The Prevalence of Metabolic Syndrome Among US Women of Childbearing Age

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Objectives. We sought to determine whether the prevalence of metabolic syndrome among US women of childbearing age (18–44 years) has increased since 1988 and to estimate its current prevalence by race/ethnicity and risk that a maternal history of select metabolic syndrome characteristics imposes on offspring.

Methods. We used survey-specific data analysis methods to examine data from the National Health and Nutrition Examination Surveys conducted from 1988 to 2004.

Results. The prevalence of the metabolic syndrome phenotype and 2 of its clinical correlates significantly increased between 1988 and 2004 (increase for metabolic syndrome phenotype = 7.6%, for obesity = 13.3%, and for elevated C-reactive protein = 10.6%; $P < .001$ for all 3). Hispanic women were more likely than were White women to possess the phenotype ($P = .004$). Women who reported that their mothers had been diagnosed with diabetes were more likely to possess the phenotype than those whose mothers had not been so diagnosed (odds ratio = 1.9; 95% confidence interval = 1.3, 2.8).

Conclusions. The current trends of metabolic syndrome among women of childbearing age demonstrate the need for additional rigorous investigations regarding its long-term effects in these women and their offspring. (*Am J Public Health.* 2008;98:1122–1127. doi:10.2105/AJPH.2007.120055)

The third report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults defined metabolic syndrome as the "clustering of metabolic and cardiovascular disease (CVD) risk factors that independently increase the risk of Type 2 diabetes and endpoints of CVD (e.g., stroke, congestive heart failure, death)."[1] Guidelines for diagnosis and management of the metabolic syndrome phenotype[2] have identified the following as clinical components of metabolic syndrome: abdominal obesity, atherogenic dyslipidemia, hypertension, impaired fasting glucose, and a pro-inflammatory state.

The prevalence of metabolic syndrome among US adults is reported to be 23.7%,[3] with an additional 30% of obese adults at risk for developing it.[4] For some racial/ethnic subgroups, the prevalence of metabolic syndrome is higher than the national average (25.7% among Blacks and 35.6% among Hispanics).[5–7] The prevalence of metabolic syndrome is reported to be similar for males and females,[8] although higher morbidity among females from metabolic syndrome has been reported.[9] Studies examining the prevalence of metabolic syndrome and its clinical correlates among women of childbearing age as a separate group, however, are lacking, in spite of its obvious importance.

It is known from previous studies that exposure to a nutrient-restrictive intrauterine environment appears to reprogram the metabolism of the developing fetus, resulting in an altered phenotype during childhood or adult life (e.g., obesity, insulin resistance, increased risk for CVD).[10–12] A number of studies have also established an association between individual metabolic pathologies (e.g., gestational diabetes) in the mother and pregnancy or birth outcomes.[5,13] Other studies have identified critical periods of in utero exposure to maternal metabolic pathologies (including intrauterine nutrition restriction) that increase the risk of impaired glucose metabolism in the offspring.[14,15]

Less is known, however, about the effects of intrauterine exposure to the "cluster" of the components of metabolic syndrome, the critical periods of fetal exposure (i.e., first trimester vs third trimester), and whether the fetus's exposure to some factors of metabolic syndrome is more harmful than its exposure to others. Thus, studies examining the prevalence of metabolic syndrome phenotype women during the childbearing years (particularly during the preconception period) are urgently needed. If an association exists between intrauterine exposure and chronic disease later in life, the impact of this emerging public health issue and its implication for health disparities has yet to be realized.

We investigated the change in prevalence of metabolic syndrome and its clinical components among US women aged 18 to 44 years since 1988. We also examined how the current prevalence of metabolic syndrome among Black and Hispanic women compared with that of non-Hispanic White women in this age group. Finally, we sought to estimate the risk for metabolic syndrome conferred from maternal disease history, specifically diabetes and cardiovascular disease. To the best of our knowledge, our study is the first to examine metabolic syndrome among women of childbearing age as a separate group.

METHODS

Data Source and Population Selection

Data were obtained from the National Health and Nutrition Examination Survey (NHANES) conducted from 1988 to 2004. The NHANES, conducted by the Centers for Disease Control and Prevention's National Center for Health Statistics, is a nationally representative sample of the civilian, noninstitutionalized US population that uses complex sampling methods to adjust for variation in sampling probability and the distribution of characteristics. Further details regarding the surveys may be found elsewhere.[16,17]

For this study, "women of childbearing age" were defined as those aged between 18 and 44 years at the time of their participation in the NHANES and who reported being

non-Hispanic White, non-Hispanic Black, or Hispanic. Because we were interested in characterizing the prevalence of metabolic syndrome within an otherwise healthy population, we excluded women who reported having been diagnosed with diabetes. We also excluded those who were pregnant at the time of their participation in NHANES and those who reported fasting for less than 8 hours before blood collection during the laboratory assessment phase of the survey.

Definition of Metabolic Syndrome

We used the National Cholesterol Education Program's and the International Diabetes Federation's definition of the metabolic syndrome phenotype as the clustering of any 3 or more of the following independent risk factors for CVD[2,18]:

- Obesity, defined as either body mass index (BMI) greater than 30 kg/m^2 or waist circumference greater than 88 cm (approximately 35 in);
- Elevated fasting glucose, defined as fasting plasma glucose above 100 mg/dL;
- Hypertension, defined as a blood pressure reading higher than 130/85 mm Hg;
- Serum triglyceride levels above 150 mg/dL;
- High-density lipoprotein cholesterol levels below 50 mg/dL.

On the basis of metabolic syndrome clinical guidelines published by the National Institutes of Health and the American Heart Association,[19] we also included a sixth criterion: the presence of a biomarker indicative of a pro-inflammatory state (i.e., C-reactive protein levels>3.0 mg/L).

Because the clinical characterization of metabolic syndrome includes 2 separate definitions of obesity, we adjusted for the 25% of participants who satisfied both definitions to avoid overestimating those possessing the phenotype. Thus, the presence of obesity was counted only once irrespective of whether the participant satisfied 1 or both definitions of obesity.

Statistical Analysis

To account for the complex sampling design, we used survey-specific proportion and logistic regression analysis. Sampling weights for the fasting subsample were used in the calculation of all estimates. Because we combined the 3 NHANES data sets covering the period 1999 to 2004, we calculated 6-year weights by the guidelines suggested by the National Center for Health Statistics for analyses that combine 2 or more cycles of NHANES data.[20]

Data presented in Table 1 show the change in the proportion of women with metabolic syndrome between NHANES 1988 to 1994 (NHANES III) and NHANES 1999 to 2004 and the statistical significance of the change. For the former, we used Stata software command svyprop (StataCorp LP, College Station, Texas) for the latter, we used Stata software command lincom (*variable*)2 lincom (*variable*)1. To obtain estimates in Table 2, we used only data from NHANES 1999 to 2004. In these analyses, we examined the difference between the subgroup of interest (i.e., non-Hispanic Blacks or Hispanics) and the non-Hispanic White population—using Stata software command svyprop—and the statistical significance of the difference—using Stata software command lincom (*variable*)2 − lincom (*variable*)1. To obtain the risk estimates in Table 3, we used only data from NHANES 1999–2004. We calculated the estimates using the Stata software logistic regression command svymlogit, and we controlled for age and race/ethnicity. Positive maternal disease history was coded 1; no maternal disease history was coded 0.

TABLE 1—Prevalence of Metabolic Syndrome and Individual Clinical Components Among Women of Childbearing Age: National Health and Nutrition Examination Surveys, 1988–1994 and 1999–2004

	NHANES 1988–1994				NHANES 1999–2004			
	Total (n=3391)	Non-Hispanic Whites	Non-Hispanic Blacks	Hispanics	Total (n=2027)	Non-Hispanic Whites	Non-Hispanic Blacks	Hispanics
No. of metabolic syndrome components, %								
≥1	63.0***	60.0**	72.8**	77.8	72.8	70.1	81.1	79.6
≥2	37.8***	34.5**	46.4	52.1*	47.8	44.7	52.3	60.1
≥3	17.8***	16.3***	22.3**	30.2	26.5	24.4	29.0	36.0
Obesity, %								
Waist circumference>88 cm	32.8***	29.3***	46.3***	47.7	461	43.5	61.6	53.8
BMI>30 kg/m^2, %	20.0***	17.6***	30.3***	28.3	28.3	25.3	46.9	31.6
Elevated fasting glucose, %	10.2	6.7***	9.9	11.4	12.0	12.2	12.7	15.1
Hypertension (blood pressure >130/85 mm Hg), %	3.0	2.5	7.1	2.2	3.2	2.6	8.0	2.5
Elevated triglycerides (>150 mg/dL), %	15.4	15.4**	10.0	27.4	19.3	20.4	10.4	23.6
Low serum HDL (<50 mg/dL), %	39.8	37.8	35.4	45.6	42.7	38.8	37.9	47.3
Pro-inflammatory state (CRP>3.0 mg/L), %	27.4***	24.2***	39.6	37.6**	38.0	37.4	41.4	44.8

Note. BMI = body mass index; HDL = high-density lipoprotein cholesterol; CRP = C-reactive protein. The metabolic syndrome phenotype was defined as the presence of 3 or more clinical components. Childbearing age was defined as 18 to 44 years. All components of metabolic syndrome are from a joint statement of the National Institutes of Health and the American Heart Association.
*$P \leq .05$; **$P \leq .005$; ***$P \leq .001$ (for significance of comparison with non-Hispanic White women).

RESULTS

Change in Prevalence of Metabolic Syndrome and Its Clinical Components

To determine whether there had been a change in the prevalence of metabolic syndrome and its clinical correlates among women of childbearing age, we conducted a comparative analysis of NHANES data collected from 1988–1994 and from 1999–2004. We show in Table 1 that an increase of 8.7 percentage points (from 17.8% to 26.5%; $P<.001$) occurred over the study period in the proportion of women possessing the metabolic syndrome phenotype.

TABLE 2—Prevalence of Metabolic Syndrome and Its Clinical Components Among Women of Childbearing Age, by Race/Ethnicity: National Health and Nutrition Examination Survey, 1999–2004

	Non-Hispanic Whites	Non-Hispanic Blacks	Hispanics
Total, No.	898	516	613
No. of metabolic syndrome components			
≥1	71.0	80.5*	80.4*
≥2	45.0	52.8*	61.0**
≥3	24.6	29.3	36.4**
Obesity, %			
Waist circumference > 88 cm	42.1	60.5***	56.0***
BMI > 30 kg/m^2, %	24.2	47.5***	32.2*
Elevated fasting glucose, %	11.9	12.2	14.6
Hypertension (blood pressure > 130/85 mm Hg), %	2.4	7.4*	2.6
Elevated triglycerides (>150 mg/dL), %	20.7	10.7	23.8
Low serum HDL (<50 mg/dL), %	41.4	42.7	53.9**
Pro-inflammatory state (CRP > 3.0 mg/L), %	36.6	43.3*	45.3*

Note. BMI = body mass index; HDL = high-density lipoprotein cholesterol; CRP = C-reactive protein. The metabolic syndrome phenotype was defined as the presence of 3 or more clinical components. Childbearing age was defined as 18 to 44 years. All components of metabolic syndrome are from a joint statement of the National Institutes of Health and the American Heart Association.
*$P \leq .05$; **$P \leq .005$; ***$P \leq .001$ (for significance of comparison with non-Hispanic White women).

TABLE 3—Effect of Mother's Medical History on the Risk of Metabolic Syndrome Phenotype Among Women of Childbearing Age: National Health and Nutrition Examination Survey, 1999–2004

	Maternal Diabetes (n = 345), OR (95% CI)	Maternal Hypertension (n = 407), OR (95% CI)	Maternal Heart Attack (n = 60), OR (95% CI)
No. of metabolic syndrome components, %			
≥1	2.1 (1.2, 3.8)	1.6 (1.0, 2.6)	0.7 (0.2, 2.0)
≥2	2.3 (1.4, 3.6)	1.4 (0.9, 2.2)	1.5 (0.5, 4.0)
≥3		1.5 (0.8, 2.5)	2.0 (0.8, 4.5)
Obesity, %			
Waist circumference > 88 cm	2.1 (1.5, 3.0)	1.4 (1.1, 1.8)	1.3 (0.5, 2.8)
BMI > 30 kg/m^2, %	1.7 (1.3, 2.3)	1.7 (1.4, 2.1)	1.2 (0.6, 2.3)
Elevated fasting glucose, %	1.9 (1.1, 3.2)	0.8 (0.5, 1.4)	0.2 (0.05, 1.2)
Hypertension (blood pressure > 130/85 mm Hg), %	1.4 (0.7, 3.0)	2.3 (1.3, 4.0)	1.5 (0.3, 7.1)
Elevated triglycerides (>150 mg/dL), %	1.1 (0.7, 1.8)	1.1 (0.7, 2.0)	1.0 (0.3, 3.1)
Low serum HDL (<50 mg/dL), %	1.6 (1.2, 2.1)	1.3 (1.0, 1.8)	1.0 (0.5, 2.1)
Pro-inflammatory state (CRP > 3.0 mg/L), %	1.7 (1.3, 2.2)	1.5 (1.1, 2.0)	1.4 (0.8, 2.6)

Note. OR = odds ratio; CI = confidence interval; BMI = body mass index; HDL = high-density lipoprotein cholesterol; CRP = C-reactive protein. ORs are adjusted for age and race/ethnicity; $P = .05$. The metabolic syndrome phenotype was defined as the presence of 3 or more clinical components. Childbearing age was defined as 18 to 44 years. All components of metabolic syndrome are from a joint statement of the National Institutes of Health and the American Heart Association.

Furthermore, we observed an increase of 10 percentage points ($P<.001$) in the number of women who would be considered at risk for developing the metabolic syndrome phenotype because they possessed at least 2 of the metabolic syndrome clinical components. We also observed a significant increase ($P<.001$) in the proportion who were obese, whether defined by waist circumference (increase of 13.3 percentage points) or by BMI (increase of 8.3 percentage points), and an increase of 10.6 percentage points ($P<.001$) in the number with clinical evidence of a pro-inflammatory state as defined by elevated serum C-reactive protein (i.e., >3.0 mg/L).

Regarding racial/ethnic disparities, we observed the greatest change over the time period among non-Hispanic Whites. Significant increases were observed for this group in the proportion possessing the metabolic syndrome phenotype, as well as those who were obese (irrespective of definition). Furthermore, among non-Hispanic Whites, we observed an increased prevalence of impaired glucose metabolism, elevated triglycerides, or evidence of a proinflammatory state. With respect to disparities, the relative prevalence of the metabolic syndrome phenotype among non-Hispanic Blacks and Hispanics over the time period was higher than among non-Hispanic Whites. Although we observed an increase over the time period in the total proportion of non-Hispanic Black and Hispanic women with impaired glucose metabolism (1.8%), hypertension (0.3%), elevated triglycerides (4.0%), or low levels of high-density lipoprotein cholesterol (2.9%), the increases were not statistically significant.

Current Racial/Ethnic-Specific Prevalence of Metabolic Syndrome

To follow up on the race/ethnic trends from 1988–2004 observed in Table 1, we sought to assess the differential prevalence of metabolic syndrome and its clinical components among non-Hispanic Whites, non-Hispanic Blacks, and Hispanics using only the 1999–2004 data. Such an analysis is especially relevant because non-Hispanic Blacks and Hispanics experience greater health disparities because of diabetes, obesity, and CVD.

Using non-Hispanic White women as the reference group, we found that a significantly higher percentage of non-Hispanic Black women possessed at least 2 of the metabolic syndrome components (45.0% vs 52.7%, respectively; $P=.03$) but not the metabolic syndrome phenotype (i.e., ≥3 components; Table 2). Upon further examination, we found that a significant number of non-Hispanic Black women were obese ($P<.001$),

hypertensive (P=.003), and had evidence of a pro-inflammatory state (P=.03), although the simultaneous presence of these 3 characteristics in individual non-Hispanic Black women was not statistically different from their simultaneous presence in non-Hispanic White women (P=.17). However, a significant percentage of Hispanic women (24.6% vs 36.4%; P=.004) possessed at least 3 of the components characteristic of the metabolic syndrome phenotype, and a significant number of them were obese (for waist circumference>88 cm, P<.001; for BMI>30 kg/m^2, P=.01), had evidence of a pro-inflammatory state (P=.01), and had low levels of high-density lipoprotein cholesterol (P=.001).

Contribution of Maternal Disease History to Metabolic Syndrome

Studies have shown that a history of maternal disease (e.g., diabetes, hypertension) is associated with an increased risk of chronic disease in the offspring.[21–23] We therefore investigated the contribution of maternal history of metabolic syndrome toward the risk of an offspring possessing the syndrome phenotype or its clinical components. Specifically, we were interested in those women who reported that their mothers had been diagnosed with diabetes (n=345), hypertension (n=407), or heart attack (n=60). As shown in Table 3, women who reported that their mothers had been diagnosed with diabetes were almost twice as likely as other women to possess the metabolic syndrome phenotype (adjusted odds ratio [AOR]=1.96; 95% confidence interval [CI]=1.3, 2.8) and were also more likely to be obese (for waist circumference >88 cm, AOR=2.1, 95% CI=1.5, 3.0; for BMI>30 kg/m^2, AOR=1.7, 95% CI=1.3, 2.3), have elevated plasma glucose levels (AOR 1.9; 95% CI=1.1, 3.2), have low levels of high-density lipoprotein cholesterol (AOR=1.6; 95% CI=1.2, 2.1), or have indication of a pro-inflammatory state (AOR=1.7; 95% CI=1.3, 2.2).

Among women whose mothers were diagnosed with hypertension or a heart attack, increased risk for the metabolic syndrome phenotype was not statistically significant. However, women who reported that their mothers had been diagnosed with hypertension were more likely to be obese (for waist circumference>88 cm, AOR=1.4, 95% CI=1.1, 1.8; for BMI>30 kg/m^2, AOR=1.7, 95% CI=1.4, 2.1), have hypertension (AOR=2.3; 95% CI=1.3, 4.0), or have indication of a pro-inflammatory state (AOR=1.5; 95% CI=1.1, 2.0).

Other Maternal Risk Factors

As part of this analysis, we considered parity, age at first pregnancy, the total number of pregnancies, and age at last pregnancy as potentially important confounders. However, we did not observe a statistically significant risk conferred by these factors for metabolic syndrome or any of its clinical components (data not shown).

DISCUSSION

To the best of our knowledge, our study is the first to examine metabolic syndrome among women of childbearing age as a separate group. Using data from NHANES (1988–1994 and 1999–2004), we report a significant increase among this population in the prevalence of metabolic syndrome phenotype and in the percentages of those at risk of developing it. Further examination revealed that the greatest increase in prevalence was among non-Hispanic White women, but rates were also high for non-Hispanic Black and Hispanic women. We found that non-Hispanic Black women were significantly more likely than were non-Hispanic White women to possess at least 2 of the clinical characteristics of metabolic syndrome and that a disproportionate number were obese (P<.001), hypertensive (P=.003), and exhibited biomarkers of pro-inflammatory state (P=003).

Compared with non-Hispanic White women, Hispanic women were more likely to have at least 3 of the clinical characteristics typical of the metabolic syndrome (P=.004). Also, a significant proportion of Hispanic women were obese (for waist circumference>88 cm, P<.001; for BMI>30 kg/m^2, P=.01), exhibited biomarkers characteristic of the proinflammatory state (P=.01), and were more likely to have low levels of high-density lipoprotein cholesterol (P=.001). Compared with women whose mothers did not have diabetes, those with mothers who did had a significantly higher risk of having the metabolic syndrome phenotype (OR=1.9; 95% CI=1.3, 2.8).

Collectively, approximately 50% of our study sample of US women of childbearing age (n=643) either had the metabolic syndrome phenotype or were at increased risk for developing it. The prevalence of metabolic syndrome—and specifically of obesity—in this age group is comparable to that previously observed in other age groups, suggesting that this metabolic pathology is not restricted to those of advancing age.[24,25] Interestingly, the change in prevalence of obesity over the study period was quite dramatic, whereas only discrete changes in the remaining clinical components of metabolic syndrome were observed. This may be because the muscular tissue is most sensitive to physiological change in the presence of metabolic disease.

We also found there was a larger proportion of study participants that satisfied the definition of obesity that uses waist circumference than those that satisfied the definition that uses body mass index. Numerous studies have identified obesity defined as waist circumference greater than 88 cm as a more significant predictor of CVD in all age groups.[26–28] We therefore suggest that in future metabolic syndrome studies, both indices of obesity, as well as its duration, be considered. Finally, because we were primarily interested in characterizing the prevalence of metabolic syndrome among otherwise healthy women of childbearing age, we did not include women who reported having been diagnosed with diabetes. However, we did observe a decrease in prevalence of diagnosed diabetes between 1988 and 2004 (from 2.6 % to 1.6 %; P=.02), which suggests that even in the absence of an overt disease phenotype (such as diabetes), the risk for CVD exists in this age group.

Obesity, impaired glucose metabolism, hypertension, and dyslipidemia are recognized as independent risk factors for CVD morbidity and mortality. The clustering of these risk factors within an individual, known as metabolic syndrome, is critical to the early onset of CVD and is recognized as an emerging public health issue.[29–31] Currently, the annual mortality rate from CVD among US women younger than 50 years exceeds the annual mortality rate from breast cancer, but the cellular

and molecular mechanisms responsible for this health disparity are not clear.[24] Previous studies have documented an increase in the prevalence of metabolic syndrome in the US population,[3,7,32,33] but there are no studies examining the trends within the US female population—especially among young women of childbearing age—even as the risk for CVD mortality in women increases. If intrauterine exposure to maternal metabolic syndrome influences the health of the offspring, its long-term implications on the current epidemics of obesity, diabetes, and CVD could be significant. The prioritizing of gestational weight gain recommendations and preconception health and weight assessments by the Centers for Disease Control and Prevention and the Institute of Medicine are timely.[34,35]

The role of intrauterine exposure to pathologies associated with obesity, hypertension, and the pro-inflammatory state needs to be examined with more rigor. Both Blacks and Hispanics have high prevalence of metabolic syndrome phenotype compared with Whites; however, the etiologic mechanisms maybe differ in the 2 groups. In fact, phenotypic differences were observed; the prevalence of hypertension was very prominent among Blacks, whereas clinical dyslipidemia was more prominent among Hispanics.

In the United States, the current epidemic of overweight and obesity is concomitant with an increased prevalence of early onset CVD and type 2 diabetes.[36,37] Numerous changes in physical activity and dietary lifestyles that have occurred in the past 30 years are suspected of contributing to these epidemics.[38,39] Given the long-term effects of obesity and CVD on life expectancy, the extension of these lifestyle trends to the adolescent population is of particular concern.[33,40,41] The continuing trend of increased prevalence of risk factors for early onset CVD could further reduce the US life expectancy, which currently ranks in the lower half of the world's 30 most developed countries.[42]

Finally, studies have reported that immediate family history of diabetes and CVD is associated with risk of CVD,[43] but we are not aware of any studies that have specifically examined the link between maternal history of diabetes or CVD and development of metabolic syndrome phenotype among women of childbearing age. Our results demonstrate that longitudinal studies should be designed that examine this risk.

Limitations

Our study had some limitations. Our data analysis was restricted to providing a "snapshot" of the prevalence of metabolic syndrome among US women of childbearing age at 2 different times within the last 2 decades. Given this cross-sectional design, we were not able to provide any insight regarding these women and their CVD health as they grew older. Additionally, we were not able to address the effect of the metabolic syndrome phenotype on reproduction or on the offspring during pregnancy. Metabolic syndrome and polycystic ovary syndrome share metabolic pathologies (i.e., dyslipidemia, impaired glucose control), and women who have polycystic ovary syndrome are at increased risk for unsuccessful reproduction.[44,45] However, we were not able to assess whether the metabolic syndrome phenotype confers the same risk; future research should consider this possibility. Because this was a cross-sectional analysis, we were not able to assess whether intrauterine exposure to the mother's metabolic syndrome phenotype is propagating an intergenerational trend of obesity and metabolic syndrome. We therefore recommend that future longitudinal studies examining the development of obesity and type 2 diabetes during childhood include intrauterine exposure to the mother's metabolic syndrome phenotype as an environmental exposure.

Conclusions

We recommend that studies of metabolic syndrome consider the relationship between the increasing prevalence of the metabolic syndrome phenotype among women of childbearing age and increasing morbidity and mortality from CVD among girls and women of all ages. Because of their role as potential birth givers, females of childbearing age deserve special attention. To date, the relationship between the maternal metabolic syndrome phenotype and the offspring's development and health is not known. Future directions for our group include plans to address these gaps in metabolic syndrome research and to use animal models to establish a possible relationship between maternal metabolic syndrome phenotype and the offspring's development and health. ■

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Contributors

R.G. Ramos developed the study hypothesis and the study design and conducted the data analysis. K. Olden supervised the development of the hypothesis and the data analysis. Both authors developed the article and refined the analysis, interpretation of the findings, and revision of the article.

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Human Participant Protection

No protocol approval was needed for this study.

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