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Breast-feeding, Leptin:Adiponectin Ratio, and Metabolic Dysfunction in Adolescents with Obesity

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Abstract

Objectives—Increased adiposity increases leptin and decreases adiponectin concentrations, resulting in an increased leptin:adiponectin ratio (LAR). In adults, components of the metabolic syndrome and other cardiometabolic risk factors, what we classify here as "metabolic dysfunction," are associated with both a high LAR and a history of being breast-fed. The relation among breast-feeding, LAR, and degree of metabolic dysfunction in obese youth is unknown. The purpose of our pilot study was to explore this relation and estimate the effect size of the relations to determine the sample size needed to power future prospective studies.

Methods—We obtained fasting levels of leptin, adiponectin, lipids, insulin, and glucose from obese youth (aged 8–17 years). Weight, height, waist circumference, blood pressure, and breast-feeding history also were assessed.

Results—Of 96 participants, 78 were breast-fed as infants, 54% of whom were breast-fed for >6 months. Wide variation was observed in LARs among children who were and were not breast-fed (>100% coefficient of variation). Overall, prevalence of metabolic dysfunction in the cohort was 94% and was not proven to be associated with higher LAR.

Conclusions—In this cohort of obese youth, we found a high prevalence of breast-feeding, metabolic dysfunction, and wide variation in the LARs. Based on the effect size estimated, future studies would need to enroll >1500 patients or identify, stratify, and selectively enroll obese patients without metabolic dysfunction to accurately determine whether breast-feeding in infancy influences LARs or metabolic dysfunction among obese youth.

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Keywords

adolescents; breast-feeding; leptin:adiponectin ratio; metabolic dysfunction; obesity

The metabolic syndrome is a known risk factor for diabetes mellitus and cardiovascular disease (CVD). In 2008 the International Diabetes Federation presented unified criteria for the metabolic syndrome in children and adolescents. These criteria include large waist circumference (>90th percentile for age and sex) plus two other criteria (elevated triglycerides, low high-density lipoprotein-cholesterol [HDL-C], elevated blood pressure, or elevated fasting glucose).[1] There are, however, several risk factors for diabetes mellitus and CVD that were not included in the 2008 criteria for the metabolic syndrome. Metabolic dysfunction is a broader term that includes risk factors such as insulin resistance, elevated low-density lipoprotein-cholesterol (LDL-C), and elevated non-HDL-C.[2] In addition, the use of waist:height ratio (WHR) ≥0.5 rather than waist circumference >90th percentile has been shown to have greater association with metabolic dysfunction and is easier to use in the clinical setting because it is an absolute number rather than the need for a table to identify the 90th percentile for a given age and sex.[3]

Although obesity is a known risk factor for metabolic dysfunction, not all obese individuals demonstrate evidence of metabolic dysfunction.[4] Depending on the criteria used to define metabolic dysfunction (one or more of the above, excluding WHR or waist circumference), its prevalence among obese adolescents ranges widely from approximately 14% to 52%.[5–7] A number of factors have been associated with a lower prevalence of metabolic dysfunction among obese individuals, including lower leptin and higher adiponectin concentrations (resulting in a lower LAR) and a history of their being breast-fed in infancy.[8–10]

In obese adolescents, adiponectin levels are inversely related to triglycerides, systolic blood pressure, and WHR and are positively related to HDL-C levels.[11] The LAR is associated with variation in the risk for metabolic dysfunction. In adults, a high LAR is associated with a greater risk of CVD and a higher prevalence of the metabolic syndrome.[10,12–14] In adults, a high LAR has been observed to be a better predictor of insulin resistance than the homeostasis model of assessment-insulin resistance (HOMA-IR) or leptin and adiponectin levels alone.[15–17] A study of obese children 8 to 14 years old found that the LAR was a poor predictor of insulin resistance, however.[18]

Breast-feeding also may influence the risk of developing metabolic disease later in life.[9] The risk of developing type 2 diabetes mellitus and CVD is lower in individuals who were breast-fed during infancy.[19,20] Other studies have suggested that a history of breast-feeding is associated with lower cholesterol, body mass index (BMI), and blood pressure in adulthood.[2,8,9,20,21] Although breast-feeding and a low LAR both are associated with a reduced risk of metabolic dysfunction, it is not known whether the beneficial effect of breast-feeding is mediated through a lower LAR. The aim of this pilot study was to test the hypothesis that a longer duration of breast-feeding is associated with a lower LAR and a lower prevalence of metabolic dysfunction among a cohort of obese children and adolescents.

Methods

Participants

Obese (BMI ≥95th percentile for age and sex)[22] children and adolescents, 8 to 17 years old, were recruited from pediatric clinics and community centers in Salt Lake County, Utah, between July 15, 2010 and January 31, 2013. The study was approved by the institutional review board at the University of Utah. Parents gave written informed consent and participants gave written assent before data collection. Participants were required to be in good health other than insulin resistance, dyslipidemia, impaired glucose tolerance, or hypertension. Exclusion criteria included not being able to speak either English or Spanish; having a genetic syndrome or other endocrine disorder known to cause obesity (eg, Prader-Willi syndrome, leptin deficiency, Cushing disease, hypothyroidism); being pregnant or a history of pregnancy; having cancer or a history of cancer; active infectious disease; a history of CVDs or stroke during the previous 36 months; plasma triglycerides >400 mg/dL (more likely caused by familial hypertriglyceridemia than dyslipidemia of obesity); diabetes mellitus (types 1 and 2); and the use of psychotropics, sulfonylureas, thiazolidinediones, insulin, glucocorticoids, antineoplastic agents, angiotensin-receptor blockers, or angiotensin-converting enzyme inhibitors.

Data Collection

Anthropometrics—Weight was obtained using a digital scale (model 5002, Scale-Tronix, White Plains, NY). Height was measured to the nearest 0.1 cm with a stadiometer (Height-Rite 225, Seca, Culver City, CA). Waist circumference was measured to the nearest 0.1 cm by wrapping a measuring tape around the torso at the level of the umbilicus. The measurement was recorded at the end of expiration while the tape was held snugly without compressing the skin. Height and waist circumference were each taken twice; if the difference between the two measurements was >1.0 cm, then a third measurement was performed and an average of the measurements was used.

Waist circumference and height were used to calculate the WHR. BMI was calculated as kilograms per square meter. Blood pressure was measured two times, usually in the right arm, using an automated sphygmomanometer (Dynamap Pro 400, GE Healthcare, Fairfield, CT). If the difference between the measurements was >5 mm Hg, then a third measurement was performed and an average of the measurements was used. Trained research personnel performed all of the measurements.

Biomarkers—Participants fasted for at least 12 hours overnight before obtaining serum concentrations of leptin, adiponectin, insulin, glucose, and a complete lipid profile. Serum leptin, adiponectin, and insulin concentrations were measured in an enzyme-linked immunosorbent assay format (Alpco Diagnostics, Salem, NH; intraassay precision 1.0%–7.4%, interassay precision 2.4%–8.4%, sensitivity 1.5 ng/mL, accuracy 92%–100%). Procedures for each assay were performed twice with a standard and control on the same plate. Samples exceeding the highest concentration of the standard curve were diluted 1:2 and reassayed in duplicate. Standard curves for all plates yielded an R^2 value of ≥0.98. Lipid analysis was performed using a manual method (colorimetric for total cholesterol and

triglycerides, dextran sulfate precipitation method for HDL-C). The coefficients of variation for total cholesterol and triglycerides were <2% and <5% for HDL-C.[23] LDL-C was calculated using the Friedewald equation. Glucose was measured using a glucose analyzer.

Metabolic Dysfunction—We defined metabolic dysfunction as the presence of at least one abnormal measurement of HDL-C, LDL-C, triglycerides, non-HDL-C, glucose, HOMA-IR, blood pressure, or WHR. Specific cutoffs for measurements included HDL-C ≤40 mg/dL, LDL-C ≥110 mg/dL, triglycerides ≥75 mg/dL for children 9 years old and younger and ≥90 mg/dL for children aged 10 to 17 years, non-HDL-C ≥145 mg/dL, glucose >100 mg/dL, HOMA-IR ≥3.16 (a surrogate for insulin resistance),[19] blood pressure ≥90th percentile or >120/80 mm Hg,[2] and a WHR ratio ≥0.5.[3,24]

Breast-feeding History—Breast-feeding history (child's age at first food or beverage other than breast milk [between 1 week and ≥6 months] and age at complete cessation of breast-feeding [between 1 week and ≥12 months]) was used to determine breast-feeding duration by noting the child's age at complete cessation of breast-feeding. Data were collected at the patient's appointment using a parent/guardian questionnaire with questions from the National Health and Nutrition Examination Survey diet behavior and nutrition survey of 2003–2004.[25] If the survey was not administered during the appointment, then the parent/guardian was contacted by a trained research assistant by telephone or e-mail to collect the information.

Statistical Analysis

The primary objectives of this study were to determine the relations among breast-feeding, its duration, LAR, and the prevalence of metabolic dysfunction in a cohort of obese children and adolescents. The breast-feeding variable was categorized as "not breast-fed" and "breast-fed," which was further divided into <3 months, 3 to 6 months, and >6 months, resulting in four groups.

To define the effect size and its variability, a generalized linear model was used to determine the relation between the LAR and breast-feeding duration while controlling for ethnicity and sex. Neither ethnicity nor sex affected the relation. In addition, logistic regression was applied in a binary response model to predict the presence of metabolic dysfunction in the presence or absence of any breast-feeding. The Kruskal-Wallis rank sum test was used to determine whether the duration of breast-feeding was significantly related to the LAR and to test whether the LAR had a significant relation with metabolic dysfunction. Statistical analyses were performed using SAS statistical software version 9.3 (SAS Institute, Cary, NC) and R version 3.3.1 (R Foundation, Vienna, Austria) and RStudio version i386 3.3.1 (RStudio, Boston, MA). Statistical significance was defined as $P < 0.05$. Research electronic data version 6.10.17 (Vanderbilt University, Nashville, TN) was used for the electronic database.[26]

Results

Participant Characteristics

A total of 124 children and adolescents were enrolled in the study. Of these, 96 had data regarding breast-feeding, anthropometry, and biomarkers. Table 1 details the participant characteristics. The ethnicity for the cohort was Hispanic (56%), non-Hispanic white (30%), and unknown (14%).

Of the 96 obese children and adolescents, all but 1 participant had an WHR ≥0.5. As such, we redefined metabolic dysfunction without WHR for statistical analyses. The resulting prevalence of metabolic dysfunction in the cohort was 94%, with 48% identified as having ≥3 criteria (Table 2). Elevated triglycerides was the most common abnormal finding (69%). The mean LAR was 8.1 (range 0.3–37.1). A history of breast-feeding was present in 78 (81%) participants. The average total time spent breast-feeding in infancy for these individuals was 8.3 months; the average time spent exclusively breast-feeding was 3.7 months. Of these, 14 (18%) were breast-fed for <3 months (4 breast-fed <1 month, 8 breast-fed <1 week), 22 (28%) for 3 to 6 months, and 42 (54%) for >6 months.

Breast-feeding Duration and LAR

There was no association between breast-feeding, as a binary variable or any duration of breast-feeding as an infant, and the LAR as an adolescent (Table 3).

Breast-feeding and Metabolic Dysfunction

There was no association between breast-feeding, as a binary variable or any duration of breast-feeding as an infant, and metabolic dysfunction as an adolescent (Table 4).

Relation Between LAR and Metabolic Dysfunction

The estimate of mean LAR was lower in those without metabolic dysfunction (1.5 [0.7–2.5]) compared with those with metabolic dysfunction (8.4 [0.3–37.1]), but the uncertainty was high. Using logistic regression for the binary variable of metabolic dysfunction compared with LAR, the estimated odds ratio was 0.398 (95% confidence interval 0.139–1.143, $P = 0.087$).

Discussion

We were unable to demonstrate a significant relation between breast-feeding duration and either the metabolic syndrome or LAR. There was a trend for higher LAR to be associated with a greater degree of metabolic dysfunction. The possible explanations for this failure to find the hypothesized relations include either that there is, in fact, no relation or that the sample size was small. We anticipated that the prevalence of metabolic dysfunction in the non–breast-fed group would be higher than in the group with a history of breast-feeding. There were, however, only 18 participants who were never breast-fed, which is much lower than we expected given that breast-feeding protects against obesity in children.[27] Also, we noted the high prevalence of metabolic dysfunction (at least 1 risk factor in addition to WHR) in our cohort. The prevalence of metabolic syndrome in our cohort of obese

adolescents was similar to that reported by other researchers.[28,29] The relation between breast-feeding and cardiometabolic profile in childhood is unclear; some studies report a protective role and others, as in our study, report no relation.[30,31]

We found no difference in the LAR in those who were breast-fed (8.2 ± 8.0) compared with those who were not (7.9 ± 8.7; Table 3). Previous studies of potential associations between the LAR or only adiponectin in infants and breast-feeding have shown a higher adiponectin concentration or no relation in obese children.[32,33] None of these studies have investigated the relation between LAR in adolescents and history of breast-feeding.

Although breast-feeding confers many benefits on infants and nursing mothers, our results do not support studies suggesting that breast-feeding is associated with a lower prevalence of metabolic dysfunction among adolescents with obesity. The research, including a systematic review and meta-analysis conducted by the World Health Organization, proposes that a history of being breast-fed may promote lower cholesterol, BMI,[2,20] and blood pressure.[20] Our study agrees instead with the studies of Parikh et al and Rudnicka et al, which found no association between breast-feeding in infancy and adult levels of LDL, triglycerides, and blood pressure.[8,34]

Differing results between the present study and the literature may stem from dissimilar participants and study designs, as well as a higher prevalence of breast-feeding (longer and exclusively) in our cohort. For example, the systematic review of 30 studies by the World Health Organization included young participants (1 year old at measurement) and late adolescent participants (15–19 years old).[20] None of the studies included the age range (8–17 years) of our participants. In addition, the review included cross-sectional, cohort, and randomized controlled trial study designs. The majority of study populations were born before 2000, which is before the global obesity epidemic.[A]

The primary limitations of this study were the unexpectedly small percentage of subjects whose parents reported no breast-feeding and the high prevalence of metabolic dysfunction. It is possible that mothers who reported breast-feeding longer than 6 months actually breast-fed only for a few months (3–6 months), which would compromise our ability to find an association between breast-feeding duration and metabolic dysfunction and LAR. It is unlikely that a parent would recall that she had breast-fed a child when she had not. Another limitation was that the study population was not representative of obese children and adolescents in the United States, because the sample population consisted of primarily Hispanic and non-Hispanic white individuals. Relations among breast-feeding, metabolic dysfunction, and LAR may differ in other populations of obese children and adolescents.

Because of the high prevalence of breast-feeding and metabolic dysfunction, our study was not adequately powered to detect a 10% difference in prevalence of metabolic dysfunction among breast-feeding groups. Based on these findings, power calculations were performed using the two-sample test for proportions at varying sample sizes ranging from 100 to 2000 patients in each group (breast-fed and non–breast-fed). A sample size of >1500 is likely

[A]Please insert a reference citation indicating the established start date (ie, year or decade) of the global obesity epidemic.

needed to have adequate power to determine whether there is a relation between breast-feeding and LAR or metabolic dysfunction.

Conclusions

The results of this pilot study reveal a high prevalence (94%) of metabolic dysfunction, high prevalence of breast-feeding (81%), and evidence of high variability of LARs among children and adolescents with obesity, regardless of whether they were breast-fed. A sample size of at least 1500 would be necessary to be confident with 80% power that there is no relation between breast-feeding and the prevalence of metabolic dysfunction among children and adolescents with obesity. A similar number of patients would be required to assess whether breast-feeding or its duration has a favorable impact on LAR. Such studies may prove useful in determining whether breast-feeding provides the same protective effects during childhood and adolescence in obese individuals as it is purported to have in normal-weight individuals.

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[B]As received, ref 25 was incomplete. The copyeditor inserted a possible URL. Please confirm whether this is the correct information.

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Key Points

- In this cohort of adolescents (8 to 17 years old) with obesity there was a high prevalence (81%) of their being breast-fed during infancy and a high prevalence (94%) of metabolic dysfunction.
- There is no association between the leptin:adiponectin ratio and breast-feeding or metabolic dysfunction.
- There is no association between breast-feeding during infancy and presence of metabolic dysfunction.
- A sample size of >1500 adolescents with obesity is needed to determine adequately the relation among breast-feeding, leptin:adiponectin ratio, and metabolic dysfunction in obesity.

Table 1

Participant characteristics (N = 96)

	Breast-fed	Non–breast-fed
Age, y, mean ± SD	11.8 ± 2.7	11.4 ± 2.6
BMI, kg/m^2, mean ± SD	29.4 ± 5.9	27.8 ± 6.2
Sex, % (n)		
Male	47 (37)	44 (8)
Female	53 (41)	56 (10)
Ethnicity, % (n)		
Hispanic	56 (44)	56 (10)
White, Non-Hispanic	22 (25)	22 (4)
Other/No Response	22 (9)	22 (4)

No significant differences were found between breast-fed and not breast-fed. BMI, body mass index; SD, standard deviation.

Table 2

Prevalence of abnormal metabolic dysfunction markers (N = 96)

Metabolic marker	Breast-fed, n = 78 (%)	Non–breast-fed, n = 18 (%)
HDL-C ≤40 mg/dL	25 (32)	5 (28)
Non-HDL-C ≥145 mg/dL	15 (19)	2 (11)
Triglycerides ≥75 mg/dL (8–9 y), ≥90 mg/dL (≥10 y)[A]	57 (73)	10 (56)
LDL-C ≥110 mg/dL	16 (21)	1 (6)
Fasting glucose >100 mg/dL	2 (3)	3 (17)
HOMA-IR ≥3.16	44 (56)	11 (61)
Systolic blood pressure >120 mm Hg or >90th percentile	27 (35)	9 (50)
Diastolic blood pressure > 80 mm Hg or >90th percentile	10 (13)	1 (6)
Total abnormal blood pressure	28 (36)	9 (50)
WHR ≥0.5	78 (100)	17 (94)

No significant differences were found between breast-fed and not breast-fed.HDL-C, high-density lipoprotein-cholesterol; HOMA-IR, homeostasis model of assessment-insulin resistance; LDL-C, low-density lipoprotein-cholesterol; WHR, waist:height ratio.

[A]It seems as though there should be 2 sets of numbers in the Triglycerides row, one for 8–9 year olds and one for the older group. Please clarify and correct, if needed.

Table 3

Breast-feeding duration and corresponding LAR (N = 96)

Breast-feeding duration (no. participants)	Leptin, ng/mL	Adiponectin, μg/mL	LAR
Non–breast-fed, n = 18	37.7 ± 25.9	6.6 ± 2.7	7.9 ± 8.7
Breast-fed, n = 78	44.6 ± 38.4	6.8 ± 3.3	8.2 ± 8.0
<3 mo, n = 14	39.9 ± 32.4	7.0 ± 2.6	6.2 ± 4.7
3–6 mo, n = 22	35.1 ± 22.8	5.7 ± 2.6	7.3 ± 5.0
>6 mo, n = 42	51.1 ± 45.4	7.2 ± 3.7	9.3 ± 9.9

No statistically significant differences were found between breast-feeding groups. LAR, leptin:adiponectin ratio.

Table 4

Breast-feeding duration and presence of metabolic dysfunction[a](N = 96)

Breast-feeding duration (no. participants)	Any metabolic dysfunction, %	1–2 criteria of metabolic dysfunction, %	3 criteria of metabolic dysfunction, %	4–5 criteria of metabolic dysfunction, %
Non–breast-fed, n = 18	89	50	22	17
Breast-fed, n = 78	95	45	36	14
<3 mo, n = 14	100	58	21	21
3–6 mo, n = 22	95	55	31	9
> 6 mo, n = 42	93	36	43	14
Overall sample, N = 96	94	46	33	15

No statistically significant differences were found between breast-feeding groups. HDL-C, high-density lipoprotein-cholesterol; HOMA-IR, homeostasis model of assessment-insulin resistance; LDL-C, low-density lipoprotein-cholesterol; WHR, waist:height ratio.

[a]Metabolic dysfunction includes ≥1 abnormal measurements of HDL-C, LDL-C, triglycerides, blood pressure, and HOMA-IR (surrogate for insulin resistance). WHR is excluded from definition because only 1 participant had WHR in the normal range.