Parental and offspring associations of the metabolic syndrome in the Fels Longitudinal Study[1–3]

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ABSTRACT

Background: Evidence shows that some causes of the metabolic syndrome (MS) begin in childhood, which could indicate a familial association, through either genetic inheritance or cohabitation.

Objective: This study examined associations between parents and adult offspring diagnoses of the MS and its risk factors.

Design: Measurements were obtained from adult participants and their adult offspring enrolled in the Fels Longitudinal Study, with simultaneous waist circumference, systolic blood pressure (SBP), diastolic blood pressure (DBP), triglycerides, HDL, and glucose observations used for diagnosis. On the basis of repeated measurements (in some cases), adult participants were classified as having the MS at least once or as never having the MS. Chi-square tests, ORs, and mixed-effects models were used to study familial associations.

Results: Maternal (OR: 2.5; 95% CI: 1.1, 5.5) and paternal (OR: 4.1; 95% CI: 1.4, 12.1) MS classifications were significantly associated with MS classification in sons. MS classification in mothers and daughters (OR: 2.7; 95% CI: 0.9, 8.7; $P = 0.08$) was similar to that in sons but was not significant, whereas fathers and daughters were not associated (OR: 1.1; 95% CI: 0.4, 3.5). Maternal MS diagnoses were significantly and positively associated with triglycerides in male offspring and were significantly associated with SBP, DBP, and triglycerides in females. Paternal diagnoses were significantly associated only with DBP and HDL in male offspring.

Conclusions: Parental MS diagnosis is significantly associated with MS diagnosis in adult male offspring, and adverse levels of certain risk factors are associated between offspring and parents, although these associations vary across risk factors and child sex. *Am J Clin Nutr* 2012;96:461–6.

INTRODUCTION

The metabolic syndrome (MS)[4] is diagnosed as a medical condition involving 5 interrelated risk factors: abdominal obesity, hypertension, hypertriglyceridemia, low HDL cholesterol, and impaired fasting plasma glucose. In an analysis of the nationally representative NHANES, Ford et al (1) found that at least one-fifth of adult Americans met the defining criteria for the MS, and the prevalence increases with age, which echoed similar findings elsewhere (2). Other studies have shown that adults with the MS have increased risks for diabetes mellitus (3) and cardiovascular disease (4). As the prevalence of obesity increases, the prevalence of the MS is also expected to increase, because obesity is an independent precursor of MS risk factors (5).

Several studies have shown that the MS and its risk factors track from childhood into adulthood, including waist circumference (WC) (6), blood pressure (BP) (7), HDL cholesterol (8), and triglycerides (5, 9, 10). Some of these studies also found that not only do some of these risk factors track into young adulthood, but the tracking of the overall index of factors was stronger than the individual risk factors (5, 10).

Although in some cases the genesis of the MS appears to begin in childhood, the exact causes remain unclear. One possible vehicle is through familial association, influenced by either genetic inheritance or cohabitation. Several studies have shown that obesity, its biomarkers, and related measures exhibit a degree of parental heritability or influence. In a retrospective study of medical records, Whitaker et al (11) found that parental obesity more than doubles the risk of adult obesity in both obese and nonobese children younger than 10 y. In a longitudinal follow-up study of the role of family history in developing insulin resistance in normoglycemic individuals, Goldfine et al (12) found that those with a family history of diabetes had higher incidence rates of type 2 diabetes than did those with no family history of diabetes.

The primary objective of this study was to characterize the associations between parents and their adult offspring in regard to the MS. The secondary objective was to characterize the associations between parents and adult offspring with regard to specific risk factors of the MS. The study of both objectives will incorporate participant data from the Fels Longitudinal Study (FLS), which affords long-term serial measurements over periods of several decades.

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[4] Abbreviations used: BP, blood pressure; DBP, diastolic blood pressure; FLS, Fels Longitudinal Study; MS, metabolic syndrome; SBP, systolic blood pressure; WC, waist circumference.

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SUBJECTS AND METHODS

Study sample

Subjects were drawn from the FLS. FLS participants were generally enrolled at birth and were not selected with regard to factors known to be associated with disease, body composition, or other clinical conditions (13). Participants of this study included 395 mothers and their 790 children (387 boys and 403 girls) and 323 fathers and their 675 children (337 boys and 338 girls). Measurements were incorporated for parents aged ≥18 y, and their offspring aged 18–35 y, as of 30 June 2009. FLS participants ≥18 y of age were measured at 2-y intervals, and, in most cases, subjects were measured on more than one occasion. Measurements were excluded from the analysis if the subject was pregnant or injured when the measurements were taken. All participants signed an informed consent form, and the Institutional Review Boards at Wright State University and Virginia Commonwealth University approved all procedures.

Risk factor measurement and metabolic syndrome determination

WC (cm) was measured twice at the suprailiac crest, and the average value was reported. Systolic BP (SBP; mm Hg) and diastolic BP (DBP; mm Hg) were recorded as the average of 2 readings taken with a manual sphygmomanometer from subjects seated in an upright position. Comprehensive lists of current medications were examined to classify subjects on the basis of BP medication use (determination of yes or no). Fasting venous blood samples were collected from participants for the measurement of glucose (mg/dL), triglycerides (mg/dL), and HDL cholesterol (mg/dL).

The defining criteria for the 5 MS risk factors were based on the MS working definition established in the Third Report of the National Cholesterol Education Program Adult Treatment Panel III on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (14, 15). These criteria were as follows: WC >102 cm for men and >88 cm for women, SBP >130 mm Hg and/or DBP >85 mm Hg and/or participants on BP medication, fasting plasma triglyceride concentration >150 mg/dL, fasting plasma HDL-cholesterol concentration <40 mg/dL for men and <50 mg/dL for women, and fasting plasma glucose concentration >100 mg/dL.

Inclusion and exclusion criteria

A diagnosis of the MS was made if the subject met ≥3 defining criteria for the 5 risk factors. Subjects were categorized as not having the MS if they met ≤2 of the defining criteria. Subject data were excluded from the primary analysis if measurements on at least 3 of the 5 risk factors were missing, because MS status could then not be determined. Subjects missing data on 2 of the 5 risk factors were included in the study if the defining criteria for 3 of the measured risk factors were either all met (subject categorized as having the MS) or all not met (subject categorized as not having the MS); otherwise, the data for these subjects were excluded from the primary analysis because their MS status was indeterminate. Furthermore, if subjects were missing only one risk factor measurement, they were included in the study if ≥3 of 4 measured risk factors would give a diagnosis of having or not having the MS; these subjects were excluded from the primary analysis if they met only 2 defining criteria for the risk factors because their MS status was indeterminate. Subjects were included in the secondary analyses if they had measurements for any specific risk factor.

Statistical methods

Parental and child MS statuses were summarized by frequencies and proportions, whereby subjects were classified as either having the MS at some or all visits or not having the MS at any visit. Measurements for MS risk factors were left in their original numeric state. Chi-square tests were used to examine the association between offspring and parental MS. Corresponding to each test, ORs and 95% CIs were also estimated. Relations between parental MS categorization and the risk factor measurements in offspring were assessed by using a repeated-measure, mixed-effects ANOVA, in which parent MS classification, subject age, and sex were included as fixed effects and including a random subject effect to account for a within-subject association, which is modeled by using an autoregressive

TABLE 1
Distribution of parental and offspring MS[1]

	Son	Daughter
Maternal MS category		
MS		
Frequency: 94 (53%)		
MS [*n* (%)]	26 (26)	13 (12)
No [*n* (%)]	74 (74)	93 (88)
Total (*n*)	100	106
No		
Frequency: 85 (47%)		
MS [*n* (%)]	10 (12)	4 (5)
No [*n* (%)]	71 (88)	78 (95)
Total (*n*)	81	82
Total		
Frequency: 179		
OR (95% CI)	2.5 (1.1, 5.5)	2.7 (0.9, 8.7)
Chi-square[2] (*P*)	5.2 (<0.01)	3.1 (0.08)
Paternal MS category		
MS		
Frequency: 70 (53%)		
MS [*n* (%)]	16 (24)	8 (10)
No [*n* (%)]	51 (76)	70 (90)
Total (*n*)	67	78
No		
Frequency: 62 (47%)		
MS [*n* (%)]	5 (7)	6 (9)
No [*n* (%)]	66 (93)	60 (91)
Total (*n*)	71	66
Total		
Frequency: 132		
OR (95% CI)	4.1 (1.4, 12.1)	1.1 (0.4, 3.5)
Chi-square[2] (*P*)	7.6 (<0.01)	0.1 (0.81)

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS categorized as MS (MS at all measurements) or No (no MS at any measurement). Chi-square tests were used to examine the association between offspring and parental MS. MS, metabolic syndrome.

[2] 1 df.

TABLE 2
Data summary for waist circumference of offspring[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (*n* = 150)	100.1 ± 2.12	95.8, 104.3	
No (*n* = 49)	98.3 ± 2.50	93.4, 103.3	
Difference (MS − No)	1.7 ± 3.29	−4.8, 8.2	0.60
Daughters			
MS (*n* = 159)	96.5 ± 2.26	92.0, 101.0	
No (*n* = 53)	89.4 ± 3.11	83.2, 95.6	
Difference (MS − No)	7.0 ± 3.85	−0.6, 14.7	0.07
Paternal MS category			
Sons			
MS (*n* = 82)	100.3 ± 2.11	96.1, 104.5	
No (*n* = 71)	95.0 ± 2.41	90.2, 100.0	
Difference (MS − No)	5.3 ± 3.21	−1.1, 11.7	0.10
Daughters			
MS (*n* = 105)	92.0 ± 2.26	87.4, 96.5	
No (*n* = 55)	87.9 ± 4.22	79.4, 96.4	
Difference (MS − No)	4.0 ± 4.79	−5.6, 13.7	0.40

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean waist circumference based on parent MS classification. MS, metabolic syndrome.

covariance structure. Corresponding to each test, means, SEs, and 95% CIs were also reported. Analyses were conducted separately for mothers and fathers and for sons and daughters. A significance level of $\alpha = 0.05$ was used for all 2-sided tests of no association, and all data summarizations and analyses were performed by using SAS version 9.2.

RESULTS

The mean (±SD) age of the participating mothers who provided at least one MS risk factor measurement was 59.1 ± 11.38 (95% CI: 57.2, 61.0) y, the mean age of their sons was 27.9 ± 5.80 (95% CI: 26.9, 28.9) y, and the mean age of their daughters was 28.5 ± 5.83 (95% CI: 27.5, 29.5) y. Similar values were found for fathers, who had a mean (±SD) age of 59.9 ± 11.4 (95% CI: 57.7, 62.1) y; the mean age of their sons was 27.8 ± 5.77 (95% CI: 26.7, 28.9) y, and the mean age of their daughters was 28.6 ± 5.83 (95% CI: 27.5, 29.7) y. Note that not all of the subjects provided measurements for each MS risk factor, and, because of our exclusion criteria, the number of subjects available for each analysis was at times less than the total number available.

MS

Summary statistics for parental and offspring MS are listed in **Table 1**. A significant association was found between sons and mothers ($P < 0.01$), whereby sons whose mothers had a diagnosis of the MS were 2.5 times as likely to develop the MS than were sons whose mothers had no diagnosis of the MS. A similar pattern was observed for daughters (OR: 2.7; 95% CI: 0.9, 8.7), but the relation was not significant ($P = 0.08$). A significant association was also found between sons and fathers ($P < 0.01$): sons whose fathers had a diagnosis of the MS were 4.1 times as likely to develop the MS than were sons whose fathers had no diagnosis of the MS. No significant relation was found between daughters and fathers ($P = 0.81$).

WC

Summary statistics for parental and offspring WC measurements are listed in **Table 2**. No significant difference in mean WC ($P = 0.60$) was found between sons from mothers categorized as having MS and those with mothers categorized as never having MS. Mean WC in daughters from mothers categorized as having the MS was not significantly different ($P = 0.07$) from that in daughters from mothers categorized as never having the MS. Similarly, mean WC from both sons and daughters from fathers categorized as having the MS did not have significantly different ($P > 0.10$) mean WCs than did sons and daughters from fathers categorized as never having the MS.

SBP

Summary statistics for parental and offspring SBP measurements are listed in **Table 3**. Whereas sons from mothers categorized as having the MS or never having the MS had similar mean SBP ($P = 0.95$), daughters from mothers categorized as having the MS (mean ± SE: 113.3 ± 2.10 mm Hg) had a significantly larger mean SBP than did daughters from mothers classified as never having the MS (101.2 ± 2.79 mm Hg; difference: 12.1 ± 3.50 mm Hg; $P < 0.01$). Neither sons nor daughters had significant differences on the basis of their father's MS classification ($P > 0.56$).

DBP

Summary statistics for parental and offspring DBP measurements are listed in **Table 4**. Whereas sons did not have

TABLE 3
Data summary for systolic blood pressure of offspring[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (*n* = 173)	119.4 ± 2.52	115.3, 123.2	
No (*n* = 213)	119.2 ± 2.00	114.4, 124.5	
Difference (MS − No)	−0.2 ± 3.22	−6.6, 6.2	0.95
Daughters			
MS (*n* = 193)	113.3 ± 2.10	109.1, 117.5	
No (*n* = 207)	101.2 ± 2.79	95.6, 106.7	
Difference (MS − No)	12.1 ± 3.50	5.2, 19.1	<0.01
Paternal MS category			
Sons			
MS (*n* = 224)	118.9 ± 2.40	114.1, 123.7	
No (*n* = 113)	116.8 ± 2.70	111.4, 122.1	
Difference (MS − No)	2.1 ± 3.61	−5.1, 9.3	0.56
Daughters			
MS (*n* = 216)	108.7 ± 2.12	104.5, 113.0	
No (*n* = 122)	108.5 ± 3.57	101.4, 115.7	
Difference (MS − No)	0.2 ± 4.15	−8.1, 8.5	0.96

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean systolic blood pressure based on parent MS classification. MS, metabolic syndrome.

TABLE 4
Data summary for offspring diastolic blood pressure[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (n = 111)	81.3 ± 1.87	77.6, 85.0	
No (n = 276)	75.8 ± 2.38	71.1, 80.5	
Difference (MS − No)	5.5 ± 3.02	−0.5, 11.5	0.07
Daughters			
MS (n = 114)	76.4 ± 2.08	72.3, 80.5	
No (n = 289)	67.8 ± 2.75	62.4, 73.3	
Difference (MS − No)	8.6 ± 3.44	1.7, 15.4	0.01
Paternal MS category			
Sons			
MS (n = 163)	83.4 ± 2.21	79.0, 87.8	
No (n = 174)	74.5 ± 2.48	69.6, 79.4	
Difference (MS − No)	8.9 ± 3.32	2.3, 15.5	< 0.01
Daughters			
MS (n = 156)	71.9 ± 2.15	67.6, 76.3	
No (n = 182)	67.9 ± 3.72	60.4, 75.3	
Difference (MS − No)	4.1 ± 4.30	−4.5, 12.7	0.35

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean diastolic blood pressure based on parent MS classification. MS, metabolic syndrome.

significantly different mean DBPs based on maternal MS classification (P = 0.07), daughters from mothers classified as having the MS had a larger mean (±SE) DBP (76.4 ± 2.08 mm Hg) than did daughters from mothers never classified as having the MS (67.8 ± 2.75 mm Hg), with the difference being significant (difference: 8.6 ± 3.44 mm Hg; P = 0.01). Conversely, sons with fathers categorized as having the MS had a significantly larger ($P < 0.01$) mean DBP (83.4 ± 2.21 mm Hg) than did sons with fathers categorized as never having the MS (74.5 ± 2.48 mm Hg), whereas no significant difference was found between daughters (P = 0.35) on the basis of their paternal MS classification.

Triglycerides

Summary statistics for parental and offspring triglyceride measurements are listed in **Table 5**. Sons with mothers classified as having the MS had significantly greater mean (±SE) triglyceride concentrations than did sons with mothers classified as never having the MS (difference: 117.0 ± 45.19 mg/dL; P = 0.01). Although daughters had a similar pattern, the mean triglyceride difference was smaller (difference = 39.8 ± 20.11 mg/dL) and was not significant (P = 0.05). No significant difference in mean triglyceride concentrations was found in sons based on paternal MS classification (P = 0.33), in part because of large variability in the measurements. Daughters also had no significant difference (P = 0.78) based on paternal MS classification.

HDL

Summary statistics for parental and offspring HDL measurements are listed in **Table 6**. Neither sons nor daughters had significantly different mean HDL concentrations based on the mothers' MS classification ($P > 0.12$). The mean (±SE) HDL concentration was significantly lower (P = 0.04) in sons with fathers classified as having the MS (44.4 ± 2.39 mg/dL) than in sons with fathers classified as never having the MS (51.7 ± 2.74 mg/dL), whereas daughters did not have significantly different mean HDL concentrations on the basis of paternal MS classification (P = 0.90).

Glucose

Summary statistics for paternal and offspring glucose measurements are listed in **Table 7**. Sons had similar mean glucose concentrations regardless of maternal or paternal MS classification, with neither difference being significant ($P > 0.35$). The mean glucose concentration for daughters with mothers classified as having the MS was not significantly different from that of daughters with mothers classified as never having the MS (P = 0.43). Daughters had similar mean glucose concentrations on the basis of paternal MS classification (P = 0.62).

DISCUSSION

With the use of data from subjects in the FLS, we found significant familial associations for diagnosis of the MS between mothers and fathers and their sons; a diagnosis of the MS was more likely in a son when either parent met the diagnostic criteria for the MS at their biennial examinations. A similar but weaker relation was found between mothers and their daughters. Maternal MS categorization was significantly associated with concentrations of HDL and triglycerides in male offspring and was significantly associated with SBP, DBP, and triglycerides in female offspring. Paternal MS categorizations were significantly

TABLE 5
Data summary for offspring triglycerides[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (n = 101)	228.2 ± 27.90	172.8, 283.6	
No (n = 82)	111.2 ± 35.55	40.6, 181.9	
Difference (MS − No)	117.0 ± 45.19	27.2, 206.7	0.01
Daughters			
MS (n = 114)	131.6 ± 11.82	108.0, 155.1	
No (n = 79)	91.8 ± 16.26	59.4, 124.2	
Difference (MS − No)	39.8 ± 20.11	−0.2, 79.9	0.05
Paternal MS category			
Sons			
MS (n = 80)	222.3 ± 47.43	127.8, 316.8	
No (n = 61)	152.5 ± 54.02	44.8, 260.1	
Difference (MS − No)	69.8 ± 71.88	−73.4, 213.0	0.33
Daughters			
MS (n = 77)	109.9 ± 14.39	53.2, 150.4	
No (n = 69)	101.8 ± 24.18	53.2, 150.4	
Difference (MS − No)	8.0 ± 28.13	−48.5, 64.6	0.78

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean triglycerides based on parent MS classification. MS, metabolic syndrome.

TABLE 6
Data summary for offspring HDL[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (*n* = 97)	45.0 ± 1.93	41.1, 48.8	
No (*n* = 86)	49.9 ± 2.47	45.0, 54.8	
Difference (MS − No)	−4.9 ± 3.13	−11.2, 1.3	0.12
Daughters			
MS (*n* = 101)	53.6 ± 2.71	48.2, 59.0	
No (*n* = 92)	57.0 ± 3.73	49.5, 64.4	
Difference (MS − No)	−3.3 ± 4.61	−12.5, 5.8	0.47
Paternal MS category			
Sons			
MS (*n* = 58)	44.4 ± 2.39	39.6, 49.1	
No (*n* = 83)	51.7 ± 2.74	26.2, 57.2	
Difference (MS − No)	−7.3 ± 3.64	−14.6, −0.1	0.04
Daughters			
MS (*n* = 73)	53.6 ± 3.00	47.6, 59.6	
No (*n* = 73)	54.3 ± 5.06	44.2, 64.5	
Difference (MS − No)	−0.8 ± 5.88	−12.6, 11.1	0.90

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean HDL based on parent MS classification. MS, metabolic syndrome.

associated with DBP and HDL in male offspring and were not significantly associated with any risk factors in females.

Although we are unaware of any studies measuring associations between parents and children with respect to the MS, other studies have focused on heritability. Poulsen et al (16) compared associations between both monozygotic and dizygotic twins and found that HDL had a strong genetic component, whereas the WC-to-hip ratio and triglycerides did not. These results are somewhat concordant with our results, because maternal MS significantly affected mean HDL concentrations in boys (but not in girls), and no familial associations or effects were found for WC. However, we did find a maternal MS effect on triglycerides in both sons and daughters. The twin study found a stronger genetic factor with respect to SBP in boys than in girls (our results showed that the mean SBP in daughters was associated with maternal MS), and, although it reported a stronger genetic factor for HDL in girls than in boys, we found the effects to be similar in sons and daughters with respect to maternal MS.

Although we did not formally test for differences in MS prevalence or risk factor levels between sons and daughters, some results merit discussion. Sons were more likely than daughters to meet the diagnostic criteria for the MS when either mothers or fathers had a diagnosis of the MS (as seen in Table 1). This increased prevalence in sons may be part of the reason that the MS was significantly associated between sons and both mothers and fathers, but not between daughters and either parent. Both maternal and paternal MS status affected mean HDL concentrations in sons but not in daughters. Maternal MS classification had a significant effect on BP (both SBP and DBP) in daughters but not in sons. Similarly, paternal MS classification had a significant effect on DBP in sons but not in daughters. Our findings of some sexual dimorphisms contrast with results found elsewhere. By using data from NHANES, both Ford et al (1) and Park et al (2) reported that men were more likely than women to meet the criteria for hypertriglyceridemia, hypertension, and hyperglycemia, whereas women were more likely than men to meet the criteria for abdominal obesity and low HDL. Although the results for the MS match what we would expect from previous studies (males are more likely to fit more MS risk factor criteria than females), the differences between our risk factor results and those appearing elsewhere may stem from our choice to keep the measurements in their numerical form, without consideration of whether those values are at risk of MS classification.

Because our focus was on the familial association of the MS and its risk factors, we must mention that the use of the MS is not without controversy. In fact, several researchers have suggested the discontinued use of the MS for both research and clinical purposes (17, 18). Because many of the criticisms are valid (eg, omission of established risk factors, heterogeneity of MS diagnoses, dichotomization of risk factors; 18), we make no counter argument, although we note that each MS risk factor was kept in its original numeric format in our secondary analyses. Rather, we note that, on the basis of our findings, the associations between parents and their sons with respect to the MS was clear, whereas the effects of parental MS categorization were not as clearly represented by each of the risk factors. This runs counter to the finding of Kahn et al (19), who stated that the MS is no greater than the sum of its parts.

One limitation of our study was that we did not account for dependence between siblings. Although there is dependence between parents and their children, preliminary findings (data not shown) indicated that dependence between siblings is small and should not affect our results. In addition, our study did not account for possible confounding by adult attributes, such as diet and lifestyle characteristics. However, although the FLS database

TABLE 7
Data summary for offspring glucose[1]

	Mean ± SE	95% CI	*P*
Maternal MS category			
Sons			
MS (*n* = 65)	92.2 ± 2.12	88.0, 96.5	
No (*n* = 85)	94.2 ± 2.72	88.8, 99.7	
Difference (MS − No)	−2.0 ± 3.45	−8.9, 4.9	0.56
Daughters			
MS (*n* = 71)	87.7 ± 7.22	73.3, 102.2	
No (*n* = 86)	77.0 ± 8.75	59.5, 94.5	
Difference (MS − No)	10.7 ± 11.35	−12.0, 33.4	0.35
Paternal MS category			
Sons			
MS (*n* = 57)	95.6 ± 2.67	90.3, 101.0	
No (*n* = 43)	92.5 ± 2.80	86.9, 98.1	
Difference (MS − No)	3.1 ± 3.88	−4.6, 10.9	0.43
Daughters			
MS (*n* = 79)	85.4 ± 3.49	78.3, 92.4	
No (*n* = 37)	88.5 ± 5.42	77.6, 99.5	
Difference (MS − No)	−3.1 ± 6.45	−16.2, 9.9	0.62

[1] Based on repeated maternal and paternal measurements from ages ≥18 y and repeated child measurements between ages 18 and 35 y. MS is categorized as MS (MS at all measurements) or No (no MS at any measurement). A repeated-measure, mixed-effects ANOVA was used to test for differences in mean glucose based on parent MS classification. MS, metabolic syndrome.

contained these measurements for some participants, enough data were not available for analysis. Furthermore, the association between parents and their offspring may be greatest when the children are young, although the lack of a uniform definition for the MS in childhood would complicate this type of study.

Another limitation was the inability to generalize these results to nonwhite subjects, because this subset of the FLS data contains no measurements on subjects belonging to races other than white. Evidence of racial disparities in MS status has been noted elsewhere, as Ford et al found racial and sex differences between Mexican Americans, African Americans, and whites (1).

The likelihood that certain risk factors are triggered together has been observed elsewhere (20). We plan to expand on this research, by estimating the familial associations across risk factors, to determine to what extent the relations between risk factors can be explained by family history. As mentioned earlier, relations found in this article can also be adjusted for adult lifestyle characteristics such as nutritional habits, alcohol and smoking behavior, level of physical activity, and occupation.

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