) by gender at ages 30 and 35 years.

	BMI		
	<25 (Normal) % (n)	25–29.9 (Overweight) % (n)	30+ (Obese) % (n)
Age 30			
Female	58.7 (296)	23.2 (117)	18.1 (91)
Male	40.2 (190)	41.0 (194)	18.8 (89)
Total	49.7 (486)	31.8 (311)	18.4 (180)
Age 35			
Female	49.8 (238)	26.4 (126)	23.9 (117)
Male	31.7 (141)	45.8 (204)	22.5 (100)
Total	41.1 (379)	35.8 (330)	23.2 (214)

Chi-square tests for gender differences in distribution of BMI: age 30 ($p<0.001$), age 35 ($p<0.001$).
Chi-square tests for changing distribution of BMI with age: females ($p<0.05$), males ($p<0.05$), total sample ($p<0.001$).

Results

Table 1 shows the distribution of BMI categorised as <25 (normal), 25–29.9 (overweight) and 30+ (obese) by age and gender. Inspection of the table shows the presence of statistically significant ($p<0.001$) gender differences in the distribution of BMI, reflecting that at both ages proportionately more women than men were classified as normal-weight (58.7% vs 40.2% at age 30; 49.8% vs 31.7% at age 35) whereas proportionately fewer women than men were classified as overweight (23.2% vs 41.0% at age 30, 26.4% vs 45.8% at age 35). However, the rates of obesity were similar in the two groups (18.1% vs 18.8% at age 30, 23.9% vs 22.5% at age 35). The table also shows a general and statistically significant ($p<0.001$) trend toward increasing adiposity from age 30 to 35: in the total sample the percentage classified as overweight or obese increased from 50.3% at age 30, to 58.9% at age 35. This trend toward increased adiposity was similar for both sexes.

Table 2 shows associations between BMI (categorised as <25, 25–29.9 and 30+) and a range of measures of childhood socio-demographic background; perinatal factors; infant/child characteristics and family functioning/childhood maltreatment. To simplify the presentation and for the purposes of data display, the measure of BMI is based upon the average of the two BMI assessments at ages 30, 35 and potential predictor variables have been dichotomised to show the profile of predictor characteristics for each level of BMI. Each association has been tested for statistical significance using the Mantel-Haenszel chi-square test of linearity, and the strength of association is summarised by the Pearson correlation (r) between BMI and the predictor (see Methods).

The table shows the presence of small to moderate linear associations (r=0.07–0.26) between adult BMI and a wide range of childhood and family factors:

Socio-demographic background: Higher adult BMI was associated with lower: maternal education ($p=0.016$); SES ($p<0.001$); and family living standards ($p<0.001$). Higher BMI was also associated with younger maternal age ($p=0.033$) and entry into a one-parent family at birth ($p<0.001$).

Perinatal factors: Higher adult BMI was associated with higher maternal and paternal BMI ($p<0.001$); greater infant weight gain (0–9 months) ($p<0.001$); maternal smoking during pregnancy ($p=0.012$); the child not being breastfed ($p<0.001$); and the introduction of solid foods at age two months or younger ($p=0.027$).

Infant and child characteristics: Those with higher BMI were more likely to be Māori/Pacific ethnicity ($p<0.001$); to have had attentional ($p=0.002$) or conduct

Table 2: Associations between averaged BMI (30, 35 years) and a series of childhood factors.

	BMI				
Measure	**<25 Normal (n=361)**	**25–29.9 Overweight (n=368)**	**30+ Obese (n=251)**	**p**	**r[a]**
Socio-demographic background					
% Mother aged ≤25 years at birth of child	43.9	49.3	52.5	0.033	0.10
% Mother lacked formal educational qualifications at birth	44.6	51.6	54.0	0.016	0.11
% Child entered one-parent family at birth	3.0	6.5	11.9	<0.001	0.14
% Family of low SES (unskilled/semiskilled occupational status)	21.3	24.3	39.2	0.030	0.11
% In lowest quartile of averaged family living standards (0–10 years)	17.8	13.6	23.4	<0.001	0.17
% In lowest quartile of averaged family income (0–10 years)	21.6	22.2	28.4	0.087	0.08
Perinatal factors					
% Maternal obesity	1.6	1.2	6.5	0.002	0.26
% Paternal obesity	2.1	4.5	9.5	<0.001	0.26
% Mother smoked during pregnancy	28.4	33.1	38.1	0.012	0.08
% Child not breastfed	21.3	43.2	42.7	<0.001	0.16
% Solids introduced at age two months or younger	67.2	75.2	74.4	0.027	0.10
% Highest quartile infant weight gain (0–9 months)	16.6	26.4	26.5	<0.001	0.17
Infant and child characteristics					
% Māori/Pacific ethnicity	8.7	15.5	20.8	<0.001	0.14
% Lowest quartile cognitive ability (7–8 years)	22.2	24.9	29.7	0.043	0.13
% Highest quartile attentional problems (7–9 years)	21.4	24.3	33.7	0.002	0.10
% Highest quartile conduct problems (7–9 years)	19.7	26.7	30.1	0.003	0.10
% One or more sleep problems (7–9 years)	33.5	34.4	45.6	0.009	0.07
Family functioning and childhood maltreatment (0–16 years)					
% Experienced change of parents (<16 years)	34.8	34.6	45.1	0.028	0.09
% Regular/severe physical punishment/maltreatment (<16 years)	13.3	20.5	24.0	<0.001	0.12
% Severe sexual abuse (<16 years)[b]	4.1	7.3	10.5	0.002	0.10

[a]Pearson correlation between averaged BMI and each risk factor with all measures scored in their natural (non-categorised) metrics.
[b]Severe sexual abuse defined as abuse involving attempted or completed sexual penetration.

(p=0.003) problems in childhood; to have sleep problems (p=0.009); and to be of lower cognitive ability (p=0.043).

Family functioning and childhood maltreatment: Higher BMI was associated with the experience of parental change(s) (p=0.028); and exposure to childhood physical (p<0.001) or sexual (p=0.002) maltreatment/abuse.

The findings in Table 2 were used to develop multivariate models of associations between risk factors and adiposity pooled over the repeated assessments at ages 30, 35 years (see Methods). These models identified a series of predictors, which included: gender; family type; parental BMI; infant growth; duration of breastfeeding; child cognitive ability; and childhood sexual

Table 3: Fitted regression models predicting mean BMI and percent overweight/obese showing: (a) estimated regression coefficients and tests of significance for each risk factor; and (b) adjusted marginal mean (SE) BMI and percent overweight/obese for levels of each risk factor pooled over repeated observations at 30, 35 years.[a]

Measure	Mean (SE) BMI	% Overweight/obese
Gender		
Male	27.1 (0.23)	63.3
Female	26.1 (0.22)	44.9
B(SE), p	-1.011 (0.328), 0.002	-0.838 (0.135), <0.001
Family status		
One parent	28.9 (0.65)	72.8
Two parent	26.5 (0.16)	52.8
B(SE), p	-2.461 (0.673), 0.002	-0.988 (0.314), 0.002
Mother's BMI (Quintiles)		
5 Highest	28.0 (0.28)	63.1
4	27.3 (0.19)	58.6
3	26.6 (0.15)	54.0
2	25.9 (0.19)	49.3
1 Lowest	25.2 (0.27)	44.7
B(SE), p	0.698 (0.113), <0.001	0.210 (0.047), <0.001
Father's BMI (Quintiles)		
5 Highest	28.3 (0.28)	64.4
4	27.5 (0.19)	59.4
3	26.6 (0.15)	54.2
2	25.9 (0.19)	49.0
1 Lowest	25.1 (0.27)	43.7
B(SE), p	0.803 (0.113), <0.001	0.236 (0.047), <0.001
Duration breastfeeding (months)		
None	26.8 (0.16)	55.1
1-3	26.4 (0.17)	52.2
4-6	26.0 (0.27)	49.1
7+	25.7 (0.39)	46.1
B(SE), p	-0.377 (0.140), 0.007	-0.139 (0.057), 0.015
Early infant weight gain 0–9 months (Quintiles)		
5 Highest	27.1 (0.29)	57.6
4	26.9 (0.20)	55.8
3	26.6 (0.15)	54.0
2	26.4 (0.19)	52.2
1 Lowest	26.2 (0.28)	50.3
B(SE), p	0.238 (0.119), 0.045	0.084 (0.048), 0.083

Table 3: Fitted regression models predicting mean BMI and percent overweight/obese showing: (a) estimated regression coefficients and tests of significance for each risk factor; and (b) adjusted marginal mean (SE) BMI and percent overweight/obese for levels of each risk factor pooled over repeated observations at 30, 35 years (continued).[a]

Cognitive ability (Quintiles)		
1 Lowest	27.1 (0.28)	58.0
2	26.9 (0.20)	56.1
3	26.7 (0.15)	54.1
4	26.4 (0.18)	52.2
5 Highest	26.2 (0.26)	50.2
B(SE), p	-0.228 (0.110), 0.039	-0.090 (0.045), 0.047
Severe sexual abuse (<16 years)		
Yes	28.9 (0.61)	69.7
No	26.5 (0.16)	52.7
B(SE), p	2.447 (0.635), <0.001	0.828 (0.271), 0.002

[a]All effects adjusted for age and the other predictors listed in the table.

abuse. Associations between these factors and measures of BMI are shown in Table 3. This table shows each predictor classified into a series of ordered groups ranging from highest risk to lowest risk of adiposity and related to: (1) mean BMI; and (2) the percentage classified as overweight/obese (BMI 25+), adjusted for age and other factors in the model. The table also reports the fitted regression coefficient and test of significance for each factor.

Table 3 shows that higher mean BMI and risk of overweight/obesity was found among those who were male; were born into single-parent families; had parents with higher BMI; showed greater weight-gain in infancy; were not breastfed; were of lower cognitive ability; or were exposed to severe childhood sexual abuse. Overall, the net impact of these risk factors on mean BMI ranged between 1–3 BMI units from lowest to highest categories of risk, with a corresponding absolute increase in the risk of overweight/obesity of between 8–20%.

The statistical models described in Table 3 were extended to include multiplicative tests of age, and gender interaction by each predictor variable (see Methods). Two interactions remained statistically significant ($p<0.05$) in the full regression models (age x breastfeeding; age x severe sexual abuse). In both cases, the associations with adiposity appeared to be somewhat stronger at age 35 than age 30.

Supplementary analyses

Previous research has suggested that the use of BMI to examine overweight and obesity may provide biased estimates of adiposity since BMI may not discriminate muscle mass and fat mass.[26] To check the validity of the BMI measure, a measure of body-size incorporating both waist circumference and BMI was examined.[27] The results of this analysis produced findings that were consistent with the analysis above.

The analyses included a small number of underweight people (BMI ≤18.5) (n=13 at age 30; n=7 at age 35). Reanalysis excluding those underweight produced results that were unchanged.

Finally, the data were reanalysed excluding the minority of participants for whom BMI was assessed on the basis of self-reported height and weight. Findings were essentially unchanged, signalling that the use of self-report data was not a serious threat to validity.

Discussion

This analysis used data from a New Zealand birth cohort, the Christchurch Health and Development Study (CHDS) to examine both the prevalence of obesity and

to identify childhood factors that contribute to the risk of later high adiposity. The study showed that when CHDS respondents were aged 30 and 35 years, approximately one-third were overweight and one-fifth were obese.

A series of socio-demographic background, perinatal factors, infant/child characteristics and family functioning/childhood maltreatment measures were selected from the CHDS database to ascertain associations between those measures and BMI, and percent overweight/obesity. As expected, gender was significantly ($p<0.001$) associated with BMI, with males having consistently higher BMI scores than females. Multivariate analyses also showed that the most important factors for later overweight/obesity were being born into a single-parent family, having parents with larger body size, and experiencing severe sexual abuse. Other predictors of later overweight/obesity were having higher infant weight gain, limited or no breastfeeding and lower cognitive ability.

The New Zealand Health Strategy aims to reduce population levels of obesity and the associated health and financial costs.[7] Several approaches were identified in the strategy to support this goal, including sport programmes, public education and better food labelling. However, the present study suggests that obesity is caused by a complex mix of childhood family background, biological endowment and individual factors beginning in pregnancy and early childhood.[9,28,29] Most of the factors identified in the present study (such as gender, parental BMI and exposure to childhood sexual abuse) are not amenable to change or modification in the context of obesity prevention. The exceptions to this were the findings for longer duration of breastfeeding and weight gain in infancy, which have been shown in a number of studies to be associated with lower risk of adult obesity.[9,13] This suggests that the promotion of breastfeeding, the encouragement of longer periods of breastfeeding infants and monitoring the use of formula and the introduction of solids could play key roles in any strategy designed to reduce obesity.

While many of the early life factors identified in the present study as playing a causal role in adult obesity are not amenable to change, it is still important to identify and understand these factors in the context of developing a risk index model for adult obesity. An understanding of the childhood factors associated with later obesity may permit the identification of individuals who will be at greater risk of later obesity, in order to inform and develop more targeted and patient-specific interventions. For example, one approach to this issue may be through the use of controlled intervention programmes, which could be targeted at high-risk families and individuals.[29,30] A systematic review of interventions in the first 1,000 days of infancy by Blake-Lamb et al[30] identified 26 completed interventions. Seven of these interventions focusing on individual/family level behaviour changes were shown to have been effective in reducing obesity. In New Zealand, there are already a number of infant and young childhood healthcare providers such as Plunket,[31] Early Start[32] and Family Start,[33] which deliver a range of home visiting, child health checks, maternal education and resources to families with young children. It may be possible to integrate evidence-based early intervention for adiposity and obesity through these or similar agencies.

The data for this study were gathered from one of only two longitudinal birth cohorts which assessed childhood risk factors and later adiposity in New Zealand.[34] Strengths of this study include high response rates, repeated-measures of adiposity and the availability of a wide-range of prospectively gathered predictors. These research design features provide study findings that are unlikely to be influenced by non-observed sources of bias. However, limitations include that the findings relate to a specific cohort, studied at specific ages, in a specific socio-cultural context. The extent to which the findings generalise to other settings remains to be examined. Nevertheless, this study aids the understanding of the causal role of a series of childhood factors on adult body-size and adiposity among a cohort of New Zealand-born individuals.

Appendix

The following tables, Appendix Table 1 and Appendix Table 2, report the sociodemographic characteristics of the Christchurch Health and Development Study (CDHS) cohort at birth, age 30 and age 35 years.

Appendix Table 1: Sociodemographic characteristics for the CHDS cohort at birth.

Measures	**Birth (n=980)**
% (n) Male	48.1 (473)
Ethnicity	
% (n) Māori/Pacific	13.6 (133)
% (n) New Zealand European/Other	86.4 (844)
Maternal educational attainment	
% (n) No formal qualifications	48.8 (477)
% (n) Secondary (high school) qualifications	31.0 (303)
% (n) Tertiary qualifications	20.2 (197)
Socioeconomic status[a]	
% (n) Semiskilled, unskilled, unemployed	20.6 (201)
% (n) Clerical, technical, skilled	55.4 (541)
% (n) Professional, managerial	24.1 (235)
Family status	
% (n) One parent family	6.0 (59)
% (n) Two parent family	94.0 (918)

[a]SES based on Elly and Irving[19] classification of the father's occupation.

Appendix Table 2: Sociodemographic characteristics for the CHDS cohort at 30 and 35 years.

Measures	**Age 30 (n=977)**	**Age 35 (n=923)**
% (n) Male	48.4 (478)	48.2 (455)
Ethnicity		
% (n) Māori/Pacific	13.6 (133)	13.4 (124)
% (n) New Zealand European/Other	86.4 (844)	86.6 (799)
Educational attainment		
% (n) No formal qualifications	10.6 (99)	9.6 (88)
% (n) Secondary (high school) qualifications	23.2 (226)	22.6 (208)
% (n) Tertiary qualifications below degree level	36.3 (354)	36.5 (336)
% (n) Bachelor's degree or higher	30.3 (295)	31.3 (288)
Personal income		
Mean (SD) Gross annual personal income (,000)[a]	48.5 (29.8)	58.5 (42.3)

[a]Personal income from all sources in New Zealand Dollars.

ISSN 1175-8716

Competing interests:
All authors excepting Dr Carter report grants from Health Research Council of New Zealand (HRC 11/792), grants from the National Child Health Research Foundation, grants from Canterbury Medical Research Foundation, grants from New Zealand Lottery Grants Board, during the conduct of the study.

Acknowledgements:
This research was funded by grants from the Health Research Council of New Zealand (HRC 11/792), the National Child Health Research Foundation, the Canterbury Medical Research Foundation and the New Zealand Lottery Grants Board.

Author information:
Geraldine FH McLeod, Research Fellow, Department of Psychological Medicine, University of Otago, Christchurch; David M Fergusson, Emeritus Professor, Christchurch Health and Development Study, Department of Psychological Medicine, University of Otago, Christchurch; L John Horwood, Professor and Director of Christchurch Health and Development Study, Department of Psychological Medicine, University of Otago, Christchurch; Joseph M Boden, Associate Professor, Department of Psychological Medicine, University of Otago, Christchurch; Frances A Carter, Clinical Psychologist, Canterbury District Health Board, Christchurch.

Corresponding author:
Professor John Horwood, Christchurch Health and Development Study, Department of Psychological Medicine, University of Otago, Christchurch 8140.
john.horwood@otago.ac.nz

URL:
http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2018/vol-131-no-1472-23-march-2018/7524

REFERENCES:

1. Ministry of Health. Understanding Excess Body Weight: New Zealand Health Survey. Wellington: Ministry of Health, 2015.
2. Swinburn B, Wood A. Progress on obesity prevention over 20 years in Australia and New Zealand. Obes Rev. 2013; 14:60–8.
3. McLeod GFH, Fergusson DM, Horwood LJ, Carter FA. Adiposity and psychosocial outcomes at ages 30 and 35. Soc Psychiatry Psychiatr Epidemiol. 2015; 51:309–18.
4. Hunger JM, Major B, Blodorn A, Miller CT. Weighed down by stigma: How weight-based social identity threat contributes to weight gain and poor health. Soc Personal Psychol Compass. 2015; 9:255–68.
5. Ministry of Health. Tier 1 statistics 2015/16: New Zealand Health Survey. Wellington: Ministry of Health, 2016.
6. Theodore R, McLean R, TeMorenga L. Challenges to addressing obesity for Māori in Aotearoa/New Zealand. Aust N Z J Public Health. 2015; 39:509–12.
7. Ministry of Health. New Zealand Health Strategy Future direction. Wellington: New Zealand Ministry of Health, 2016.
8. Wong G. Challenge science supports social investment in children. Auckland: University of Auckland, 2017.
9. Lewis KH, Basu S. Epidemiology of obesity in the United States. Metabolic Syndrome: A Comprehensive Textbook. 2016:13–31.
10. Monasta L, Batty G, Cattaneo A, et al. Early-life determinants of overweight and obesity: A review of systematic reviews. Obes Rev. 2010; 11:695–708.
11. Baidal JAW, Locks LM, Cheng ER, Blake-Lamb TL, Perkins ME, Taveras EM. Risk factors for childhood obesity in the first 1,000 days: A systematic review. Am J Prev Med. 2016; 50:761–79.
12. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. The Lancet. 2011; 378:804–14.
13. Fergusson DM, McLeod GFH, Horwood LJ. Breastfeeding, infant growth and body mass index at 30 and 35 years. Paediatr Perinat Epidemiol, 2014; 545–52.
14. Belsky DW, Moffitt TE, Houts R, et al. Polygenic risk, rapid childhood growth, and the development of obesity: Evidence from a 4-decade longitudinal study. Arch Pediatr Adolesc Med. 2012; 166:515–21.
15. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and

long-term risk for obesity: A 32-year prospective birth cohort study. Pediatrics. 2008; 122:955–60.

16. Fergusson DM, Horwood LJ. The Christchurch Health and Development Study: Review of findings on child and adolescent mental health. Aust N Z J Psychiatry. 2001; 35:287–96.
17. Fergusson DM, Horwood LJ. The Christchurch Health and Development Study. In: Joyce P, Nicholls G, Thomas K, Wilkinson T, (eds) The Christchurch Experience: 40 Years of Research and Teaching. Christchurch: University of Otago, 2013; 79–87.
18. Gundersen C, Mahatmya D, Garasky S, Lohman B. Linking psychosocial stressors and childhood obesity. Obes Rev. 2011; 12:e54–e63.
19. Elley WB, Irving JC. Revised socio-economic index for New Zealand. NZJES. 1976; 11:25–36.
20. Wechsler D. Manual for the Wechsler Intelligence Scale for Children - Revised. New York: Psychological Corporation, 1974.
21. Fergusson DM, Horwood LJ, Ridder E. Show me the child at seven: The consequences of conduct problems in childhood for psychosocial functioning in adulthood. J Child Psychol Psychiatry. 2005; 46:837–49.
22. Fergusson DM, Boden JM, Horwood LJ. Exposure to childhood sexual and physical abuse and adjustment in early adulthood. Child Abuse & Neglect. 2008; 32:607–19.
23. Zeger SL, Liang K-Y. Longitudinal data analysis for discrete and continuous outcomes. Biometrics. 1986; 42:121–30.
24. Wooldridge JM. Econometric analysis of cross section and panel data. 2nd ed. Cambridge, MA: MIT Press, 2010.
25. Carlin JB, Wolfe R, Coffey C, Patton GC. Analysis of binary outcomes in longitudinal studies using weighted estimating equations and discrete-time survival methods: Prevalence and incidence of smoking in an adolescent cohort. Stat Med. 1999; 18:2655–79.
26. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes. 2008; 32:S56–S9.
27. National Heart Lung and Blood Institute. Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults: The Evidence Report. Obesity Education Initiative Expert Panel on the Identification Evaluation and Treatment of Obesity in Adults (US). Bethesda (MD): National Heart, Lung, and Blood Institute, 1998.
28. Allison KC, Sarwer DB. Diet, Exercise, and Behavior Therapy in the Treatment of Obesity and Metabolic Syndrome In: Ahima RS, (ed) Metabolic Syndrome. Switzerland: Springer International Publishing 2016; 783–98.
29. Cloutier MM, Wiley J, Wang Z, Grant A, Gorin AA. The Early Childhood Obesity Prevention Program (ECHO): An ecologically-based intervention delivered by home visitors for newborns and their mothers. BMC Public Health. 2015; 15:584.
30. Blake-Lamb TL, Locks LM, Perkins ME, Baidal JAW, Cheng ER, Taveras EM. Interventions for childhood obesity in the first 1,000 days a systematic review. Am J Prev Med. 2016; 50:780–9.
31. Royal New Zealand Plunket Society Incorporated. Plunket: What we do. Wellington: Royal New Zealand Plunket Society Incorporated, cited 23 January 2017. Available from: http://www.plunket.org.nz/what-we-do/
32. Early Start Project. Early Start Project. Christchurch: Early Start Project, cited 23 January 2017. Available from: http://www.earlystart.co.nz/
33. Ministry of Health. Family Start. Wellington: Ministry of Health, cited 23 January 2017. Available from: http://www.health.govt.nz/our-work/life-stages/child-health/well-child-tamariki-ora-services/family-start
34. Legge J. Review of New Zealand Longitudinal Studies. Wellington: Families Commission 2005.