Published in final edited form as:
J Pediatr. 2009 September ; 155(3): S6.e9–S6.13. doi:10.1016/j.jpeds.2009.05.024.

Additive Utility of Family History and Waist Circumference to Body Mass Index in Childhood for Predicting Metabolic Syndrome in Adulthood

Christine M. Schubert, PhD, **Stephen Cook, MD**, **Shumei S. Sun, PhD**, and **Terry T.-K. Huang, PhD, MPH**

Department of Biostatistics, School of Medicine and Emerging School of Public Health, Virginia Commonwealth University, Richmond, VA (C.S., S.S.); Department of Pediatrics, School of Medicine, University of Rochester, Rochester, NY (S.C.); and Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD (T.H.)

Abstract

Objective—To determine whether waist circumference (WC) and family history of disease increase the predictive utility of body mass index (BMI) for adult metabolic syndrome (MetS).

Study design—A subsample of 161 men and women from the Fels Longitudinal Study with childhood and adulthood measures were analyzed. Using logistic regression, childhood BMI categories (50th, 75th, and 85th percentiles), WC categories (75th and 90th percentiles), and family history of type 2 diabetes mellitus or cardiovascular disease were modeled separately and in combinations to predict adult MetS. Predicted probabilities and *c*-statistics were compared across models.

Results—The addition of family history to BMI improved the predicted probability of adult MetS from 29% to 52% (Δ*c*-statistic = 0.13). The combination of WC and BMI was more predictive than BMI alone but did not outperform the combination of family history and BMI. In 3 of the 4 models with a combination of family history, WC, and BMI, the predicted probability of adult MetS did not exceed that from the combination of family history and BMI.

Conclusions—Family history of type 2 diabetes or cardiovascular disease is a useful addition to BMI in childhood to predict the future risk of adult MetS.

Although body mass index (BMI) and waist circumference (WC) are highly correlated, their respective role in the screening of children at risk for metabolic disorders continues to be debated. Age- and sex-adjusted BMI generally has been used to ascertain obesity in children in light of the relative ease of obtaining weight and height measurements and the ready

Reprint requests: Dr Terry Huang, Director, Obesity Research Strategic Core, Eunice Kennedy Shriver National Institute of Child Health and Human Development, 6100 Executive Boulevard, 4B11, Bethesda, MD 20892-7510. huangter@mail.nih.gov.

The contents of this article do not necessarily represent the views and policies of the National Institutes of Health.

Author Disclosures

The following authors have no financial arrangement or affiliation with any corporate organization or manufacturer of any product discussed in this supplement: Christine M. Schubert, Stephen Cook, Shumei S. Sun, Terry T.-K. Huang.

availability of standardized growth charts. In contrast, the measurement of WC in children is not well standardized. But because WC is a proxy of visceral fat, the type of adipose tissue known to predispose individuals to high risk of metabolic disease,[1] there is much interest in researching whether WC can replace or improve BMI as a screening tool.

Findings regarding BMI versus WC have been inconsistent across studies. Some reports have indicated WC as a predictor for insulin resistance, high blood pressure, and dyslipidemia, independent of BMI.[2–5] In addition, Lee et al[4] reported that in African-American and Caucasian children, the addition of WC to BMI percentile increased the explained variance in systolic blood pressure by 15% and in triglycerides and high-density lipoprotein cholesterol (HDL-C) levels by 3% and 7%, respectively. More recently, Rubin et al[6] reported that in a sample of normal and overweight adolescents, WC was superior to BMI in terms of correlations with levels of adipocytokines, including adiponectin, resistin, and interleukin-6.

In contrast to the foregoing findings, however, Garnett et al[7] reported that a high BMI at age 8 years resulted in a 7-fold increased risk for cardiovascular outcomes at age 15 years, compared with a 4-fold increase risk associated with a high WC at this age. A study of adolescents in Hong Kong found that BMI and WC demonstrated similar sensitivity and specificity levels in relation to metabolic syndrome (MetS).[8] In the Fels Longitudinal Study, in which participants were followed from birth through adulthood, the divergence of BMI and WC values occurred at around the same age (age 6 to 8 years in boys and 13 years in girls) in adults with and without MetS.[9] Finally, Janssen et al[10] found that in children age 5 to 18 years in the Bogalusa Heart Study, including BMI and WC in the same regression model to predict coronary arterial disease risk factors resulted in only minimal additional variance above that predicted by BMI or WC alone.

The inconsistent findings in the literature may be the result of collinearity in regression models. Because BMI and WC are so highly correlated, estimates of their standard errors may be unstable when both are entered simultaneously into the same regression model. As such, the relative significance of BMI or WC may not be determinable using this approach. For example, in the report of Janssen et al,[10] although the combination of BMI and WC did not seem to improve the prediction of BMI or WC alone, high WC appeared to increase the risk of coronary arterial disease within BMI subgroups in stratified analysis. Another methodological problem in the literature is that most studies reporting on this issue have not examined childhood data in relation to adult outcomes, so it is uncertain whether correlations between BMI or WC and metabolic risk factors in childhood and adolescence translate to the same patterns of prediction when adult outcomes are considered. Finally, when the goal is to improve the screening of at-risk children beyond that can be achieved by BMI, whether other measures, such as family history, can serve this purpose equally well or better than WC is unclear. Positive family history has been shown to independently predict type 2 diabetes mellitus (T2D) in a population cohort followed for 30 years.[11]

In the present study, we used data from the Fels Longitudinal Study to examine the predictive probabilities of BMI, WC, family history of T2D and cardiovascular disease, and combinations thereof in relation to adult MetS. Our use of predictive probabilities as a point

of comparison eliminated the collinearity problem between BMI and WC, because standard errors were not at issue.

Methods

Data from a subsample of 161 male and female subjects of the Fels Longitudinal Study were examined. Details of the Fels Longitudinal Study have been reported previously.[12] All subjects underwent at least one examination during childhood (age 6 to 20 years) and adulthood (25 to 55 years). Further inclusion criteria included the ability to be classified according to MetS status during adulthood and complete childhood measurements, including weight (kg) and height (cm) to calculate BMI (kg/m^2), WC (cm), and a family history for either T2D or cardiovascular disease. BMI and WC were measured using standard techniques.[12] BMI percentile was computed using the Centers for Disease Control and Prevention's 2000 growth charts.[13] WC was classified as either above or below the 75th percentile and, separately, above or below the 90th percentile, using both the reference values of Fernandez et al[14] and Cook et al (see this Supplement). Family history of T2D or cardiovascular disease was determined from questionnaire data reported in the Fels Longitudinal Study by parents and siblings of the subjects. This direct information was supplemented by the subjects' self-report of disease status for their parents and siblings. A positive family history was recorded as "yes," a negative history as "no." T2D was reported directly, but a combination of conditions was used to define cardiovascular disease, including stroke, congestive heart failure, angina, heart attack, and angioplasty or bypass surgery.

Adult MetS was chosen as the outcome measure for the present study because of the limited cases of T2D and cardiovascular disease in the study group. Adult MetS, comprising high WC, hyperglycemia, hypertriglyceridemia, low HDL-C, and hypertension, was defined based on Adult Treatment Panel III guidelines,[15] consistent with most US population-based reports. All blood chemistry measurements were obtained with the subjects in the fasting state.

Data Analysis

BMI percentile was grouped into < 50th percentile, 50th to 85th percentile, and > 85th percentile. WC percentile was grouped as > 75th percential or > 90th percentile, as described previously. These variables were used together and separately and with or without family history in logistic regressions to predict adult MetS status. Predictive values for each BMI or WC group and family history status were computed, and corresponding *c*-statistics for each model were recorded.

Results

Characteristics of the study group are summarized in Table I. Overall, 8.7% of the sample was overweight in childhood, and 19.3% had MetS in adulthood. Using the reference values of Fernandez et al,[14] 14.9% of the sample had a WC above the 75th percentile and 3.7% had a WC above the 90th percentile in childhood. Using the reference values of Cook et al (see this Supplement), these prevalences were 13.7% and 1.2%, respectively. A positive family

history of T2D or cardiovascular disease was seen in 37% of the subjects. Mean values of childhood anthropometric and adult anthropometric and blood chemistry data are given in Table II.

The predicted probabilities of BMI, family history, WC, and different combinations of these measures in relation to adult MetS are presented in Table III. The predicted probability of adult MetS was 18% to 19% in children with BMI < 85th percentile and 29% in overweight children (model 1). The inclusion of family history increased this predicted probability to 30% in normal-weight children and 52% in overweight children (model 2). Adding high WC (> 90th percentile based on the reference values of Fernandez et al[14]) to BMI yielded predicted probabilities of 34% in those with a childhood BMI between the 50th and 84th percentiles and 41% in those with a childhood BMI > 85th percentile (model 4). Comparable models were estimated using Cook et al's reference value for high WC (model 8), where the predicted probability of adding WC > 90th percentile to overweight status reached 50%.

When BMI, family history, and WC were all considered simultaneously (models 5, 6, 9, and 10), the highest predicted probability hovered around 50% in all cases of overweight status combined with a positive family history and high WC (75th or 90th percentile cutoff) in all models except model 6, in which the probability reached 61% for the combination of BMI > 85th percentile, positive family history, and WC > 90th percentile (using reference values of Fernandez et al[14]).

Discussion

This longitudinal study has examined whether adding family history and/or WC to BMI in childhood improves the predictive probability of MetS in adulthood. Our statistical approach avoided the collinearity problem in estimating standard errors in regression models while still allowing us to ascertain whether clinically significant benefits can be derived from using multiple tools for risk screening in children. Our results demonstrate a marked increase in risk for adult MetS when BMI exceeded the 85th percentile in childhood. The addition of family history to BMI significantly improved the predicted probability of adult MetS. The combination of high WC (> 90th percentile) and BMI was more predictive than BMI alone but did not outperform the combination of family history and BMI. In 3 of the 4 models in which BMI, family history, and WC were all included simultaneously, the predicted probability of adult MetS was not better than that derived from the combination of family history and BMI.

Consistent with the results of Janssen et al,[10] we found that in children with BMI between the 50th and 84th percentiles and between the 85th and 94th percentiles, WC—particularly at the 90th percentile cutpoint—increased the predicted probability of adult MetS. Another study found evidence of the utility of WC in predicting metabolic abnormalities in normal-weight children.[16] These findings suggest that WC might be useful within stratified BMI categories to discriminate children at lower versus higher metabolic risk, given a particular BMI level; however, this evidence likely is obscured by comparing only the *P* values from regression models with both BMI and WC entered simultaneously.

In 2007, an Expert Committee on childhood obesity guidelines[17] recommended blood screening of all children and adolescents with BMI > 85th percentile. But pediatricians have been reluctant to accept this recommendation, given the time and financial costs involved in screening all children in the overweight but not obese category. Our results show that among overweight but not obese children (BMI between the 85th and 94th percentile), those with no family history of T2D or cardiovascular disease had a 25% risk of adult MetS, but that this risk more than doubled, to > 50%, in the presence of a positive family history. Thus, in light of our findings, although adding WC to BMI appears to improve prediction of adult MetS, family history seems to be an even better additional measure with BMI. In most cases, family history can be assessed more easily and accurately than WC, given the latter variable's lack of standardized technique and reference values. Thus, adding family history to BMI might be useful to help identify children who should undergo more extensive screening, to reduce the clinical burden and facilitate screening adherence among pediatricians.

The present study is unique because it is the first to use extensive childhood and adulthood follow-up data, with family history and WC modeled in combination with BMI in childhood for the prediction of MetS in adulthood. However the study's long-term follow-up limited our sample size. Our sample included only 1 subject with BMI > 95th percentile in childhood, which limited our ability to compare the role of family history and WC in children with frank obesity. Furthermore, whereas others have shown differences in WC by sex and ethnicity given a specific BMI level,[18] our study included only Caucasian children, and our modest sample size did not allow for meaningful sex comparisons.

In conclusion, despite our sample's limitations, our results suggest that WC can augment BMI to refine the screening of metabolically at-risk children, but a family history of T2D and cardiovascular disease appears to increase the predicted probability of adult MetS even more than WC when considered in conjunction with BMI in childhood. Given the relative ease of assessing family history compared with WC, this may have important implications for identifying children who are overweight but not yet obese requiring additional detailed screening for metabolic risks.

Glossary

BMI	Body mass index
HDL-C	High-density lipoprotein cholesterol
MetS	Metabolic syndrome
T2D	Type 2 diabetes mellitus
WC	Waist circumference

References

1. Calabro P, Yeh ET. Intra-abdominal adiposity, inflammation, and cardiovascular risk: new insight into global cardiometabolic risk. Curr Hypertens Rep. 2008; 10(1):32–8. [PubMed: 18367024]

2. Genovesi S, Antolini L, Giussani M, Pieruzzi F, Galbiati S, Valsecchi MG, et al. Usefulness of waist circumference for the identification of childhood hypertension. J Hypertens. 2008; 26(8): 1563–70. [PubMed: 18622233]
3. Hirschler V, Aranda C, de Calcagno ML, Maccalini G, Jadzinsky M. Can waist circumference identify children with the metabolic syndrome? Arch Pediatr Adolesc Med. 2005; 159(8):740–4. [PubMed: 16061781]
4. Lee S, Bacha F, Arslanian SA. Waist circumference, blood pressure, and lipid components of the metabolic syndrome. J Pediatr. 2006; 149(6):809–16. [PubMed: 17137898]
5. Lee S, Bacha F, Gungor N, Arslanian SA. Waist circumference is an independent predictor of insulin resistance in black and white youths. J Pediatr. 2006; 148(2):188–94. [PubMed: 16492427]
6. Rubin DA, McMurray RG, Harrell JS, Hackney AC, Haqq AM. Do surrogate markers for adiposity relate to cytokines in adolescents? J Investig Med. 2008; 56(5):786–92.
7. Garnett SP, Baur LA, Srinivasan S, Lee JW, Cowell CT. Body mass index and waist circumference in midchildhood and adverse cardiovascular disease risk clustering in adolescence. Am J Clin Nutr. 2007; 86(3):549–55. [PubMed: 17823416]
8. Ng VW, Kong AP, Choi KC, Ozaki R, Wong GW, So WY, et al. BMI and waist circumference in predicting cardiovascular risk factor clustering in Chinese adolescents. Obesity (Silver Spring). 2007; 15(2):494–503. [PubMed: 17299123]
9. Sun SS, Liang R, Huang TT, Daniels SR, Arslanian S, Liu K, et al. Childhood obesity predicts adult metabolic syndrome: the Fels Longitudinal Study. J Pediatr. 2008; 152(2):191–200. [PubMed: 18206688]
10. Janssen I, Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Bouchard C, et al. Combined influence of body mass index and waist circumference on coronary artery disease risk factors among children and adolescents. Pediatrics. 2005; 115(6):1623–30. [PubMed: 15930225]
11. Morrison JA, Friedman LA, Wang P, Glueck CJ. Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later. J Pediatr. 2008; 152(2):201–6. [PubMed: 18206689]
12. Roche, AF. Growth, Maturation, and Body Composition: The Fels Longitudinal Study 1929–1991. Cambridge, UK: Cambridge University Press; 1992.
13. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, et al. CDC growth charts: United States. Adv Data. 2000; 314:1–27. [PubMed: 11183293]
14. Fernandez JR, Redden DT, Pietrobelli A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. J Pediatr. 2004; 145(4):439–44. [PubMed: 15480363]
15. Grundy SM. Metabolic syndrome scientific statement by the American Heart Association and the National Heart, Lung, and Blood Institute. Arterioscler Thromb Vasc Biol. 2005; 25(11):2243–4. [PubMed: 16258150]
16. Plachta-Danielzik S, Landsberg B, Johannsen M, Lange D, Muller MJ. Association of different obesity indices with blood pressure and blood lipids in children and adolescents. Br J Nutr. 2008; 100(1):208–18. [PubMed: 18346303]
17. Barlow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007; 120(Suppl 4):S164–92. [PubMed: 18055651]
18. Lee S, Kuk JL, Hannon TS, Arslanian SA. Race and gender differences in the relationships between anthropometrics and abdominal fat in youth. Obesity (Silver Spring). 2008; 16(5):1066–71. [PubMed: 18356853]

Table I

Sample characteristics

	n	%
Sex		
Male	69	42.9
Female	92	57.1
Race		
Caucasian	161	100.0
Family history		
Cardiovascular disease		
Yes	42	26.1
No	119	73.9
T2D		
Yes	30	18.6
No	131	81.41
Combined		
Yes	60	37.3
No	101	62.7
Adult MetS status		
Yes	31	19.3
No	130	80.8
BMI percentile		
< 50th	82	50.9
50th to 84th	64	39.8
85th to 94th	14	8.7
95th	1	0.6
WC		
Fernandez et al values		
> 90th percentile	6	3.7
> 75th percentile	24	14.9
Cook et al values		
> 90th percentile	2	1.2
> 75th percentile	22	13.7

Table II

Means of childhood and adulthood data

	n	Mean	SD
Childhood			
Age, years	161	14.52	2.61
WC, cm	161	71.55	8.43
Weight, kg	161	52.98	13.38
Height, cm	161	162.24	13.78
BMI, kg/m^2	161	19.81	2.98
BMI percentile	161	48.33	25.28
Adulthood			
Age, years	161	35.89	5.75
WC, cm	161	92.61	14.37
BMI, kg/m^2	161	26.24	5.54
Systolic blood pressure, mm Hg	160	111.15	11.98
Diastolic blood pressure, mm Hg	160	72.41	9.94
Blood chemistry			
HDL-C	155	51.17	13.08
Triglycerides	155	145.06	120.60
Glucose	156	91.79	10.38

Table III

Predicted probabilities of adult MetS

Model	*c*-statistic	Predicted probability(95% CI)
1 BMI percentile group	0.53	
<50th		0.18 (0.11–0.28)
50th–84th		0.19 (0.11–0.30)
85th–94th		0.29 (0.11–0.56)
2 BMI percentile + Hx	0.66	
<50, Hx −		0.11 (0.06–0.21)
<50, Hx +		0.29 (0.18–0.45)
50–84, Hx −		0.11 (0.05–0.22)
50–84, Hx +		0.30 (0.17–0.47)
85–94, Hx −		0.25 (0.09–0.52)
85–94, Hx +		0.52 (0.22–0.81)
WC by Fernandez et al		
3 BMI percentile + WC>75th	0.53	
<50, WC<75		0.18 (0.11–0.28)
<50, WC>75		**0.22 (0.05–0.64)**
50–84, WC<75		0.18 (0.10–0.31)
50–84, WC>75		0.22 (0.06–0.58)
85–94, WC<75		0.24 (0.04–0.71)
85–94, WC>75		0.29 (0.11–0.56)
4 BMI percentile + WC>90th	0.54	
<50, WC<90		0.18 (0.11–0.28)
<50, WC>90		**0.34 (0.05–0.82)**
50–84, WC<90		0.19 (0.11–0.30)
50–84, WC>90		0.34 (0.06–0.82)
85–94, WC<90		0.23 (0.07–0.55)
85–94, WC>90		0.41 (0.10–0.81)
5 BMI percentile + Hx + WC>75th	0.66	
<50, Hx −, WC<75		0.11 (0.05–0.21)
<50, Hx −, WC>75		**0.17 (0.03–0.58)**
<50, Hx +, WC<75		0.30 (0.18–0.45)
<50, Hx +, WC>75		**0.42 (0.09–0.84)**
50–84, Hx −, WC<75		0.11 (0.05–0.22)
50–84, Hx −, WC>75		0.17 (0.04–0.51)
50–84, Hx +, WC<75		0.29 (0.16–0.47)
50–84, Hx +, WC>75		0.41 (0.11–0.79)
84–94, Hx −, WC<75		**0.16 (0.02–0.62)**
84–94, Hx −, WC>75		0.25 (0.09–0.52)
84–94, Hx +, WC<75		**0.39 (0.07–0.85)**
84–94, Hx +, WC>75		0.53 (0.22–0.81)

Model	*c*-statistic	Predicted probability(95% CI)
6 BMI percentile + Hx + WC>90th	0.66	
<50, Hx −, WC<90		0.11 (0.06–0.21)
<50, Hx −, WC>90		**0.18 (0.02–0.69)**
<50, Hx +, WC<90		0.29 (0.17–0.45)
<50, Hx +, WC>90		**0.42 (0.07–0.87)**
50–84, Hx −, WC<90		0.11 (0.05–0.22)
50–84, Hx −, WC>90		**0.18 (0.02–0.69)**
50–84, Hx +, WC<90		0.30 (0.17–0.47)
50–84, Hx +, WC>90		0.42 (0.07–0.87)
84–94, Hx −, WC<90		0.22 (0.06–0.53)
84–94, Hx −, WC>90		0.32 (0.07–0.76)
84–94, Hx +, WC<90		**0.47 (0.16–0.81)**
84–94, Hx +, WC>90		0.61 (0.18–0.92)
WC by Cook et al		
7 BMI percentile + WC>75th	0.54	
<50, WC<75		0.18 (0.11,0.28)
<50, WC>75		**0.28 (0.07,0.69)**
50–84, WC<75		0.18 (0.10,0.30)
50–84, WC>75		0.27 (0.08,0.63)
85–94, WC<75		0.19 (0.03,0.61)
85–94, WC>75		0.29 (0.11,0.57)
8 BMI percentile + WC>90th	0.53	
<50, WC<90		0.18 (0.11,0.28)
<50, WC>90		**0.40 (0.03,0.94)**
50–84, WC<90		0.19 (0.11,0.30)
50–84, WC>90		**0.41 (0.03,0.94)**
85–94, WC<90		0.25 (0.08,0.55)
85–94, WC>90		0.50 (0.06,0.94)
9 BMI percentile + Hx + WC>75th	0.66	
<50, Hx −, WC<75		0.11 (0.05,0.21)
<50, Hx −, WC>75		**0.21 (0.04,0.62)**
<50, Hx +, WC<75		0.30 (0.18,0.45)
<50, Hx +, WC>75		**0.48 (0.13,0.86)**
50–84, Hx −, WC<75		0.10 (0.05,0.21)
50–84, Hx −, WC>75		0.20 (0.05,0.55)
50–84, Hx +, WC<75		0.28 (0.15,0.46)
50–84, Hx +, WC>75		0.46 (0.14,0.82)
84–94, Hx −, WC<75		0.13 (0.01,0.54)
84–94, Hx −, WC>75		0.25 (0.09,0.53)
84–94, Hx +, WC<75		**0.35 (0.06,0.81)**
84–94, Hx +, WC>75		0.53 (0.23,0.82)
10 BMI percentile+ Hx + WC>90th	0.66	

Model	*c*-statistic	Predicted probability(95% CI)
<50, Hx −, WC<90		0.11 (0.06–0.21)
<50, Hx −, WC>90		**0.10 (0.004–0.77)**
<50, Hx +, WC<90		0.30 (0.17–0.45)
<50, Hx +, WC>90		**0.27 (0.02–0.90)**
50–84, Hx −, WC<90		0.11 (0.05–0.23)
50–84, Hx −, WC>90		**0.10 (0.004–0.78)**
50–84, Hx +, WC<90		0.30 (0.17–0.48)
50–84, Hx +, WC>90		**0.28 (0.02–0.90)**
84–94, Hx −, WC<90		0.25 (0.08–0.55)
84–94, Hx −, WC>90		**0.23 (0.02–0.84)**
84–94, Hx +, WC<90		**0.53 (0.19–0.84)**
84–94, Hx +, WC>90		0.50 (0.06–0.94)

Hx, family history.

Values in bold are imputed due to lack of sample for those cells.