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Hostility Predicts Metabolic Syndrome Risk Factors in Children and Adolescents

Katri Räikkönen
University of Helsinki

Karen A. Matthews and Kristen Salomon
University of Pittsburgh

The authors tested in 134 African American and European American children whether hostility measured at study entry predicted the metabolic syndrome risk factors an average of 3 years later. Hostility was measured with the Cook–Medley Hostility Scale (W. W. Cook & D. M. Medley, 1954) and with ratings of Potential for Hostility from interview responses. Metabolic syndrome was based on having at least 2 of the following risk factors above the 75th percentile of scores for their age, race, and gender group: body mass index, insulin resistance index, ratio of triglycerides to high-density lipoprotein cholesterol, and mean arterial blood pressure. Children who exhibited high hostility scores at baseline were likely to exhibit the metabolic syndrome at the follow-up. The results highlight the potential importance of early prevention and intervention of behavioral risk factors for cardiovascular disease.

Key words: children, hostility, metabolic syndrome, prospective

Epidemiological studies have established that obesity, hyperinsulinemia, dyslipidemia, and elevated blood pressure, independently and in combination, predict cardiovascular morbidity and mortality events and non–insulin-dependent diabetes mellitus in adults (DeFronzo & Ferrannini, 1991; National Cholesterol Education Program, 2001; Reaven, 1988). Coexistence of obesity, hyperinsulinemia, dyslipidemia, and hypertension has been termed the metabolic syndrome. Among children and adolescents, the extent of atherosclerotic lesions is accelerated by weight, insulin, elevated lipids, and blood pressure, with some data again arguing for additive effect (Berenson, Srinivasan, Bao, et al., 1998; Berenson, Srinivasan, & Nicklas, 1998).

The clustering of cardiovascular risk factors comprising the metabolic syndrome begins in early childhood (e.g., Arslanian & Suprasongsin, 1996; Berenson, Srinivasan, Bao, et al., 1998; Bergström, Hernell, Persson, & Vessby, 1996; Chen, Srinivasan, Elkasabany, & Berenson, 1999a; Csabi, Török, Jeges, & Molnar, 2000), increases with age (Chen et al., 2000), and persists from childhood to adulthood (Katzmarzyk et al., 2001). Early determinants of the metabolic syndrome include heredity (Bao et al., 1997; Chen, Srinivasan, Elkasabany, & Berenson, 1999b; Edwards et al., 1997; Perusse, Rice, Despres, Rao, & Bouchard, 1997; Srinivasan, Elkasabani, Dalferes, Bao, & Berenson, 1998), small birth weight, weight gain and obesity in childhood (Bevdekar et al., 1999; Freedman, Dietz, Srinivasan, & Berenson, 1999; Vanhala, Vanhala, Keinänen-Kiukaanniemi, Kumpusalo, & Takala, 1999; Vanhala, Vanhala, Kumpusalo, Halonen, & Takala, 1988), endocrine abnormalities as a result of treatment in long-term survivors of childhood cancer (Talvensaari & Knip, 1997; Talvensaari, Lanning, Tapanainen, & Knip, 1996), premature puberty (Ibanez, Potau, Chacon, Pascual, & Carrascosa, 1998), and poor health habits, such as a excessive caloric intake and sedentary lifestyle (Berenson, Srinivasan, & Nicklas, 1998; Ferguson et al., 1999). Despite clear evidence that suggests that psychological attributes promote the metabolic syndrome risk factor clustering in adulthood (e.g., Räikkönen, Keltikangas-Järvinen, Aldercreutz, & Hautanen, 1996; Räikkönen, Matthews, & Kuller, 2002; Räikkönen, Matthews, Kuller, Reiber, & Bunker, 1999), only two studies have reported prospective data on psychological influences on the metabolic risk factors among youth. High baseline activity and/or aggression and anger in 6- to 15-year-old (Ravaja & Keltikangas-Järvinen, 1995) and in 12- to 21-year-old (Ravaja, Keltikangas-Järvinen, & Keskivaara, 1996) Finnish boys and young men predicted higher level of risk factors comprising the metabolic syndrome 3 years later.

The current study examined psychological influences on the clustering of cardiovascular risk factors comprising the metabolic syndrome during childhood and adolescence. The major objective was to test whether hostility at study entry was associated with the metabolic syndrome at study entry and 3 years later among a sample of African American and European American children (ages 8–10) and adolescents (ages 15–17) across an average interval of 3 years. We focused on hostility as the prime psychological attribute for several reasons. First, hostility is an important correlate of visceral adiposity, elevated blood pressure, the metabolic syndrome (Räikkönen et al., 1996; Räikkönen, Matthews, & Kuller, 2001; Räikkönen, Matthews, Kuller, et al., 1999), and cardiovascular mortality (Miller, Smith, Turner, Guijarro, & Hallet, 1996) in adulthood. Second, hostility in youth is correlated with obesity in cross-sectional and longitudinal studies (e.g., Ravaja & Keltikangas-Järvinen, 1995; Ravaja et al., 1996). Third,

Katri Räikkönen, Department of Psychology, University of Helsinki, Helsinki, Finland; Karen A. Matthews and Kristen Salomon, Department of Psychiatry, University of Pittsburgh.

Correspondence concerning this article should be addressed to Karen A. Matthews, Department of Psychiatry, University of Pittsburgh, 3811 O'Hara Street, Pittsburgh, Pennsylvania 15213. E-mail: matthewska@msx.upmc.edu

hostility is a stable characteristic in youth (Woodall & Matthews, 1993). Should hostility be associated with the risk factors comprising the metabolic syndrome, it should have long-lasting effects that persist into adulthood.

Method

Participants

One hundred thirty-four children and adolescents who participated in two testing sessions approximately 3.1 years apart (*SD* = 0.84; range = 1.4–6.2) and had complete data to categorize them into metabolic syndrome categories constituted the sample used in this investigation. They were recruited from school districts in the metropolitan Pittsburgh, PA, area with the goal of sampling children (ages 8–10) and adolescents (ages 15–17), with about equal numbers of Black and White and male and female participants. The school districts targeted served a range of socioeconomic status (SES) communities. Children whose parents had an advanced educational degree (e.g., PhD, MD, JD) were excluded. This allowed for an approximate matching by parental education across the ethnic groups. Other eligibility criteria for participation in both sessions were no history of cardiovascular disease or any condition that would require medication that might effect the cardiovascular system (e.g., high blood pressure, asthma, oral contraception), no drug or alcohol abuse, no history of mental illness, no professional counseling within the past year, less than 80% above ideal weight according to height and weight tables, no smoking within 12 hr prior to the session, and having an optional echocardiogram at Session 1 (adolescents only). Children signed an assent form and adolescents and the participants' parents signed a consent form prior to participation in the study.

An additional 15 participants had been involved in two sessions but had lacked blood specimens, had not been fasting at the time of the blood draw, or lacked blood pressure measurements; 29 had participated only in the first session but had not participated in the second because they were lost to follow-up (n = 14), they refused (n = 14), or had died (n = 1). Comparison of the baseline characteristics of the 134 participants in the present analysis and those who were eligible but did not participate in the follow-up showed no differences in the hostility measures, body mass index (BMI), blood pressure, lipids, or proportions of females or Blacks. Participants providing complete data at both study sessions were more likely to come from intact families and to have parents with higher Hollingshead scores.

Measures

Metabolic syndrome. The baseline and follow-up examinations followed the same protocols. BMI (weight in kilograms divided by height in meters squared) was used as an index of obesity. Serum glucose was measured by enzymic determination using Sigma Diagnostics (St. Louis, MO) glucose reagent for dilution and using the Abbott (Chicago, IL) VP Supersystem spectrophotometer. Serum insulin was determined by radioimmunoassay using DPC's Coat-A Count procedures. The kits for these procedures contained human sera calibrators, which had been lyophilized for maximal stability. Insulin resistance was assessed using the insulin resistance index (IRI = fasting insulin (uU/ml) × fasting glucose (mmol/L)/22.5; D. R. Matthews et al., 1985). Triglycerides (TG) were estimated using enzymatic procedures in a centrifugal analyzer and total high density lipoprotein cholesterol (HDL-C) was determined after selective precipitation by heparin/manganese chloride and removal by centrifugation of very low density and low density lipoprotein. The ratio of TG to HDL-C was used as an index of dyslipidemia (Reaven, 1988). Blood pressure (BP) levels were measured using an IBS Model SD-700A automated sphygomomanometer (IBS Corp., Waltham, MA) and a standard occluding cuff and microphone placed over the brachial artery in accordance with published guidelines. If manual readings did not match the IBS readings within 4 mm Hg, the cuff was adjusted and the procedure repeated until two consecutive matched readings were obtained. Then three measures were taken during a 10-min resting period and the last two were averaged. Mean arterial BP (diastolic BP plus one-third pulse pressure) was used in the analyses.

We used the definition of the metabolic syndrome from the Bogalusa Heart Study in children, adolescents, and young adults to classify our participants into those with and without the metabolic syndrome (Chen et al., 2000). Children were first classified whether they had scores in the upper quartile of the distributions for each age, gender, and race group for each of the following: IRI, TG to HDL-C ratio, BMI, and mean arterial BP. Hereafter, participants are described as having the metabolic syndrome if they had two or more of the risk factors in the top quartile.

Hostility. We used the 26-item version of the Cook–Medley Hostility Scale (Cook & Medley, 1954; Costa, Zonderman, McCrae, & Williams, 1985) for measuring hostile attitudes and subscales of Cynical Attitudes, Hostile Affect, and Aggressive Responding. Minor wording changes were made to make some items more age-appropriate (e.g., we changed *acquaintances* to *school friends*). The 26-item version is highly correlated with the full scale (r = .95) and demonstrates good test–retest reliability and internal consistency (Woodall & Matthews, 1993). The Type A Adolescent Structured Interview was used for measuring overall Potential for Hostility and was administered to the study participants by trained interviewers and tape recorded for coding by two trained raters. The interview is administered in such a way as to provide opportunities for competitive and hostile behaviors to emerge. Raters made a clinical judgment of Potential for Hostility, based primarily on style of responses to the interviewer rather than on the content of their answers. Additional ratings of hostile content, hostile intensity, and hostile style were made (Dembroski, MacDougall, Costa, & Grandits, 1989). Previous studies of children in our laboratory have indicated that hostility ratings based on the Type A Adolescent Structured Interview responses have adequate interrater reliability (Woodall & Matthews, 1993).

Statistical Analyses

Differences in the mean values of baseline measures of hostility between children and adolescents classified as exhibiting or not exhibiting the metabolic syndrome were compared by univariate analyses of variance (ANOVAs). Then multinomial logistic regression analyses were performed and risk ratios and 95% confidence intervals (CIs) were computed to compare those who did not have the metabolic syndrome at either examination versus those who had the metabolic syndrome at only baseline, at only the follow-up, or at both examinations.

Because the interval between the two examinations varied from 1.4 to 6.2 years, analyses were repeated with interval in years and age group at baseline as covariates. Although the metabolic syndrome categorization in the current study (cf. Chen et al., 2000) was defined within age, ethnic, and gender groups, we (Gump, Matthews, & Räikkönen, 1999) and others (Barefoot et al., 1991; Scherwitz, Perkins, Chesney, & Hughes, 1991) have previously demonstrated that these demographic variables are correlated with hostility. Therefore, we repeated the analyses with age group, interval ethnicity, and gender, as well as the family Hollingshead total score indexing SES (Hollingshead, 1985) as covariates. Hollingshead rating was based on the education and occupational prestige of the parents. If the mother was not working outside the home, the father's occupation only was considered. Logarithmic transformations were computed for non-normally distributed variables where appropriate.

Results

Table 1 presents mean values of the study variables separately for children and adolescents. Adolescents had significantly higher BMI at both examinations, IRI at the baseline examination, and

Table 1
Characteristics of the Sample at Baseline and Follow-Up

Characteristic	Children (n = 91)		Adolescents (n = 43)		
	M	*SD*	*M*	*SD*	*p*
Baseline					
Family Hollingshead total scores	31.1	10.3	35.6	9.7	.02
Cook–Medley Hostility scores					
Total	12.9	4.9	14.5	3.7	.07
Aggressive Responding	4.2	2.0	4.9	1.9	.07
Hostile Affect	2.4	1.6	2.6	1.3	.46
Cynicism	6.3	2.6	6.8	2.3	.30
Potential for Hostility ratings					
Total	2.2	0.9	2.5	0.7	.09
Content	2.6	0.8	3.2	0.8	.001
Intensity	1.4	0.7	1.6	0.8	.19
Style	1.8	0.9	1.7	0.8	.65
BMI (kg/m^2)	17.7	2.3	23.1	3.9	.001
Insulin resistance index	1.9	0.8	2.8	1.7	.001
Triglycerides/HDL-C	0.6	0.4	0.6	0.3	.60
Mean arterial blood pressure	77.3	7.3	78.4	6.3	.40
3-year follow-up					
BMI (kg/m^2)	20.2	3.2	24.5	3.6	.001
Insulin resistance index	3.2	1.4	2.9	1.6	.42
Triglycerides/HDL-C	0.8	0.6	0.9	0.6	.72
Mean arterial blood pressure	76.6	7.0	83.0	8.4	.001

Note. BMI = body mass index; HDL-C = high-density lipoprotein cholesterol.

mean BP at the follow-up examination than had children. Furthermore, adolescents had higher baseline Cook–Medley Total and Aggressive Responding scores and higher ratings on overall Potential for Hostility and Hostile Content based on their Type A structured interview responses at baseline than had children.

We also tested the effects of ethnicity, gender, and family SES Hollingshead scores on the individual risk factors comprising the metabolic syndrome and on hostility measures. White participants had higher TG/HDL-C ratios than Blacks at baseline and at follow-up (ps $< .02$), girls had higher IRI than boys at follow-up ($p < .03$), and participants from lower Hollingshead SES families exhibited higher mean arterial BP than participants from higher SES families at follow-up ($p < .02$). Black participants had higher Cook–Medley cynicism scores than Whites ($p < .001$), and boys and participants from lower Hollingshead SES families had higher Hostile Style ratings (ps $< .03$) than girls and participants from higher SES families. Other associations between the demographic variables and metabolic and hostility measures were not significant (ps $> .07$).

Pearson correlation coefficients among the measures of hostility were significant and ranged from .19 to .82 (ps $< .04$), except for the following: Interview Style with Cook–Medley scores and Interview Content with Cook–Medley Aggressive Responding (rs $< .18$, ps $> .051$).

Metabolic Syndrome Risk Factor Clustering

The number of participants with 0, 1, 2, 3, and 4 risk factors above the 75th percentile was 46, 53, 24, 7, and 4, respectively, at the baseline evaluation, and 57, 44, 21, 7, and 5, respectively, at the follow-up. The observed number of participants with 0, 1, 2, 3, and 4 risk factors above the 75th percentile differed significantly from that of expected number at both study sessions, χ^2s(4, N = 134) > 23.4, ps $< .001$. The primary reason was the observed number of participants with four risk factors above the quartile criterion was different (greater) from that expected (ps $< .001$) at both examinations.

The metabolic syndrome was exhibited by 35 participants (26.1%) at baseline, with 20 of those not exhibiting the metabolic syndrome at follow-up, and by 33 participants (24.6%) at follow-up, with 18 of those being classified as having the metabolic syndrome at follow-up only (and not at baseline).

Principal components factor analysis with varimax rotation was conducted on the risk factors. The factor analyses revealed two factors (eigenvalues = 1.78 and 1.07) from the baseline risk factors and one factor (eigenvalue = 1.89) from the follow-up risk factors, with the solutions explaining 71.6% and 47.2% of the variance. At baseline and at follow-up evaluations, respectively, factor loadings on the first factor were as follows: IRI = 0.87 and 0.69, BMI = 0.82 and 0.74, TG/HDL-C ratio = 0.57 and 0.60, and mean arterial BP = 0.10 and 0.72. At baseline, mean arterial BP loaded on the second factor at 0.91. The above analyses utilized the risk factors as continuous variables. Factor analyses using categorized risk factors data showed substantively identical results.

Hostility and the Metabolic Syndrome

Children and adolescents classified as having or not having the metabolic syndrome at baseline did not show any significant differences in the baseline measures of hostility (see Table 2). Children and adolescents with the metabolic syndrome at follow-up had higher baseline scores on the Cook–Medley Total

Table 2
Means (and Standard Deviations) of Hostility Scores at Baseline According to Metabolic Syndrome at Baseline and Follow-Up

	Metabolic syndrome					
	Baseline			Follow-up		
Hostility scores at baseline	No (n = 99)	Yes (n = 35)	p	No (n = 101)	Yes (n = 33)	p
Cook–Medley Hostility scores						
Total	13.1 (4.6)	14.1 (4.6)	.31	12.8 (4.5)	15.1 (4.6)	.02
Aggressive Responding	4.2 (2.1)	4.9 (1.8)	.09	4.2 (2.1)	5.0 (1.8)	.07
Hostile Affect	2.4 (1.5)	2.5 (1.5)	.81	2.3 (1.5)	2.9 (1.4)	.04
Cynicism	6.4 (2.5)	6.5 (2.4)	.80	6.3 (2.5)	6.7 (2.6)	.26
Potential for Hostility ratings						
Total	2.3 (0.9)	2.3 (0.8)	.97	2.3 (0.8)	2.4 (0.8)	.31
Content	2.8 (0.9)	2.8 (0.9)	.76	2.7 (0.8)	2.9 (1.0)	.31
Intensity	1.5 (0.8)	1.3 (0.5)	.26	1.4 (0.8)	1.4 (0.7)	.98
Style	1.7 (0.9)	1.8 (0.8)	.55	1.6 (0.7)	2.2 (1.1)	.001

Note. The p values are from the t tests comparing those with and without the metabolic syndrome.

Hostility and Hostile Affect scales and expressed a more Hostile Style in the Type A structured interview (see Table 2).

Tests of stability and change between the study sessions in the metabolic syndrome classification (see Table 3) showed that children and adolescents who developed the metabolic syndrome by the time of the follow-up examination, that is, were classified as having the metabolic syndrome at follow-up only; had higher baseline Cook–Medley Total, Hostile Affect, Cynicism, and Aggressive Responding Hostility scores; and had higher baseline ratings on Hostile Style in the Type A structured interview compared to children and adolescents who did not have the metabolic syndrome at either examination. Children and adolescents who had the metabolic syndrome at the study entry only scored higher on baseline Aggressive Responding than children and adolescents who did not have the metabolic syndrome at either examination. There were no other significant effects. Covariate analyses indicated that the associations were independent of duration in years between baseline and follow-up, age, ethnicity, gender, and family Hollingshead SES score (ps $< .047$; data not shown).

To determine whether the significant associations were due to a single risk factor, we examined associations between hostility scores and individual risk factors in children who remained free from the metabolic syndrome or developed the metabolic syndrome by the time of the follow-up examination. (Note: Those who had metabolic syndrome at baseline and follow-up or baseline only were not in these analyses.) Cook–Medley Hostility total scores were associated with being classified into the top quartile versus the lower three quartiles of BMI scores and IRI scores (see Figure 1). The association with BMI was primarily due to the Hostile Affect and Aggressive Responding ratings, whereas the association with IRI was marginal with Aggressive Responding ratings (see Figure 2). Associations between Hostile Style and being in the top quartiles of BMI and IRI were significant (see Figure 3). There were no other significant associations.

Table 3
Association Between Hostility Scores at Baseline and the Metabolic Syndrome at Baseline and/or Follow-Up, Compared With No Metabolic Syndrome at Either Baseline or Follow-Up

	Metabolic syndrome at follow-up only		Metabolic syndrome at baseline only		Metabolic syndrome at baseline and follow-up	
Hostility scores at baseline	B[a]	Odds ratio (95% CI)	B[b]	Odds ratio (95% CI)	B[c]	Odds ratio (95% CI)
Cook–Medley Hostility scores						
Total	.20	1.22 (1.06–1.41)	.10	1.11 (0.98–1.24)	.06	1.06 (0.92–1.21)
Aggressive Responding	.35	1.41 (1.07–1.87)	.31	1.36 (1.04–1.78)	.16	1.17 (0.86–1.61)
Hostile Affect	.44	1.55 (1.06–2.27)	.09	1.09 (0.77–1.53)	.16	1.17 (0.78–1.77)
Cynicism	.25	1.29 (1.02–1.64)	.12	1.13 (0.92–1.39)	−.02	.99 (0.79–1.23)
Potential for Hostility ratings						
Total	.35	1.42 (0.78–2.58)	.04	1.04 (0.56–1.94)	.12	1.13 (0.58–2.19)
Content	.40	1.49 (0.81–2.72)	.17	1.19 (0.65–2.17)	.12	1.13 (0.59–2.17)
Intensity	.06	1.07 (0.55–2.07)	−.43	.65 (0.27–1.56)	−.28	.76 (0.32–1.80)
Style	1.02	2.78 (1.54–5.02)	.35	1.42 (0.76–2.66)	.47	1.60 (0.83–3.08)

Note. CI = confidence interval.
[a] n = 18. [b] n = 20. [c] n = 15.

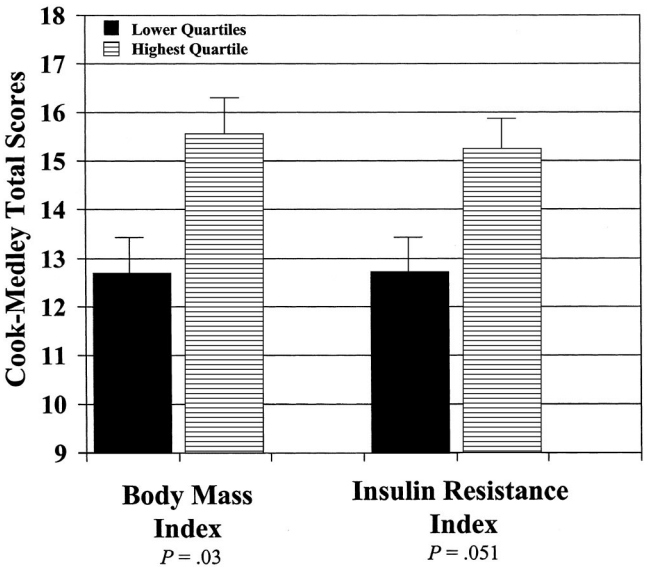

Figure 1. Mean total Cook–Medley Hostility scores in individuals with body mass index and insulin resistance index values in the highest and lower three quartiles of the distribution of scores. Error bars represent standard errors of the means.

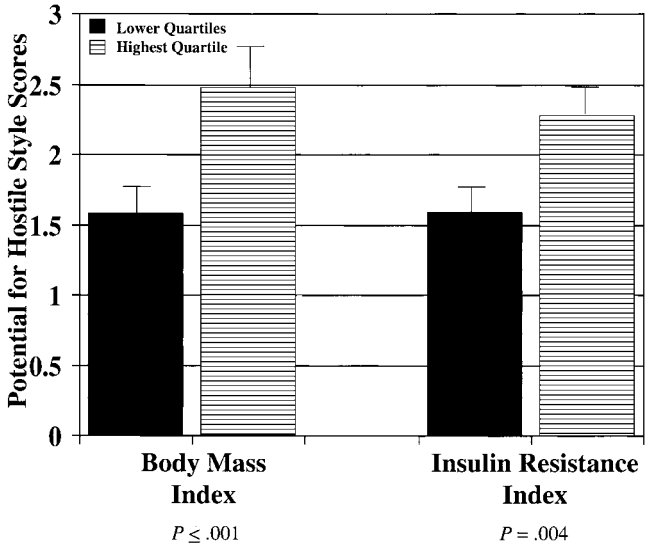

Figure 3. Potential for Hostile Style scores in individuals with body mass index and insulin resistance index values in the highest and lower three quartiles of the distribution of scores. Error bars represent standard errors of the means.

Discussion

Previous studies of adults have established associations between psychological attributes and the clustering of obesity, hyperinsulinemia, dyslipidemia, and elevated BP, the key risk factors comprising the metabolic syndrome. Our factor analyses largely confirmed that such clustering exists in children and adolescents. Following the lead of Chen et al. (2000), we defined the metabolic syndrome as having at least two of the following risk factors above the 75th percentile of the distributions of scores for the same, age, ethnicity, and gender groups: BMI, IRI, ratio of TG to HDL-C, and mean arterial BP. Our results demonstrated that baseline hostility measures predicted those participants who were classified as having the metabolic syndrome at follow-up only versus those who did not have it at either exam.

Results also showed that two risk factors comprising the metabolic syndrome, namely obesity and insulin resistance, were largely responsible for the association between hostility and the metabolic syndrome. Mounting evidence exists showing that insulin resistance may be the primary initiating factor underlying the individual components of the metabolic syndrome (e.g., DeFronzo & Ferrannini, 1991; Ferrannini, Haffner, Mitchell, & Stern, 1991;

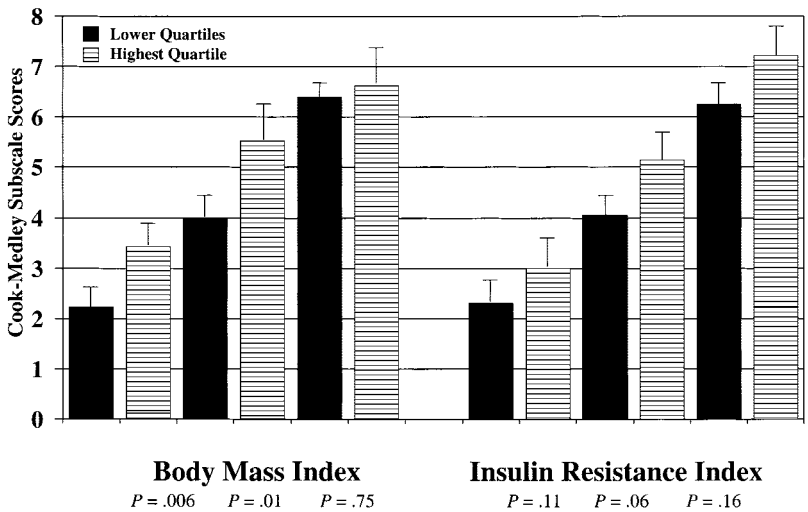

Figure 2. Mean Cook–Medley Hostile Affect, Aggressive Responding, and Cynical Attitudes subscale scores, respectively, in individuals with body mass index and insulin resistance index values in the highest and lower three quartiles of the distribution of scores. Error bars represent standard errors of the means.

Reaven, 1988), even though the strength of the association of insulin resistance with the other components varies between and within populations. Obesity may temporally precede insulin resistance in children, rather than vice versa (Srinivasan, Myers, & Berenson, 1999), and obesity and attendant insulin resistance may account for the clustering of the metabolic syndrome risk variables from childhood to adulthood (Chen et al., 2000). Regardless of their relative significance, the current associations between hostility and BMI or IRI may reflect the central roles played by BMI and IRI in the development of the metabolic syndrome.

It has been postulated that adrenal, gonadal, and growth hormones play a mediating role in the associations between psychological variables and risk factors comprising the metabolic syndrome (Björntorp, 1991; Bouchard, Despres, & Mauriege, 1993). The sexual maturation process is characterized by a complex interplay among various gonadal, adrenal steroid, and growth hormones that rise dramatically during the developmental period from childhood to adulthood. Owing to the maturation process per se, mediation through the endocrine factors remains difficult to determine and awaits future experimental/longitudinal studies. Other proposed mediators include unhealthy lifestyles, such as physical inactivity, dietary factors (Berenson, Srinivasan, & Nicklas, 1998), smoking, and alcohol. Data in adolescents and young adults suggest that hostility is related to the unhealthy lifestyles including smoking and alcohol (Johnson, Hunter, Amos, & Elder, 1989; Räikkönen & Keltikangas-Järvinen, 1991). Finally, Bouchard, Despres, and Mauriege suggested that susceptibility to stress exposure, neuroendocrine responses, and their metabolic, anthropometric, and hemodynamic consequences are influenced by DNA sequence variation at a number of loci. Interestingly, it has been shown that hostile individuals experience their environments as more stressful (e.g., Räikkönen, Matthews, Flory, & Owens, 1999). Moreover, twin and parent–offspring studies suggest a genetic basis for hostility (Carmelli, Swan, & Rosenman, 1990; Matthews, Rosenman, Dembroski, Harris, & MacDougall, 1984). The above proposed pathways are not mutually exclusive, but may rather interact in the development of the metabolic syndrome.

Several factors limit the generalizability of our findings to other populations. First, marked changes occur in the individual risk factors comprising the metabolic syndrome due to aging and sexual maturation. For example, BMI increases slowly (Rosner, Prienas, Loggie, & Daniels, 1998) and children usually experience transient insulin resistance at puberty (Moran et al., 1999). Aging and sexual maturation also influence the risk factor clustering as well. The risk factors cluster at all ages starting from childhood (Arslanian & Suprasongsin, 1996; Berenson, Srinivasan, Bao, et al., 1998; Bergström et al., 1996; Chen et al., 1999a; Csabi et al., 2000). However, clustering may be more substantial during preadolescence and adulthood than during adolescence (Chen et al., 2000). Thus, even though age, gender, and ethnicity were taken into account in examining the metabolic syndrome risk factor clustering within the current sample, the age-related pattern in the clustering restricts the generalizability of our findings to different aged populations.

Second, the observed clustering of risk factors differed significantly from chance, primarily due to the substantially greater number than expected for participants with high levels of four risk factors. Previous studies have shown that adults with two or more risk factors are at increased risk for cardiovascular events (see, e.g., National Cholesterol Education Program, 2001), and studies in children and young adults have shown that the severity of asymptomatic coronary and aortic atherosclerosis increases linearly with the number of zero to four risk factors (Berenson, Srinivasan, Bao, et al., 1998).

Finally, the method of measuring obesity by BMI and insulin resistance by IRI in the current study may be challenged. There exists no clear consensus on how obesity should be measured or classified during childhood and adolescence. Data show that measures of central body fat distribution such as waist to hip ratio or skinfold thickness do not predict unfavorable metabolic profile better than BMI (Bergström et al., 1996). We did not use the euglycemic clamp method to estimate insulin resistance. Nevertheless, the methods of measurement and definition of the metabolic syndrome were comparable to those reported in the Bogalusa Heart Study (Chen et al., 2000), a large prospective epidemiological study on cardiovascular risk in children.

In adults, hostility traits are related to mortality due to cardiovascular and all causes. Ratings of Potential for Hostility have the strongest association with cardiovascular morbidity, whereas the Cook–Medley reports of hostile attitudes toward others have the strongest association with all causes of mortality (Miller et al., 1996). In the current study, hostility was measured with the children and adolescent versions of the same methods. Perhaps the metabolic risk factors' clustering mediates the association between hostility and mortality in adulthood, a possibility that has not been tested because of the treatment of individual risk factors as separate covariates. Consequently, the effect of hostility on metabolic syndrome gives further justification for the evaluation of behavioral risk in young individuals and provides a clear rationale for both prevention and intervention. Interventions designed to reduce hostility, if undertaken early in life, may prevent the development of cardiovascular disease.

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