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Duration of Lactation is Associated with Lower Prevalence of the Metabolic Syndrome in Midlife—SWAN, the Study of Women's Health Across the Nation

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Abstract

Objective—To evaluate whether lactation duration is associated with lower prevalence of metabolic syndrome (MetSyn) in midlife, parous women.

Study Design—Cross-sectional cohort analysis of 2, 516 parous, midlife women using multivariable logistic regression to determine the independent association of lactation and lactation duration on prevalence of MetSyn.

Results—1,620 (64.4%) women reported a history of breastfeeding, with average lifetime duration of lactation of 1.16 (± 1.04) years. MetSyn was present in 536 (21.3%) women. Adjusting for age, smoking history, parity, ethnicity, socioeconomic status, study site, physical activity, caloric intake and high school body mass index (BMI), women with prior lactation had significantly lower odds of MetSyn (odds ratio [OR] = 0.79, 95% confidence interval [CI]= 0.63–0.99). Furthermore, increasing duration of lactation was similarly associated with lower odds of MetSyn (OR= 0.88, 95% CI= 0.77–0.99).

Conclusions—Duration of lactation is associated with lower prevalence of MetSyn in a dose-response manner in midlife, parous women.

Keywords

lactation; metabolic syndrome; parity

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Condensation: Lactation is associated with lower prevelance of the metabolic syndrome in midlife, parous women.

Introduction

The metabolic syndrome (MetSyn) is a clustering of the metabolic abnormalities: insulin resistance, dyslipidemia, hypertension (HTN) and obesity. Women with MetSyn are at increased risk of diabetes mellitus (DM)[1], major cardiovascular events[2], and increased all-cause mortality[3]. Lifestyle factors including smoking, poor diet and sedentary lifestyle are associated with increased risk of MetSyn.

Lactation creates a metabolic drain that leads to altered energy homeostasis. The studies associating weight loss with lactation, however, have been mixed [4,5] Lactation increases HDL levels[6], decreases triglyceride levels[7] and improves insulin sensitivity[5, 8, 9] in the postpartum period. Each of these changes represents an improvement in the characteristics of MetSyn. There is evidence that this enhanced metabolic efficiency persists in the immediate post-lactational period[10]. Duration of lactation has also been associated with a decreased incidence of type 2 DM[11] and possibly HTN[12] later in life, demonstrating that lactation may confer long-term benefits to the mother.

Although several studies have characterized the effects of lactation on carbohydrate and lipid metabolism, no study, to our knowledge, has examined the association between lactation duration and MetSyn. We performed a cross-sectional analysis of the association between lifetime duration of lactation and the prevalence of MetSyn in a cohort of midlife women who participated in the Study of Women's Health Across the Nation (SWAN). We hypothesized that duration of lactation is associated with a lower prevalence of MetSyn in midlife women.

Materials and Methods

The Study of Women's Health Across the Nation (SWAN)

SWAN is a multisite, multiethnic longitudinal study of 3,302 mid-life women developed to characterize patterns of health in women as they traverse the menopausal transition. Women enrolled in the SWAN study were recruited from community-based samples at seven clinical sites. At each site a Caucasian sample and a pre-specified non-Caucasian sample were recruited. African-American women were recruited at Detroit, MI; Boston, MA; Chicago, IL and Pittsburgh, PA. Hispanic women were recruited at Newark, NJ. Chinese and Japanese women were recruited at Oakland, CA and Los Angeles, CA sites, respectively. Ethnicity was self-reported based on pre-defined categories. Women could not self-classify into more than one group. Briefly, in 1995–7, a cross-sectional cohort of 16,065 women aged 40–55 completed a short screening interview, most often by telephone. The longitudinal SWAN cohort was recruited from women who had completed the screening questionnaire; 50% of eligible women were enrolled in the longitudinal cohort. The study design and recruitment process has been previously described in detail[13].

Eligibility for the longitudinal SWAN cohort included: 1) age 42–52 years; 2) an intact uterus and at least one ovary; 3) at least one menstrual period within the past 3 months; and 4) not having taken any reproductive hormones for at least 3 months. Common baseline and annual follow-up protocols were used at all sites; these included interviewer-administered forms, self-administered forms, anthropometry and phlebotomy. All data-collectors were trained and certified by SWAN. For the purposes of this study, the baseline dataset was used. For the current study, participants were required to have had at least one live birth; 2,726 met these criteria.

Written informed consent was obtained from all participants. Staff bilingual in Spanish, Cantonese and Japanese were available at the relevant sites, and all questionnaires were available in translation.

Lactation History

Participants answered retrospective questions about number of pregnancies and lactation duration following each live birth. All women who reported a live birth were included. Duration of lactation was coded in months, with less than 1 month of lactation effort coded as zero. Parous women who chose not to breastfeed were coded as zero. After the first year of lactation the infant receives the majority of its caloric needs from alternate sources. Therefore for the purposes of this study, for women who breastfed longer than one year/pregnancy each lactation interval was truncated at 1 year. Physical Measurements:

At baseline, 12-hour fasting blood samples were collected. Blood was refrigerated, centrifuged within 2 hours of phlebotomy, aliquoted, frozen and batched for approximately monthly shipment (Medical Research Laboratories International, Highland Heights, KY). Blood pressure, height, weight, waist and hip circumference were measured using standardized procedures. For these analyses, body mass index (BMI) was characterized as underweight (< 18.5 kg/m^2), normal (≥18.5 and <25 kg/m^2), overweight (≥25 and <30 kg/m^2), or obese (≥30 kg/m^2).

Demographic, Dietary and Lifestyle Factors

Demographic variables including age, income, education, self-identified race, employment and socioeconomic status were collected from interviewer- and self-administered questionnaires. Smoking status was dichotomized into current and past/never smokers. Weight at completion of high school was retrospectively self- reported. Socioeconomic status was categorized into 3 levels based on self-report of difficulty in paying for basics (food, shelter, heat). Daily caloric intake was measured using a modification of the 1995 Block Food Frequency (FFQ)[14], with addition of ethnic-specific foods to the questionnaires used at sites enrolling Hispanic, Chinese and Japanese participants. Ethnicity was categorized as African-American, Caucasian, Chinese, Japanese, or Hispanic. Physical activity questions were adapted from the Kaiser Physical Activity Survey, adapted from the Baecke physical activity questionnaire [15].

Definition of Metabolic Syndrome

Dichotomous variables were created for each component of the MetSyn based on the National Cholesterol Education Program (NCEP) III criteria[16]: 1) abdominal obesity (waist circumference >80cm for Chinese and Japanese, >88cm for Caucasians, African-Americans, and Hispanics); 2) hypertriglyceridemia (fasting triglycerides ≥150 mg/dl); 3) low HDL cholesterol <50 mg/dl; 4) elevated blood pressure (average systolic blood pressure ≤130 mm Hg or average diastolic blood pressure ≥85 mm Hg, or on antihypertensive medication); and 5) impaired fasting glucose (fasting glucose ≥110 mg/dl and ≤125 mg/dl). Participants were classified as having MetSyn if they satisfied three or more of the above criteria.

Laboratory Assays

All lipid and lipoprotein fractions were analyzed on EDTA-treated plasma. Total cholesterol was analyzed by enzymatic methods. HDL cholesterol was isolated using heparin-2M manganese chloride. Serum insulin was measured using radioimmunoassay (Diagnostics Products Corporation Coat-a-Count, Los Angeles, CA) and monitored as part of the monthly quality assurance program by the DM Diagnostic Laboratory at the University of Missouri (Columbia, MO). Glucose was measured using a hexokinase-coupled reaction (Roche Molecular Biochemicals Diagnostics, Indianapolis, IN).

Analytic Sample

Of the total cohort of 2,726 parous women, 210 women with missing lactation or MetSyn data were excluded from this analysis. The final analytic sample included 2,516 women.

Statistical Analysis

Associations between demographic and clinical characteristics stratified by MetSyn were assessed using Student's t-tests or Kruskall-Wallis tests for continuous variables. χ^2 or Fisher's exact tests were used to assess these associations for categorical variables. The association of lifetime duration of lactation with MetSyn was tested using the Wilcoxon rank-sum test. Spearman's rank correlations were performed between lifetime duration of lactation and current BMI, waist circumference, systolic blood pressure, diastolic blood pressure, and fasting levels of total cholesterol, triglycerides, HDL cholesterol, and glucose.

To evaluate the effect of lactation on the development of MetSyn, two logistic regression models were constructed with (1) lactation *ever* (yes/no) and (2) duration of lactation. Multivariable models included age, physical activity and daily caloric intake as continuous variables; high school BMI, parity, socio-economic status, study site, current smoking and ethnicity as categorical variables. Interaction product terms of lactation history with each covariate were created and tested separately in models that included all main effects terms. In a similar manner, interaction product terms of duration of lactation with each covariate were created and tested separately in models with all main effects terms. In addition, to determine whether parity affected the association between lactation history and MetSyn, logistic regression models were stratified on parity (1, 2, 3, and ≥4) and adjusted for the same covariates above.

To evaluate the independent effect of lactation on each individual component of the MetSyn, five additional adjusted logistic regression models were constructed with elevated blood pressure, abdominal obesity, impaired fasting glucose, low HDL, and elevated triglycerides as dependent outcome variables. Each model included the same covariates as the full model (above).

Since the full model could not be adjusted for current BMI due to collinearity with the outcome variable (MetSyn), we performed a sensitivity analysis in which MetSyn without the waist circumference component was our outcome variable. The logistic regression model with lactation (yes/no) as the independent variable was adjusted for all covariates noted above with the addition of current BMI.

All statistical tests used a two-tailed α of 0.05. All analyses were performed using STATA 8.2 (StataCorp LP, College Station, TX).

Results

At baseline, the SWAN cohort consisted of 2,516 parous women with a mean (SD) age of 46.4 (2.7) years, mean BMI of 28.4 (7.2) kg/m^2, and a median (interquartile range) parity of 2.0 (1.0) live births per woman. Of these women, 1,620 (64.4%) reported a history of lactation. The mean lifetime duration of lactation among women who breastfed was 1.16 (± 1.04) years.

There were 536 (21.3%) prevalent cases of MetSyn. Among those who breastfed, 297 (18.3%) met the criteria for MetSyn compared to 239 (26.7%) among those who did not ($p< 0.01$). Women who developed MetSyn were more likely to have a higher BMI at time of interview ($p< 0.01$) and at completion of high school ($p< 0.01$), be African American ($p < 0.01$) or Hispanic ($p< 0.01$), smoke ($p= 0.02$), and be of lower socioeconomic status ($p= 0.04$) (Table 1). They breastfed for shorter periods of time (Wilcoxon rank-sum, $p<0.01$).

Spearman's rank correlations were computed on all women, and found duration of lactation inversely correlated with current BMI ($r_s= -0.16$, $p<0.01$), waist circumference ($r_s= -0.18$, $p< 0.01$), systolic blood pressure ($r_s= -0.17$, $p<0.01$), diastolic blood pressure ($r_s= -0.09$, $p< 0.01$),

fasting levels of glucose (r_s= −0.09, p<0.01), insulin (r_s= −0.15, p<0.01), triglycerides (r_s= −0.06, p< 0.01), total cholesterol (r_s= −0.06, p< 0.01), and LDL cholesterol (r_s= −0.07, p< 0.01). There was a positive correlation with fasting HDL levels (r_s= 0.07, p<0.01). While all of the above correlations were statistically significant, it is worth noting that the strength of these associations was relatively small.

Logistic regression analyses were performed to assess the association of lactation with MetSyn. Parous women who had *ever* breastfed had a significantly lower prevalence of MetSyn: unadjusted OR of 0.62 (95% CI: 0.51, 0.75). This association remained significant in the multivariable model adjusting for age, current smoking, parity, ethnicity, socioeconomic status, study site, physical activity, caloric intake and high-school BMI: OR of 0.77 (95% CI: 0.62, 0.96) (Table 2). The model was not adjusted for current BMI due to collinearity with the waist circumference component of our outcome variable (r_s=0.92, p< 0.01). We entered MetSyn without the waist circumference component into the multivariable model, and after adjusting for current BMI the relationship between lactation and this newly defined metabolic cluster remained statistically significant (p= 0.02). There were no statistically significant interactions of lactation history with any covariate, thus no interaction terms were included in the final model. Logistic regression analyses were then performed to assess the association of lactation history with each component of the MetSyn. Women who had ever breastfed were significantly less likely to have impaired fasting glucose (p <0.01), elevated blood pressure (p= 0.048), and abdominal obesity (p< 0.01).

Logistic regression analysis found a significant association of duration of lactation with MetSyn: the unadjusted OR for each year of lactation was 0.80 (95% CI: 0.72, 0.91). The multivariable model adjusting for all the covariates mentioned above also found a significant association: the OR per each additional year of lactation was 0.88 (95% CI: 0.77, 0.99) for MetSyn (Table 3). There were no statistically significant interactions of lactation duration with any covariate, thus no interaction terms were included in the final model. Logistic regression analyses exploring the duration of lactation with each MetSyn component demonstrated significant inverse relationships with elevated blood pressure (p= 0.04) and abdominal obesity (p < 0.01)

When the multivariable model was stratified by parity (Table 4), a statistically significant inverse relationship between duration of lactation and MetSyn was seen in women who had one (OR: 0.57; 95% CI: 0.34, 0.95), or two (OR: 0.69; 95% CI: 0.47, 0.998) successful pregnancies. However, in women who had four or more successful pregnancies, this inverse relationship no longer persisted.

Comment

A protective association between a history of lactation and MetSyn has recently been demonstrated[17]. Our study supports and extends these observations to show that the rate of MetSyn is significantly lower with increasing duration of lactation, suggesting a dose-response relationship. However, a threshold appears to be reached between the third and fourth pregnancies, after which any protective effect no longer remains. This finding was unexpected, as increasing parity should be associated with increasing duration of lactation. Increasing parity, however, is also associated with increased weight retention and has been independently associated with increased prevalence of MetSyn[17]. We hypothesized, therefore, that the effects of increasing parity outweigh the benefits of increased duration of lactation between the third and fourth pregnancies. A history of lactation has been shown to attenuate the adverse changes in LDL cholesterol, and longer duration of breastfeeding (>3 months) has been shown to attenuate the decrements in HDL cholesterol associated with pregnancy up to 16 months post delivery[18]. We also found a statistically significant correlation between a duration of lactation

and HDL cholesterol, and an inverse correlation with LDL cholesterol. Furthermore, when examining the association of lactation on the individual components of MetSyn, we found that lactation was associated with significantly lower odds of elevated blood pressure, abdominal obesity, and impaired fasting glucose after adjusting for multiple risk factors.

Duration of lactation has been associated with decreased incidence of type 2 DM among parous women[15]. This finding is in agreement with our observation of a statistically significant protective effect of lactation on fasting glucose. Increasing duration of lactation has been shown to protect against the development of HTN in Korean women[16]. We found that duration of lactation had a significant protective effect against development of elevated blood pressure.

The strengths of our study lie in its large sample, and comprehensive collection of metabolic and lactation data. Breastfeeding data was collected as a continuous variable, not extrapolated from a categorical collection. Another strength is the multiethnic composition of our cohort that suggests generalizability of our results to other populations.

Our study has a number of limitations. A cross-sectional analysis cannot construct a causal or temporal relationship between the variable of interest and the outcome measure. It is possible that MetSyn preceded lactation. Alternatively, women who are prone to developing MetSyn may have difficulty initiating lactogenesis. Several studies have suggested that maternal obesity may be associated with decreased breastfeeding initiation and duration [19,20,21]. However, our multivariable regression model is adjusted for high school BMI. Furthermore, in a post-hoc analysis when the model is stratified by high school BMI, the point estimates are similar and protective for the development of MetSyn in each category except the highest (data not shown). Lactation may protect against obesity, and this may be driving the association with MetSyn. This is difficult to evaluate in our model due to collinearity with our outcome variable. However, in adjusted analyses lactation was significantly associated with several components of the MetSyn in addition to abdominal obesity. Furthermore, when we removed the waist circumference component of MetSyn, and re-entered it into the multivariable model and adjusted for current BMI the relationship between history of lactation and this metabolic cluster remained statistically significant. Women who choose to breastfeed were more likely to choose other healthy behaviors and this “healthy lifestyle” bias may lead to confounding. We adjusted for markers of a healthy lifestyle, including diet, exercise, and smoking history. After adjusting for these markers, the protective association of lactation on development of MetSyn remained unchanged. Recall bias may be another limitation of this study; women were asked to provide lactation histories and weight at completion of high school several years after the prevalent outcomes. However, several studies have found both reporting of breastfeeding duration22, 23 and recall of high school weight24 to be a valid, reliable measure up to 20 years later.

Lactation may prime the metabolic system by making it a more energy-efficient machine, and this metabolic efficiency may persist in the post-lactational period. In the immediate post-lactational period, fasting plasma free fatty acids both basally and in response to noradrenaline infusion are significantly lower than those observed during lactation or in bottle-feeding and non- pregnant controls [8]. Similarly, post- lactation the response of plasma glycerol to noradrenaline is significantly lower than these same controls [8]. Each of these changes represents an improvement in metabolic efficiency.

Lactation may decrease visceral adiposity, and indeed we demonstrated a negative association between lactation and abdominal obesity. Central fat accumulation has been postulated to be a physiological basis for reduced postprandial thermogenesis[25]. A redistribution of fat from central stores through lactation may lead to improved postprandial thermogenesis, and a more efficient metabolism.

Lactation may also decrease the prevalence of MetSyn by improving insulin sensitivity. Unfortunately, most of the data on lactation and carbohydrate metabolism has been collected from gestational diabetics and confined to the immediate postpartum period. Nonetheless, improved glucose tolerance, fasting glucose, and total area under the glucose tolerance curve have been shown in breastfeeding gestational diabetics[5]. Gestational diabetics who lactate have a higher disposition index, indicating a more efficient pancreatic beta-cell function[7]. We found a significant inverse correlation between duration of lactation and fasting levels of both glucose and insulin. Longitudinal studies in women with intact carbohydrate metabolism are needed to evaluate whether lactation has a long-term impact on insulin sensitivity.

In conclusion, we have found that duration of lactation is associated with prevalence of MetSyn in parous midlife women in a dose-response manner. This association is most marked after the first and second pregnancies, and appears to reach a threshold by the fourth pregnancy. These changes may be mediated by changes in insulin resistance, visceral adiposity and/or free fatty acid metabolism. Further research is needed to confirm and elaborate upon these results. In addition to the pediatric benefits of breastfeeding, these findings of maternal benefit may encourage more women to initiate and maintain breastfeeding behavior.

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Table 1

Characteristics of cohort stratified by presence or absence of MetSyn

	Absence of MetSyn (N= 1,980)	Presence of MetSyn (N= 536)	P- value
Lifetime			
Lactation (years)	1.12 (0.96)	1.05 (0.99)	< 0.01
Age	46.5 (2.2)	46.7 (2.1)	0.48
Daily caloric intake	1852.1 (786.5)	1936.3 (793.5)	0.09
Physical activity	7.8 (1.7)	7.2 (1.6)	0.28
High School			
BMI	20.6 (2.9)	22.4 (2.9)	< 0.01
Current BMI	26.3 (7.2)	34.5 (6.6)	< 0.01
Parity n (%):			
Para 1	400 (20.1)	114 (21.2)	0.61
Para 2	842 (42.4)	188 (34.9)	< 0.01
Para 3	469 (23.6)	125 (23.2)	0.81
Para 4 or greater	275 (13.9)	112 (20.7)	< 0.01
Ethnicity:			
African Amer	589 (29.8)	195 (36.4)	< 0.01
Caucasian	862 (43.6)	219 (40.9)	0.86
Hispanic	163 (8.2)	66 (12.3)	< 0.01
Chinese	168 (8.5)	25 (4.7)	< 0.01
Japanese	195 (9.9)	30 (5.6)	0.02
Lowest SES category	170 (8.6)	79 (14.7)	0.04
Current Smokers	788 (39.8)	249 (46.5)	0.02

Continuous variables presented as mean (standard deviation) with p-values calculated by t-tests or Kruskal- Wallis tests, as appropriate. Categorical variables presented as n (%) with p-values calculated by χ^2

Table 2

Impact of a history of lactation (*ever/never)* on MetSyn and components of MetSyn, adjusted for multiple covariates

Model Outcome[a]	Odds Ratio[b]	95% CI	P- value
MetSyn	0.77*	0.62, 0.96	0.02
Elevated Blood Pressure	0.83*	0.68, 0.998	0.048
Abdominal Obesity	0.70*	0.58, 0.86	<0.01
Impaired Fasting Glucose	0.59*	0.40, 0.87	<0.01
Low HDL	0.85	0.70, 1.02	0.08
Elevated Triglycerides	0.93	0.74, 1.18	0.58

[a] Logistic regression models adjusted for age, smoking history, parity, ethnicity, study site, socioeconomic status, physical activity, daily caloric intake, and high school BMI.

[b] Odds ratio for history of ever breastfeeding as predictor of each model outcome in analyses adjusting for multiple covariates

* denotes statistical significance

Table 3

Impact of duration of lactation *(per year)* on MetSyn and components of MetSyn, adjusted for multiple covariates.

Model Outcome[a]	Odds Ratio[b]	95% CI	P- value
MetSyn	0.88*	0.77, 0.99	0.03
Elevated Blood Pressure	0.90*	0.81, 0.996	0.043
Abdominal Obesity	0.86*	0.78, 0.96	<0.01
Impaired Fasting Glucose	0.81	0.63, 1.03	0.09
Low HDL	0.99	0.89, 1.10	0.85
Elevated Triglycerides	0.90	0.79, 1.02	0.10

[a] Logistic regression models adjusted for age, smoking history, parity, ethnicity, study site, socioeconomic status, physical activity, daily caloric intake, and high school BMI.

[b] Odds ratio for duration of breastfeeding as predictor of each model outcome in analyses adjusting for multiple covariates

* denotes statistical significance

Table 4

Multivariable logistic regression models assessing the relationship of a history of lactation with prevalence of MetSyn stratified by parity

Parity:	Odds Ratio	95% CI	P value
Para 1	0.57*	0.34, 0.95	0.03
Para 2	0.69*	0.47, 0.998	0.048
Para 3	0.69	0.43, 1.10	0.12
Para 4 and greater	1.31	0.68, 2.54	0.41

* denotes statistical significance