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ORIGINAL ARTICLE

Predictive associations between alternative measures of childhood adiposity and adult cardio-metabolic health

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Objective: To estimate associations between alternative measures of childhood adiposity and indicators of cardio-metabolic health in adulthood, both unadjusted and adjusted for changes in adiposity from childhood to adulthood.
Design and Methods: The study consisted of a 20-year follow-up of 2188 adults who had participated in the 1985 Australian Schools Health and Fitness Survey when they were between 7 and 15 years of age. Baseline and follow-up measures of body composition included height and weight, waist and hip circumferences and skinfold thicknesses at four sites. At follow-up, participants attended study clinics where component indicators of the metabolic syndrome (MetS) (waist circumference, blood pressure, fasting blood glucose and lipids) were measured.
Results: Waist circumference and skinfold measures were the strongest predictors of subsequent MetS (2009 Joint Scientific Statement definition) in early adulthood. For example, relative risks (RRs) for children in the highest (vs lowest) quarter of waist circumference were 4.8 (95% confidence interval (CI): 2.5–9.2) for males and 5.8 (95% CI: 2.4–14.2) for females. After adjusting for change in waist circumference from childhood to adulthood, each 10 cm increase in childhood waist circumference was associated with an approximate twofold increase in risk for adult MetS (RR = 2.1 (95% CI: 1.7–2.7) among males and RR = 2.3 (95% CI: 1.6–3.4) among females).
Conclusion: Elevated waist circumference and skinfold thickness measures in childhood appear to be the strongest predictors of subsequent MetS in early adulthood. The increased risk associated with higher waist circumference in childhood appears to be independent of changes in waist circumference from childhood to adulthood.
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Keywords: childhood obesity; metabolic syndrome; measurement; longitudinal study

Introduction

It is well known that the prevalence of overweight and obesity in children and adolescents has increased dramatically over recent decades, especially in developed countries.[1] These increases have been linked, in turn, to increases in the prevalence of a range of adverse cardio-metabolic health indicators in pre-adult populations, including the metabolic syndrome (MetS) and type 2 diabetes mellitus.[2] Higher body mass in childhood has also been associated with an increased risk of later cardiovascular mortality and morbidity in some,[3–5] but not all,[6,7] studies which have followed historical cohorts of children into adulthood. Only a limited number of studies, however, have estimated the extent to which the higher childhood and adolescent body composition values observed in more contemporary cohorts predict the occurrence of adverse cardio-metabolic health consequences in early adulthood.[8–10] Of the studies which have explored this issue, all but a few have relied upon the body mass index (BMI) as their sole or primary measure of body composition. Although BMI is commonly used and easy to obtain, it remains unclear whether it is superior to other feasible measures of childhood body composition (for example, waist circumference, skinfold thickness) as an indicator of long-term cardio-metabolic health risk.[11]

In addition to quantifying the extent to which higher childhood body composition predicts future cardio-metabolic risk, it is important to separate the effects of

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childhood adiposity from those due to post-childhood changes in body composition. Some previous studies have attempted to address this issue by adjusting childhood effects for adult body composition levels.[10,12,13] However, such an approach effectively removes any indirect effects of childhood adiposity that act through higher levels of adult body composition due to tracking over time and may also bias childhood effect estimates due to the 'reversal paradox'.[14]

This study sought to address the above gaps in current understanding of the long-term cardio-metabolic risks associated with excess adiposity in childhood. Specifically, predictive associations between childhood adiposity and adult cardio-metabolic health were compared across a range of alternative, yet feasible, measures of body composition. These associations were examined both unadjusted and adjusted for subsequent changes in body composition from childhood to adulthood.

Materials and methods

Data for these analyses were collected as part of the Childhood Determinants of Adult Health study, a prospective cohort study with baseline data collected in 1985 on a representative sample of 8498 school children aged 7–15 years as part of the Australian Schools Health and Fitness Survey (ASHFS). Sampling procedures and methods of data collection for ASHFS have been presented elsewhere.[15] Follow-up of ASHFS participants was performed from May 2004 to May 2006, when aged 26–36 years. In total, 60.8% ($n=5170$) were enrolled and provided follow-up data, 19.5% ($n=1658$) were unable to be traced, 9.6% ($n=817$) did not respond to contact, 9.0% ($n=767$) refused to participate and 1.0% ($n=86$) were deceased. Of those enrolled at follow-up, 2410 attended 1 of 34 study clinics held throughout Australia for physical measurements and blood sampling. Clinics were located to maximize the proportion of participants living within a 10 km radius, of which 55% attended. The studies were ethically approved and written informed consent obtained.

These analyses were restricted to the 2188 non-pregnant participants who provided a fasting blood sample at the follow-up study clinic and had weight, height and waist circumference measured at both study time points. Participants were similar to the baseline sample according to sex (49.9 vs 49.1% female, $P=0.55$), childhood physical activity (442 vs 439 min per week, $P=0.81$) and childhood smoking status (43.8 vs 44.5% ever smokers, $P=0.57$). In contrast, participants had a lower BMI (18.1 vs 18.3 kg m^{-2}, $P=0.01$) and were more likely to have lived in a higher socioeconomic area (28.3 vs 22.0% in highest category, $P<0.001$) in childhood. However, the increase in BMI from child to adulthood was very similar between participants in this analysis and the 2615 non-participants who reported height and weight at follow-up enrolment (mean increase in BMI 6.8 vs 6.9 kg m^{-2}, $P=0.93$).

Body composition measures

Height and weight were measured at both time points and BMI calculated as weight, kg/(height, m)^2. Waist circumference was taken at the level of the umbilicus at baseline and the narrowest point between the lower costal border and iliac crest at follow-up. Hip circumference was measured at the level of the greatest posterior protuberance of the buttocks. Waist-to-hip ratio (WHR) was calculated by dividing waist by hip circumference and waist-to-height ratio was calculated by dividing waist circumference by height.

Among those aged 9, 12 or 15 years at baseline, tricep, bicep, subscapular and suprailiac skinfolds were measured to the nearest 0.1 mm using Holtain Calipers (Holtain, Crymych, UK). At follow-up, skinfold thicknesses were measured to the nearest 0.5 mm at the tricep, bicep, iliac crest and supraspinale, and values >40 mm were truncated and 'true' skinfold values imputed from BMI and waist circumference using Tobit Regression.[16] The sum of skinfold measurements at each time point was used for analysis.

Clinical measures at follow-up

Blood pressure was measured using an OMRON HEM907 Digital Automatic Blood Pressure Monitor (Omron Corporation, Kyoto, Japan) with the mean of three readings used for analysis. Blood samples were collected after a 12 h overnight fast and assays conducted to enzymatically measure fasting glucose, high-density lipoprotein cholesterol and triglycerides using an Olympus AU5400 automated analyzer (Olympus Optical, Tokyo, Japan). Fasting plasma insulin was measured by a microparticle enzyme immunoassay kit (AxSYM, Abbot Laboratories, Abbot Park, IL, USA) and by electrochemiluminescence immunoassay (Elecsys Modular Analytics E170; Roche Diagnostics, Mannheim, Switzerland) with inter-assay standardization.

Metabolic syndrome and continuous metabolic syndrome score

Metabolic syndrome (MetS) status was determined using the 2009 definition proposed jointly by the International Diabetes Federation, National Heart, Lung and Blood Institute, American Heart Association, World Heart Federation, International Atherosclerosis Society, and International Association for the Study of Obesity.[17] Specifically, participants were classified as having MetS when three or more of the following conditions were met: waist circumference $\geqslant 102$ cm (men) or $\geqslant 88$ cm (women); triglycerides $\geqslant 1.70$ mmol l^{-1} or specific drug treatment for elevated triglycerides; high-density lipoprotein cholesterol <1.0 mmol l^{-1} in men or <1.29 mmol l^{-1} in women or specific treatment for this lipid abnormality; raised blood pressure (systolic $\geqslant 130$ mmHg or diastolic $\geqslant 85$ mmHg or treatment of previously diagnosed hypertension); and fasting plasma glucose $\geqslant 5.6$ mmol l^{-1} or specific treatment for elevated blood glucose. The waist circumference cut-off points cited in the above definition were the higher threshold values

recommended for use in Caucasian populations. However, as also recommended in the joint statement, we created for comparison an alternative classification of MetS using waist circumference threshold values of $\geqslant$94 cm (men) and $\geqslant$80 cm (women). Select results using this alternative MetS definition are presented in the online-only appendix.

A continuous MetS risk score was also created using the methods described by Wijndaele *et al*.[18] Briefly, principal component analysis with varimax rotation was applied separately by sex to the normalized MetS risk factors to derive the principal components accounting for large proportions (eigenvalue $\geqslant$1.0) of the MetS variance. Similar to previous studies using this method, two similar principal components were identified which explained 34 and 26% of the variance in men and 31 and 25% of the variance in women. These principal components were then summed, weighted according to the relative proportion of variance explained, to compute continuous MetS risk score.

Covariate measures

Data on smoking, alcohol intake, socioeconomic status and physical activity at baseline and follow-up were ascertained using self-completed questionnaires. Residential postcode at baseline was used to derive area-level socioeconomic status based on the Australian Bureau of Statistics index of relative socioeconomic disadvantage derived from the 1981 population census, with the study subjects classified into quartiles. Additional details have been published elsewhere.[19]

Statistical analyses

Baseline characteristics of subjects included in this analysis were compared with other subjects using the *t*-test for continuous variables and χ^2-test for categorical variables (results presented earlier). Mean and standard deviation values or, for skewed variables, median and interquartile values were calculated for measures of body composition in childhood and adulthood and for adult cardio-metabolic risk factors. *A priori*, all further analyses were initially conducted separately by sex and childhood age (7–11 vs 12–15 years; roughly corresponding to pubertal status) and stratified results presented when substantial differences were observed. Spearman correlations were used to examine tracking of body composition measures from childhood to adulthood and to assess rank-order associations between alternative measures of body composition taken in childhood and continuous cardio-metabolic risk factors in adulthood adjusted for sex, length of follow-up and age. Predictive associations between alternative childhood body composition measures and MetS in adulthood were quantified using Poisson log-linear regression models. Identical logistic regression models were run in order to obtain area under receiver–operating curve (AUC) values for each childhood body composition measure.

Using the life course analysis approach outlined by De Stavola *et al*.,[20] Poisson log-linear regression was used to quantify the total (that is, mediated through adult waist circumference and unmediated) effect of higher waist circumference during childhood on adult cardio-metabolic risk indicators and the effect of changes in waist circumference over the follow-up period. These associations are presented both minimally adjusted for age and length of follow-up as well as adjusted for adult alcohol consumption and smoking status, which were found to change some estimates of association by $\geqslant$10% when added to the minimally adjusted model. Further adjustment for socioeconomic status, smoking status, alcohol consumption and physical activity in childhood or for physical activity, education and diet in adulthood did not substantially change estimates of association between waist circumference and cardio-metabolic risk and were, therefore, not included in final models. SAS Version 9.1 (SAS Institute, Cary, NC, USA) was used for all analytic procedures.

Results

Childhood and adult body composition and adult cardio-metabolic measures are described for the analysis sample in Table 1. The mean (s.d.) length of follow-up was 19.9 (0.6) years and the mean (s.d.) age at follow-up was 31.5 (2.6) years. Spearman correlations between body composition measures taken in childhood and those taken in adulthood were strongest for BMI (0.54 for males and females), somewhat weaker for waist circumference, waist-to-height ratio and skinfold measures (0.42–0.45 for males and 0.45–0.46 for females) and lowest for WHR (0.24 in males and females). Correlations between baseline and follow-up measures of WHR, waist-to-height ratio and skinfolds were stronger for those aged 12–15 years at baseline compared with those aged 7–11 years (see Supplementary Table S1 in the online-only Supplementary information).

After adjusting for age and length of follow-up, childhood body composition measures were most strongly correlated with adult insulin and continuous MetS risk score measures with weaker, but statistically significant, associations observed with all other cardio-metabolic risk measures except for systolic blood pressure (Table 2). The sum of skinfolds measured in childhood was the body composition measure most strongly correlated with many of the adult cardio-metabolic risk factors. Although this measure was only taken on those aged 9, 12 or 15 years at baseline, results were similar when all body composition measures were restricted to this subset (data not shown). Correlations for the other childhood body composition measures were similar with the exception of WHR, for which substantially weaker correlations were observed. Correlations were generally stronger among those who were older at baseline.

The relative risk of developing MetS in adulthood across quarters of alternative childhood body composition measures are shown in Table 3. Because of significant effect

Table 1 Participant characteristics in childhood (1985) and adulthood (2004–2006), by sex, Childhood Determinants of Adult Health study

	n	*Male*	n	*Female*
Body composition measures				
Childhood				
Weight, kg	1097	41.0 ± 13.5	1091	39.4 ± 12.1
Height, cm	1097	148.5 ± 16.1	1091	145.5 ± 14.4
Waist circumference, cm	1097	64.7 ± 8.2	1091	61.9 ± 7.8
Waist-to-hip ratio	1096	0.85 ± 0.04	1091	0.81 ± 0.06
Body mass index, kg m^{-2}	1097	18.1 ± 2.6	1091	18.1 ± 2.8
Sum of skinfold thicknesses	366	26.0 (21.8–32.8)	384	37.0 (29.4–46.5)
Waist-to-height ratio	1097	0.44 ± 0.03	1091	0.43 ± 0.04
Adulthood				
Weight, kg	1097	85.5 ± 14.7	1091	68.4 ± 14.8
Height, cm	1097	179.6 ± 6.7	1091	165.8 ± 6.3
Waist circumference, cm	1097	89.4 ± 10.4	1091	77.8 ± 11.2
Waist-to-hip ratio	1097	0.85 ± 0.05	1091	0.75 ± 0.06
Body mass index, kg m^{-2}	1097	26.5 ± 4.2	1091	24.9 ± 5.1
Sum of skinfold thicknesses	1090	65.7 ± 26.6	1084	78.3 ± 31.9
Waist-to-height ratio	1097	0.50 ± 0.06	1091	0.47 ± 0.07
Adult cardio-metabolic measures				
Systolic blood pressure (mm Hg)	1097	125.3 ± 10.8	1091	111.4 ± 10.3
Diastolic blood pressure (mm Hg)	1097	75.0 ± 8.9	1091	70.4 ± 8.6
HDL cholesterol (mmol l^{-1})	1097	1.29 ± 0.26	1091	1.54 ± 0.34
Triglycerides (mmol l^{-1})	1097	1.0 (0.7–1.6)	1091	0.8 (0.6–1.1)
Fasting glucose (mmol l^{-1})	1097	5.18 ± 0.45	1091	4.84 ± 0.40
Insulin (mmol l^{-1})	1094	6.1 (4.3–9.1)	1088	5.8 (4.3–8.2)
cMetS risk score[a]	1097	0.0 ± 0.71	1091	0.0 ± 0.71
Metabolic syndrome[b], %	1097	10.3	1091	3.9

Abbreviations: cMetS, continuous metabolic syndrome risk score; HDL, high-density lipoprotein. Data are means ± s.d. or median (interquartile range) unless otherwise noted. [a]Defined as proposed in a 2009 joint statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity using WC thresholds of ⩾102 cm for men and ⩾88 cm for women.

Table 2 Spearman correlations between body composition measures in childhood with cardio-metabolic risk factor levels in adulthood by childhood age

Body composition measure	*Risk factors in adulthood*						
	Systolic blood pressure	*Diastolic blood pressure*	*HDL cholesterol*	*Triglycerides*	*Glucose*	*Insulin*	*cMetS*
Aged 7–11 years							
Body mass index	0.03	0.07*	−0.06*	0.05	0.06*	0.11**	0.24**
Waist circumference	0.04	0.06*	−0.05	0.03	0.04	0.16**	0.24**
Waist-to-hip ratio	−0.03	0.02	−0.01	0.04	−0.01	0.07*	0.05
Waist-to-height ratio	−0.02	0.06*	−0.03	0.07*	0.01	0.11**	0.20**
Sum of skinfolds	0.05	0.12	−0.04	0.15*	0.03	0.13*	0.22**
Aged 12–15 years							
Body mass index	0.05	0.11**	−0.11**	0.14**	0.12**	0.19**	0.30**
Waist circumference	0.06	0.12**	−0.14**	0.16**	0.08*	0.22**	0.33**
Waist-to-hip ratio	0.04	0.05	−0.04	0.06	0.00	0.11**	0.13**
Waist-to-height ratio	0.04	0.09**	−0.11**	0.16**	0.07*	0.23**	0.29**
Sum of skinfolds	0.08	0.15**	−0.16**	0.15**	0.09	0.25**	0.36**

Abbreviations: cMetS, continuous metabolic syndrome risk score; HDL, high-density lipoprotein. Data are Spearman correlation coefficients adjusted for age at measurement, sex and length of follow-up. Except for sum of skinfolds, analyses based on the 1210 participants aged 7–11 years and 971 participants aged 12–15 years with complete data on all risk factors; analyses of skinfolds based on $n = 258$ participants aged 7–11 years and $n = 490$ participants aged 12–15 years. *$P < 0.05$; **$P < 0.001$.

modification by sex that was observed for some body composition measures, results are presented separately by sex. Although all measures of childhood body composition were predictive of adult MetS risk, significant differences in predictive ability were also observed as indicated by AUC values. Among males, waist circumference was the best

Table 3 Association between childhood body composition measures and metabolic syndrome in adulthood, by sex, Childhood Determinants of Adult Health study, 1985–2006

Childhood body composition	*Metabolic syndrome*[a] *in adult males*				*Metabolic syndrome in adult females*			
	N/n	%	*RR*[b]	*95% CI*	N/n	%	*RR*[b]	*95% CI*
Waist circumference								
First quarter	257/11	4.3	1.0	REF	304/3	1.0	1.0	REF
Second	296/31	10.5	2.5	1.2, 4.9	310/10	3.2	3.3	0.9, 12.0
Third	295/20	6.8	1.6	0.8, 3.3	253/9	3.6	3.6	1.0, 13.4
Fourth quarter	249/51	20.5	4.8	2.5, 9.2	224/21	9.4	5.8	2.4, 14.2
P_{trend}			<0.0001				<0.0001	
AUC				0.686				0.721
Body mass index								
First quarter	279/16	5.7	1.0	REF	313/6	1.9	1.0	REF
Second	314/28	8.9	1.5	0.8, 2.8	269/5	1.9	1.0	0.3, 3.3
Third	258/26	10.1	1.7	0.9, 3.2	285/11	3.9	2.0	0.7, 5.4
Fourth quarter	246/43	17.5	3.0	1.7, 5.4	224/21	9.4	5.0	2.0, 12.3
P_{trend}			<0.0001				<0.001	
AUC				0.641				0.705
Waist-to-hip ratio								
First quarter	287/21	7.3	1.0	REF	299/4	1.3	1.0	REF
Second	300/28	9.3	1.3	0.7, 2.2	281/14	5.0	3.9	1.3, 11.8
Third	268/26	9.7	1.3	0.7, 2.3	281/11	3.9	2.9	0.9, 9.1
Fourth quarter	241/38	15.8	2.1	1.3, 3.6	230/14	6.1	4.6	1.5, 14.1
P_{trend}			0.02				0.02	
AUC				0.612				0.672
Sum of skinfolds								
First quarter	94/6	6.4	1.0	REF	104/1	1.0	1.0	REF
Second	99/9	9.1	1.5	0.5, 4.1	113/1	0.9	0.9	0.1, 14.8
Third	102/11	10.8	1.7	0.6, 2.0	97/3	3.1	3.4	0.4, 32.6
Fourth quarter	71/11	15.5	2.6	1.0, 7.2	70/7	10.0	11.2	1.4, 91.3
P_{trend}			<0.001				<0.01	
AUC				0.624				0.777

Abbreviations: AUC, area under the curve; CI, confidence interval. [a]Defined as proposed in a 2009 joint statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity using waist circumference thresholds of $\geqslant$102 cm for men and $\geqslant$88 cm for women. [b]Relative risks (RRs) from Poisson regression in log-linear model adjusted for childhood age and length of follow-up.

predictor of future MetS risk (AUC = 0.686), with those in the top age-specific quarter of waist circumference being at approximately five times greater risk than those who were in the bottom quarter (absolute risk difference of 16.2%). Among females, skinfold thickness in childhood was the strongest predictor of adult MetS (AUC = 0.777), with those in the top age-specific quarter having a 11-fold higher risk compared with those who were in the bottom quarter. However, this relative risk was for the subsample aged 9, 12 or 15 years at baseline and also reflects the very low risk (1.0%) of MetS observed among those in the lowest reference quarter of skinfold thickness. Among the entire sample of female participants, waist circumference was the most predictive measure of adult MetS risk (AUC = 0.721) and the absolute risk difference across extreme quarters was similar to that observed for skinfold thickness in the subsample (8.4 vs 9.0%). When MetS was defined using the lower waist circumference thresholds, observed associations were similar but somewhat attenuated for all body composition measures with the exception of skinfold thickness (see Supplementary Table S2 in the online-only Supplementary information).

Childhood waist circumference continued to be a significant predictor of MetS in young adulthood after adjusting for subsequent change in waist circumference from childhood to adulthood (Table 4). Among the component indicators of MetS, waist circumference in childhood was most strongly associated with elevated triglyceride levels among males and elevated glucose and blood pressure among females. Compared with an identical difference in childhood waist circumference, a 10 cm change in waist circumference from childhood to adulthood was associated with a larger increase in risk for most component risk indicators, and for MetS in females but not males. Absolute percentages of adult MetS across categories of childhood waist circumference and change in waist circumference from childhood to adulthood are graphically displayed in Figure 1.

Table 4 Effect of childhood waist circumference and change in waist circumference from childhood to adulthood on risk of elevated cardio-metabolic health indicators in adulthood, by sex

Outcome	*Predictor*	*Males*		*Females*	
		RR[a] *(95% CI)*	*RR*[b] *(95% CI)*	*RR*[a] *(95% CI)*	*RR*[b] *(95% CI)*
Elevated fasting glucose	Child WC	1.1 (0.9, 1.4)	1.1 (0.9, 1.4)	1.5 (1.1, 2.1)	1.5 (1.1, 2.2)
	Change in WC	1.4 (1.2, 1.6)	1.4 (1.2, 1.6)	1.8 (1.5, 2.2)	1.8 (1.5, 2.2)
Elevated blood pressure	Child WC	1.2 (1.0, 1.4)	1.2 (1.0, 1.4)	1.5 (1.2, 2.0)	1.6 (1.2, 2.1)
	Change in WC	1.2 (1.1, 1.4)	1.2 (1.1, 1.4)	1.5 (1.3, 1.8)	1.5 (1.3, 1.8)
Elevated triglycerides	Child WC	1.4 (1.2, 1.7)	1.4 (1.2, 1.7)	1.3 (0.9, 1.7)	1.2 (0.9, 1.7)
	Change in WC	1.6 (1.5, 1.8)	1.6 (1.5, 1.8)	1.7 (1.5, 2.0)	1.7 (1.5, 2.0)
Low HDL cholesterol	Child WC	1.3 (1.0, 1.6)	1.3 (1.0, 1.6)	1.3 (1.1, 1.6)	1.3 (1.1, 1.5)
	Change in WC	1.6 (1.4, 1.8)	1.6 (1.4, 1.8)	1.5 (1.3, 1.6)	1.4 (1.3, 1.6)
Insulin ⩾75th	Child WC	1.6 (1.3, 1.9)	1.6 (1.3, 1.9)	1.6 (1.3, 1.8)	1.5 (1.3, 1.8)
	Change in WC	1.9 (1.7, 2.0)	1.9 (1.7, 2.0)	1.7 (1.6, 1.9)	1.7 (1.5, 1.8)
Metabolic syndrome[c]	Child WC	2.1 (1.7, 2.6)	2.1 (1.7, 2.6)	2.4 (1.7, 3.5)	2.3 (1.6, 3.4)
	Change in WC	2.0 (1.8, 2.3)	2.0 (1.8, 2.4)	2.7 (2.2, 3.2)	2.8 (2.2, 3.5)

Abbreviations: CI, confidence interval; HDL, high-density lipoprotein; WC, waist circumference. [a]Relative risks (RRs) for a 10 cm increase in WC measures adjusted for the other WC measure (child WC or change in WC), age at baseline and length of follow-up. [b]In addition, adjusted for adult smoking status and alcohol consumption. ($n=1017$ males, $n=1045$ females). [c]Defined as proposed in a 2009 joint statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity using WC thresholds of ⩾102 cm for men and ⩾88 cm for women.

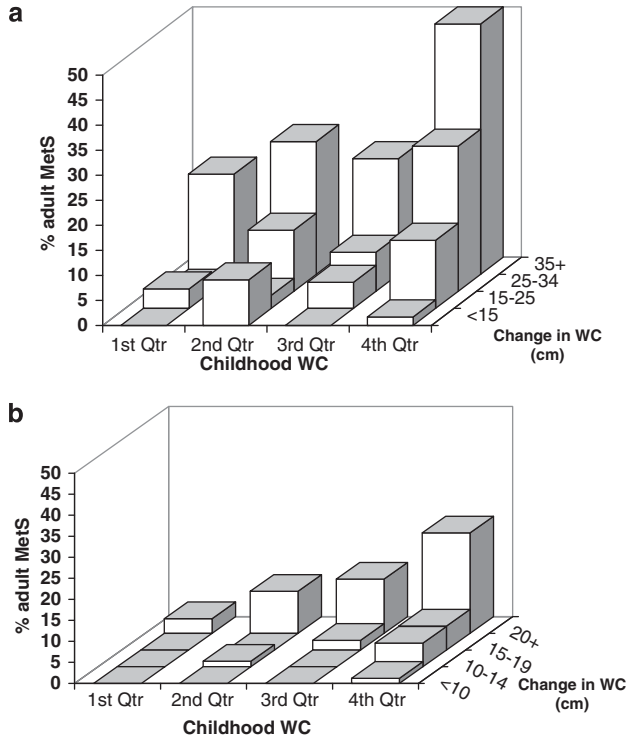

Figure 1 Percentage of males (**a**) and females (**b**) with adult metabolic syndrome across categories of childhood waist circumference (WC) and change in waist circumference from childhood to adulthood.

Discussion

This study examined the association between adiposity levels in childhood and cardio-metabolic health in early adulthood and compared the strength of this association across a range of alternative body composition measures. Although all of the alternative measures of childhood body composition were significantly associated with subsequent cardio-metabolic health, the strongest predictors of adult MetS were waist circumference and, in a female subsample, the sum of skinfolds. Specifically, participants whose waist circumference was in the top 25% for their sex and childhood age were 5–6 times more likely to be classified with MetS at age 26–36 years than were participants with childhood waist circumferences in the bottom quarter.

The findings of this study that higher levels of adiposity in childhood are associated with an increased risk of cardio-metabolic disease in adulthood is consistent with results from most,[21,22] but not all,[6,7] previous longitudinal studies. For example, Baker *et al.*[3] observed that a 1-unit increase in BMI *z*-score among a large cohort of Danish school children aged 7–13 years was associated with a 10–22% increased risk among boys, and a 5–15% increased risk among girls, of having a coronary heart disease event in adulthood. More similar to this study was findings from the Bogalusa Heart Study in the US, which reported that children in the highest age-, race- and sex-specific BMI quartile when aged 8–17 years were 10 times more likely to meet criteria for Syndrome X

(that is, MetS) in early adulthood than were children in the lowest BMI quartile.[9] In contrast, an analysis of data from a birth cohort of over 11 000 participants in Aberdeen Scotland found no association between BMI at primary school entry and incident coronary heart disease in later adulthood, although those who were classified as obese in childhood (highest 2.5% of the BMI distribution) had a 2.4 times greater risk of stroke compared with all other children.[6]

One important limitation in current understanding of the association between childhood adiposity and adult health is the limited range of measures that have been used to assess body composition. Almost all previous studies have used BMI as the primary measure of adiposity, most likely due to the common availability of height and weight data even in historical cohorts of children. Although a few contemporary cohorts such as the Bogalusa Heart Study and the Cardiovascular Risk in Young Finns Study have also measured skinfolds in childhood, none to our knowledge have directly compared the utility of these two measures with other potentially useful measures such as waist circumference and the WHR. This gap, which has been noted by other research groups,[6] is noteworthy given the unique risks associated with centrally distributed adiposity[23] and the fact that waist circumference has been observed in cross-sectional studies to be a better predictor of cardiovascular disease risk factors in childhood[11] and to predict cardio-metabolic health risk in adults beyond that explained by BMI.[24] Nonetheless, the range of measures included in this study, while practical for clinical use and population-wide screening, are not gold-standard measures of childhood body composition and this limitation should be considered when interpreting the findings of this study.

Another limitation of most previous studies is a failure to account for changes in body composition from childhood to adulthood. If the objective of the analysis is solely to examine the predictive relationship between childhood body composition and adult health outcomes, this limitation is of minor concern. However, if a goal is to disentangle the effects of elevated adiposity in childhood from the effects of adult adiposity, it becomes necessary to account for changes in adiposity over time. A few studies have attempted to adjust for adult body composition values by simultaneously including childhood and adult BMI values in their regression models.[10,12,13,25] Coefficients from models such as these can be difficult to interpret as the childhood and adult values are essentially repeated measures of the same variable and the adult measure of body composition likely lies on the causal pathway between childhood body composition and the adult health outcome.[20] In this study, a life course analysis approach was taken in which the effects of childhood waist circumference were adjusted for changes in waist circumference from childhood to adulthood. In this model, the childhood coefficient quantifies the combined direct and indirect effects of higher childhood waist circumference on adult cardio-metabolic health indicators, in which the additional risk attributable to changes over time is quantified by the coefficient for the change in waist circumference variable. Using this approach, the adverse risks associated with higher waist circumference levels in childhood were observed to be independent of the risks associated with changes in abdominal adiposity from childhood to adulthood. However, change in waist circumference was a stronger predictor for most indicators of cardio-metabolic risk in adulthood. Thus, although childhood body composition appears to have a significant effect on adult cardio-metabolic risk, changes in body composition later in life appear to have even stronger effects.

An additional strength of this study was its use of a relatively contemporary cohort who had childhood body composition measurements taken in 1985. Most previous studies have followed up on historical cohorts of children lacking the extreme body composition values observed in contemporary children. Although obesity levels in this cohort were not as extreme as those reported in present day children, study participants were fully exposed to the factors leading to the increased rates of obesity in contemporary young adults. A limitation of this study was the number of baseline participants who either did not enroll in the follow-up study or were unable to attend one of the study clinics held throughout Australia. As non-participants had higher BMI values, on average, in childhood than did participants, bias due to this loss to follow-up cannot be ruled out. However, if non-participants were also more likely to have adverse cardio-metabolic risk factor profiles in adulthood than participants, the effect of this bias would be to underestimate the magnitude of the associations observed. Thus, the observed associations between childhood body composition and adult cardio-metabolic risk may be conservative.

In summary, this report provides additional evidence that elevated body composition in childhood is a strong predictor of developing the MetS in early adulthood. Elevated waist circumference and skinfolds appear to be the strongest predictors of this risk, which is independent of changes in body composition from childhood to adulthood.

Conflict of interest

The authors declare no conflict of interest.

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