Influence of Breastfeeding on Obesity and Type 2 Diabetes Risk Factors in Latino Youth With a Family History of Type 2 Diabetes

JAIMIE N. DAVIS, PHD[1]
MARC J. WEIGENSBERG, MD[1,2]
GABRIEL Q. SHAIBI, PHD[3]
NOE C. CRESPO, MPH[1]
LOUISE A. KELLY, PHD[1]
CHRISTIANNE J. LANE, PHD[1]
MICHAEL I. GORAN, PHD[1]

OBJECTIVE — To determine whether breastfeeding is related to total adiposity, regional adiposity, and glucose and insulin dynamics in overweight Latino youth throughout puberty.

RESEARCH DESIGN AND METHODS — The relation between breastfeeding and diabetes risk was determined in 240 overweight (BMI ≥85th percentile) Latino children (aged 8–13 years) with a positive family history of type 2 diabetes. Children were examined at baseline (Tanner pubertal stage 1) and for 2 more years as they advanced in pubertal maturation. Children were divided into the following categories: never breastfed (n = 102), breastfed 0–5.99 months (n = 61), breastfed 6–11.99 months (n = 24), and breastfed ≥12 months (n = 53). Tanner pubertal stage was determined by physical examination. Visceral and subcutaneous abdominal fat were determined by magnetic resonance imagining, and total body fat, total lean tissue mass, and percent body fat were measured by dual-energy X-ray absorptiometry. Fasting and post-challenge glucose were assessed with a 2-h oral glucose tolerance test. Insulin sensitivity (SI), acute insulin response (AIR), and disposition index ([DI] an index of β-cell function) were measured by frequently sampled intravenous glucose tolerance test and minimal modeling. Data were analyzed using linear mixed-effects modeling.

RESULTS — There were no significant effects of breastfeeding categories on adiposity (i.e., total fat mass, total lean tissue mass, percent body fat), fat distribution (visceral and subcutaneous abdominal fat), fasting glucose or 2-h glucose, or insulin dynamics (SI, AIR, and DI) at Tanner pubertal stage 1 or on changes in these variables over pubertal transitions in overweight Latino youth.

CONCLUSIONS — In this population of high-risk Latino youth, there were no significant protective effects of breastfeeding on adiposity or type 2 diabetes risk factors at Tanner pubertal stage 1 or across advances in maturation.

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Whether breastfeeding protects against obesity later in life is a controversial topic in literature. Some investigators have shown that breastfeeding has protective effects against childhood obesity (1–5), while others have not observed an association (6–8). Moreover, the protective effect of breastfeeding may vary according to ethnicity (6,9,10). This debate may be hampered because of prior study designs in that the majority of studies have used either global measures of adiposity such as BMI or less accurate estimates of body composition such as skinfolds. To date, few studies have examined the effects of breastfeeding on general adiposity using dual-energy X-ray absorptiometry (DEXA) (11–13), and no study has examined the effects of breastfeeding on fat distribution using magnetic resonance imaging (MRI). Moreover, no previous study has assessed the protective breastfeeding effects on adiposity or risk factors for type 2 diabetes across pubertal transition from Tanner stage 1 to 5.

Recently, several investigators have demonstrated that breastfeeding was also protective against type 2 diabetes in Caucasian children (14) and adults (15) and in Pima Indian children and adults (16). Type 2 diabetes is no longer thought of as an adult disease and is occurring at increasing rates in younger and minority populations (17,18). We have previously shown that over 30% of overweight Latino children with a positive family history of type 2 diabetes exhibit impaired glucose tolerance (IGT) (19). We have found that Latino children are more insulin resistant than Caucasian children, independent of body fat content (20). Although we recently showed that insulin sensitivity (SI) in Latino children declines over time, this decline is unrelated to changes in body fat or maturation. However, we found that the acute insulin response to glucose worsens in advanced Tanner stages, thus indicating the diminishing capacity of β-cells to respond to the decline in SI as children mature (21). These results suggest that pubertal transi-

From the [1]Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California; the [2]Department of Pediatrics, LAC+USC Medical Center, Los Angeles, California; and the [3]College of Nursing and Healthcare Innovation, Arizona State University, Phoenix, Arizona.

Address correspondence and reprint requests to Michael I. Goran, 2250 Alcazar St., CSC 215, Los Angeles, CA 90033. E-mail: goran@usc.edu.

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Abbreviations: AIR, acute insulin response; DEXA, dual-energy X-ray absorptiometry; DI, disposition index; FSIVGT, frequently sampled intravenous glucose tolerance; GDM, gestational diabetes mellitus; IGT, impaired glucose tolerance; MRI, magnetic resonance imaging; OGTT, oral glucose tolerance test; SI, insulin sensitivity.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances;

tion may be an additional risk factor for the development of pediatric type 2 diabetes. Despite these alarming findings in overweight Latino youth, the effects of breastfeeding on type 2 diabetes risk factors in this population have not been examined. Morever, no study has examined the protective effects of breastfeeding on type 2 diabetes risk factors using precise measures of SI, e.g., frequently sampled intravenous glucose tolerance (FSIVGT), in prepubescent children or across pubertal transitions.

Therefore, the purpose of this study was to assess the relation between breastfeeding, adiposity, and type 2 diabetes risk factors using precise measures (i.e., DEXA, MRI, and FSIVGT tests) at baseline (Tanner pubertal stage 1) and across puberty transition in overweight Latino youth with a positive family history of type 2 diabetes. We hypothesized that breastfeeding for longer durations would have protective effects on baseline and changes in adiposity and type 2 diabetes risk factors in prepubescent Latino children and across maturation.

RESEARCH DESIGN AND METHODS

Subjects

The design, data collection procedures, and findings of the University of Southern California longitudinal SOLAR (Study of Latino Adolescents at Risk for Diabetes) has been described in detail elsewhere (19,22), but neither analysis of breastfeeding status nor its relationship with adiposity and type 2 diabetes risk factors has been published. Although we intend to follow this cohort throughout puberty (i.e., up to 18 years of age), for the present study, complete longitudinal data are only available for the first 3 years. From the 240 participants (8–13 years of age) at year 1, 170 participants were measured at year 2 and 150 participants at year 3. Physical characteristics (i.e., age, weight, height, BMI, BMI percentile) are available for the entire sample in all three yearly visits, body composition (i.e., total fat mass, total lean tissue) and fasting and 2-h glucose data are available for >85% of the original sample at all visits, and fat distribution (i.e., subcutaneous and visceral fat stores) and insulin dynamics are available for >70% of the original sample at all visits. Children were categorized as follows: *1*) 8–13 years of age, *2*) BMI ≥85th percentile for age and sex based on the Centers for Disease Control and Prevention guidelines (23), *3*) Latino ancestry (all four grandparents were of Latino origin as determined by parental self-report), and *4*) family history of type 2 diabetes in at least one parent, sibling, or grandparent. None of the children were taking medications known to affect body composition, had syndromes or diseases known to affect body composition or fat distribution, or had any major illness since birth. The institutional review board of the University of Southern California approved this study. Informed consent and assent were obtained from both the parents and the child before testing commenced. All procedures were carried out at the University of Southern California General Clinical Research Center.

Breastfeeding status

A detailed medical history and physical examination was performed by a licensed pediatric care provider. Tanner pubertal staging was based on breast stage in girls and pubic hair stage in boys (24,25). Parental interview at year 1 detailed the family history of diabetes, gestational diabetes mellitus ([GDM] of mother related to child in this study), child's birth weight, whether the mother breastfed, and length of breastfeeding. Breastfeeding status was analyzed as both a continuous variable and as a categorical variable (i.e., never breastfed, breastfed 0–5.99 months, breastfed 6–11.99 months, and breastfed ≥12 months).

Body composition and fat distribution

Height and weight were measured using a beam medical scale and wall-mounted stadiometer to the nearest 0.1 kg and 0.1 cm, respectively, and the average of the two measurements was used for analysis. BMI and BMI percentiles for age and sex were determined using EpiInfo 2000, version 1.1 (Centers for Disease Control and Prevention, Atlanta, GA). Body composition (total fat mass and total lean tissue mass) was assessed with a total body DEXA scan, using a Hologic QDR 4500W (Hologic, Bedford, MA). Visceral and subcutaneous abdominal fat were measured by MRI at the Los Angeles County/University of Southern California Imaging Science Center using a 1.5 Signal LX-Ecospeed I.5 Tesla magnet (General Electric) and a single slice at the level of the umbilicus.

Insulin and glucose dynamics

After an overnight fast, a 2-h oral glucose tolerance test (OGTT) was conducted using a dose of 1.75 g glucose/kg body wt (to a maximum of 75 g). Blood was sampled and assayed for glucose and insulin at −5 min (fasting state) and 120 min (2 h) relative to glucose ingestion. The OGTT results were used to determine normal glucose tolerance (2-h glucose <140 mg/dl) or impaired glucose tolerance ([IGT] 2-h glucose ≥140 and <200 mg/dl) using criteria proposed by the American Diabetes Association (18).

Approximately 1–2 weeks after the OGTT visit, children were admitted for an overnight visit for determination of body composition and insulin dynamics, i.e., SI, insulin area under the curve (acute insulin response [AIR]), and the disposition index ([DI] a product of AIR and SI and a measure of pancreatic β-cell function). The children were served dinner and an evening snack. Only water and noncaloric and noncaffeinated beverages were allowed between 8:00 P.M. and the time of the testing the following morning, when an FSIVGT test was performed to determine SI, AIR, and DI. A topical anesthetic (EMLA cream; Aztrozeneca, Wilmington, DE) was applied to the antecubital area of both arms, and an hour later a flexible intravenous catheter was inserted into both arms. Two fasting blood samples, at −15 and −5 min, were obtained for determination of basal glucose and insulin values. At time zero, glucose (25% dextrose; 0.3 g/kg body wt) was administered intravenously. Blood samples were then collected at the following time points: 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 min. Insulin (0.02 units/kg body wt) (Humulin R [regular insulin for human subjects]; Eli Lilly, Indianapolis, IN) was injected intravenously at 20 min. Plasma was analyzed for glucose and insulin, and values were entered into the Minmod Millennium 2003 computer program (version 5.16; Richard N. Bergman, University of Southern California) for determination of SI, AIR, and DI.

Glucose from the OGTT was analyzed on a Dimension Clinical Chemistry system using an in vitro hexokinase method (Dade Behring, Deerfield, IL). Blood samples from the FSIVGT test were centrifuged immediately to obtain plasma, and aliquots were frozen at −70°C until assayed. Glucose was assayed in duplicate on a Yellow Springs Instrument 2700 Analyzer (Yellow Springs Instrument; Yellow Springs, OH) using the glucose

oxidase method. Insulin was assayed in duplicate using a specific human insulin ELISA kit from Linco (St. Charles, MO).

Statistical procedures

Dependent variables that were not normally distributed, i.e., BMI percentile, total fat, total lean tissue mass, visceral and subcutaneous abdominal fat, SI, DI, and AIR, were log transformed. χ^2 was used to assess differences in descriptive categorical data (i.e., sex, GDM), and ANOVA was used to assess differences in descriptive continuous data (i.e., birth weight and age) at baseline (i.e., Tanner pubertal stage 1) between breastfeeding categories. Linear mixed-effects modeling was used to evaluate the effects of breastfeeding at baseline and on longitudinal changes in total fat, total lean tissue, percent body fat, visceral and subcutaneous abdominal fat, fasting and 2-h glucose, and insulin dynamics (SI, AIR, and DI) over puberty transition from Tanner pubertal stage 1 to 5. Breastfeeding status was analyzed as both a continuous variable and as a categorical variable (i.e., never breastfed, breastfed 0–5.99 months, breastfed 6–11.99 months, and breastfed ≥12 months). Visual examinations of the dependent variables (transformed and nontransformed) over Tanner stage were explored to allow for the possibility of nonlinear effects. If nonlinear, than a quadratic Tanner stage was included in the model. The following covariates were entered into the model: birth weight, GDM, total body fat and lean tissue mass, changes in body fat and lean tissue mass (when adiposity and type 2 diabetes risk factors were dependent variables, change in body composition was defined as the absolute difference between the first and last waves of measurement) and SI (when AIR was a dependent variable only). The model was run with and without entering baseline and changes in body composition as covariates. By modeling across Tanner pubertal stage, we were able to test whether breastfeeding status affects adiposity and type 2 diabetes risk factors at prepubescent stages (i.e., Tanner stage 1) and across pubertal transition (transition from Tanner stage 1 to 5). When using linear mixed-effects modeling, results included an intercept level (i.e., Tanner stage 1) of the dependent variable, a rate of change over Tanner stages in the dependent variable, and an interaction effect of Tanner stage and breastfeeding category on the dependent variable. In addition, the moderator effect of GDM and birth weight on the relationship between breastfeeding and the outcome variable at baseline and across puberty was entered into the model (i.e., GDM and body weight × breastfeeding status × Tanner). The stratified moderator effect with breastfeeding and outcomes variables within different levels of birth weight (divided by median split) and GDM (yes vs. no) was also entered into the model. For all models, Tanner was centered at baseline Tanner 1 and dependent variables were centered at their mean values for ease of interpretation. All analyses were performed using SPSS version 13.0 (SPSS, Chicago, IL), with significance set at $P < 0.05$.

RESULTS — For year 1 ($n = 240$), the percentage of participants in Tanner stages 1, 2, 3, 4, and 5 were 41, 28, 9, 13, and 9%, respectively. For year 2 ($n = 170$), the percentage of participants in Tanner stages 1, 2, 3, 4, and 5 were 22, 28, 11, 23, and 16%. For year 3 ($n = 150$) they were 12, 23, 13, 25, and 27%. Physical and metabolic characteristics of breastfeeding categories (i.e., subjects who were never breastfed, breastfed 0–5.99 months, breastfed 6–11.99 months, and breastfed ≥12 months) at Tanner pubertal stage 1 are displayed in Table 1. The presence of GDM was significantly different between breastfeeding categories ($P < 0.05$). Visual examinations of the dependent variables (transformed and nontransformed) over Tanner stages were all linear, except for transformed AIR; therefore, the quadratic Tanner stage was included in the model when AIR was the dependent variable. Using linear mixed-effects modeling with covariates GDM, birth weight, and sex, there were no significant effects of breastfeeding on general adiposity (i.e., BMI, BMI percentile, total fat, total lean tissue mass, percent body fat), body fat distribution (i.e., visceral or subcutaneous adipose tissue), fasting or 2-h glucose, or insulin dynamics (i.e., SI, DI, AIR) at Tanner pubertal stage 1 or on longitudinal changes in these variables across pubertal (i.e., Tanner stage) transitions ($P \geq 0.20$; data not shown). These results remained nonsignificant once baseline and changes in body composition parameters were entered into the model as covariates. When examining the moderating effect of GDM and birth weight, we found that there was a significant interaction effect of birth weight and breastfeeding status on DI and AIR across puberty ($P < 0.05$), although not at baseline. There was also a significant interaction of GDM and breastfeeding status on subcutaneous abdominal tissue at baseline and across puberty ($P < 0.05$). However, the stratified analyses with GDM (yes vs. no GDM status) and with birth weight (divided by a median split) on the relationship between breastfeeding and the outcome variable did not produce differential effects.

We also ran the analyses with a variety of different breastfeeding variables: breastfeeding as a continuous variable, a dichotomous breastfeeding category (i.e., never breastfed vs. ever breastfed), shorter duration of breastfeeding (i.e., breastfed 0–2.99 months and 3–5.99 months), and a combination of breastfed 6–11.99 months and ≥12 months into one category. For all the different breastfeeding variables assessed, the same nonsignificant findings persisted ($P \geq 0.20$; data not shown).

CONCLUSIONS — The major findings of this study indicated that there are no significant overall protective effects of breastfeeding on general adiposity, fat distribution, or type 2 diabetes risk factors in overweight Latino youth with a positive family history of type 2 diabetes. Few studies have examined the relationship of breastfeeding and general adiposity using DEXA (11,12), and none have assessed the relationship between breastfeeding and fat distribution using MRI. To our knowledge, this is the first study to examine the relationship between breastfeeding status and insulin dynamics using precise measures of SI. We are also the first to look at the protective effects of breastfeeding on longitudinal changes in adiposity and type 2 diabetes risk factors across maturation stages.

Although numerous studies have examined the influence of breastfeeding on type 1 diabetes (26,27), few have looked at the association between breastfeeding and type 2 diabetes (14,16). Most notably, the study by Pettitt et al. (16) on 720 Pima Indians 10–39 years of age found that exclusive breastfeeding for at least 2 months was associated with a lower rate of type 2 diabetes, as measured by OGTT. Young et al. (14) conducted a case-control study of 138 native Canadian children 10–17 years of age and determined that prolonged breastfeeding (>6 months) was a strong protector against the risk of type 2 diabetes, as determined with American Diabetes Association fasting blood glucose criteria. Two other studies

Table 1—*Physical and metabolic characteristics of breastfeeding categories at Tanner pubertal stage 1 at all visits (baseline)*

	Never breastfed	Breastfed 0–5.99 months	Breastfed 6–11.99 months	Breastfed ≥12 months
n (total)	64	47	8	35
Male sex	48 (75.0)	39 (83.0)	8 (100)	26 (74.3)
GDM	23 (35.9)	6 (12.8)	1 (12.5)	12 (34.3)
Birth weight (kg)	3.6 ± 0.8	3.6 ± 1.1	3.5 ± 0.8	3.6 ± 0.6
Age (years)	9.9 ± 1.4	10.4 ± 1.7	10.3 ± 1.5	10.5 ± 1.1
BMI (kg/m^2)	26.4 ± 4.4	26.5 ± 4.9	24.1 ± 4.2	28.7 ± 4.5
BMI percentile	97.1 ± 3.4	97.3 ± 2.2	94.6 ± 4.5	97.9 ± 2.9
n	61	45	8	30
Total fat (kg)	20.9 ± 7.3	21.0 ± 9.4	16.6 ± 7.3	25.3 ± 7.5
Total lean tissue mass (kg)	31.0 ± 7.3	32.4 ± 8.0	30.2 ± 4.4	35.1 ± 6.6
n	53	33	7	24
Subcutaneous abdominal fat (cm^2)	290.6 ± 102.1	314.6 ± 179.6	236.8 ± 122.3	316.7 ± 97.9
Visceral abdominal fat (cm^2)	45.8 ± 20.4	44.1 ± 18.1	33.6 ± 15.4	51.8 ± 20.4
n	61	47	8	(*n* = 32)
Fasting glucose (μU/ml)	88.3 ± 5.5	89.4 ± 3.9	86.6 ± 4.7	91.0 ± 5.6
2-h glucose (μU/ml)	124.5 ± 16.2	119.4 ± 16.6	115.9 ± 14.0	123.7 ± 18.3
n	57	9	7	27
SI ($\times 10^{-4}$ min^{-1}/μU/ml)	2.5 ± 1.3	2.4 ± 1.9	4.6 ± 2.3	1.9 ± 1.0
AIR (μU/ml × 10 min)	1,703.5 ± 1,351.1	1,994.8 ± 1,179.5	949.9 ± 943.6	1,711.0 ± 943.6
DI ($\times 10^{-4}$ /min^{-1})	3,112.0 ± 1,178.1	3,371.6 ± 1,639.4	3,188.7 ± 1,236.0	2,605.7 ± 941.4

Data are *n*, *n* (%), and mean ± SD. Statistical analyses were performed using log-transformed data (i.e., BMI percentile, total fat, total lean tissue mass, visceral, subcutaneous adipose tissue, SI, DI, and AIR), but data are shown as nontransformed values for ease of interpretation. Differences in sex and GDM status were assessed using χ^2, while ANOVA assessed differences in birth weight and age between breastfeeding groups. GDM status was significantly different between breastfeeding groups ($P < 0.05$). Using mixed modeling after adjusting for covariates (i.e., GDM, age, sex, and body composition) as appropriate and AIR (with dependent variable SI only), there were no significant differences in adiposity or metabolic parameters between breastfeeding categories at Tanner pubertal stage 1 or in change in these variables across maturation.

have shown an inverse association between breastfeeding and fasting glucose levels in young children (28) and 2-h glucose in adults (15). In two large prospective cohorts of young and middle-aged women, duration of lactation was inversely related with the risk of type 2 diabetes, independent of other diabetes risk factors including BMI, diet, exercise, and smoking status (29). To date, no study has assessed the protective effects of breastfeeding on type 2 diabetes risk factors using precise measures of SI, e.g., FSIVGT test. In addition, many of these studies have controlled for more crude measures of adiposity, such as weight or BMI (14,16,29). Even though we ran the analyses with and without controlling for total fat and total lean tissue, which we have previously shown (30,31) to influence glucose and insulin dynamics, one could argue that excess weight or adiposity is the mechanism in which breastfeeding affects type 2 diabetes risk factors; this suggests that investigators should examine the effects that breastfeeding has on both obesity and type 2 diabetes risk factors with and without controlling for adiposity. In this study, breastfeeding was not associated with type 2 diabetes risk factors, as measured with OGTT or an FSIVGT test, with and without controlling for adiposity measures at baseline or across pubertal transitions.

Whether breastfeeding protects against childhood obesity remains controversial, but there appears to be more research supporting the inverse association of breastfeeding status and length of breastfeeding with childhood obesity (1,4,5). A very large meta-analysis of 61 studies showed that initial breastfeeding protects against obesity, as measured by BMI later in life (5). Twenty-eight of these studies showed that breastfeeding was associated with a reduced risk of obesity compared with formula feeding (odds ratio 0.87 [95% CI 0.85–0.89]). However, in our study, breastfeeding was not protective against childhood obesity using BMI measures.

To date, many investigators have used skinfolds to assess adiposity, and few studies have evaluated the relationship of breastfeeding status to more precise measures of adiposity. A study of 563 New Zealand children followed from birth to 7 years of age showed no significant differences in body fatness (i.e., skinfolds) between infants exclusively breastfed and formula fed for the first 3 months of life (32). A study by Tulldahl et al. (11) found that 781 Swedish adolescents who were exclusively breastfed for >3 months tended to have lower skinfolds than those who were not breastfed or breastfed as infants for <3 months, but there was no significant difference in body fat measured by DEXA in a subgroup of these adolescents ($n = 194$) between breastfeeding categories (11). A recent study by Burdette et al. (12) showed that breastfeeding was not associated with adiposity as measured with DEXA in 313 children at age 5 years. In this study, breastfeeding status was neither associated with total fat, percent body fat, or total lean tissue mass as measured by DEXA or with fat distribution as measured by MRI.

In addition, a longer duration of breastfeeding also appears to be protective against childhood obesity (33,34). Most notably, von Kries et al. (33) found a clear dose response for the duration of breastfeeding on the prevalence of obesity in German children, aged 5–6 years; the prevalence was 3.8% for 2 months of exclusive breastfeeding, 2.3% for 3–5 months, 1.7% for 6–12 months, and 0.8% for >12 months. In this study,

the duration of breastfeeding was not related to a significant reduction in general adiposity or fat distribution. Other breastfeeding duration categories were assessed, i.e., never breastfed versus ever breastfed, 0–2.99 months, and 3–5.99 months, but no association to adiposity measures were noted.

Only three previous studies have examined the effects of breastfeeding on obesity among Latinos, and two of them found no association between breastfeeding and childhood obesity. Grummer-Strawn et al. (10) found that duration of breastfeeding showed a protective relationship with the risk of overweight only among non-Hispanic whites, whereas length of breastfeeding was not protective against risk of overweight in non-Hispanic African Americans or Hispanics. Zive et al. (6) found that neither breastfeeding status nor the duration of breastfeeding was correlated with indicators of adiposity (i.e., sum of seven skinfolds) in 331 (43% white and 57% Mexican American) children 3 or 4 years of age; however, obesity was not common in this sample. In contrast, a study by Hediger et al. (9) of 2,685 children, 3–5 years of age and ~30% Mexican-American, showed that subjects who were breastfed had a reduced risk of being at risk of overweight (85–94th BMI percentiles) compared with never-breastfed subjects, but there was no reduced risk of being overweight (≥95th BMI percentile) between breastfeeding categories. These results indicate that ethnic disparities may exist in the relationship between breastfeeding and obesity protection.

Contrary to our hypotheses, we did not note any significant relations between breastfeeding and adiposity or type 2 diabetes risk factors at prepubescent stages or throughout puberty. One explanation for the null findings is that breastfeeding rates are lower in overweight and obese mothers (9), suggesting that familial dietary habits and activity patterns and genetics may supersede breastfeeding as a risk factor for childhood obesity and type 2 diabetes. The socioeconomic status also plays a role in promoting childhood obesity (35); however, all participants in this study are from low-income areas. Another explanation for the null findings is that our sample is very homogeneous due to the strict inclusion criteria, and more differences in adiposity and type 2 diabetes risk factors between breastfeeding groups may have been apparent if normal-weight Latino children or other ethnicities were included. However, we have shown that the degree of overweight and fat distribution (i.e., visceral fat measured with MRI) is not a predictor of IGT and that IGT is more associated with poor β-cell function and insulin resistance (22). Thus, within this population of overweight Latino children with a family history of type 2 diabetes, some are more insulin resistant and more likely to develop pre-diabetes while others are not. In this cohort, we wanted to explore which environmental factors, i.e., in terms of breastfeeding status, are influencing adiposity and type 2 diabetes risk factors. The homogeneity of this particular sample gives us the unique opportunity to assess factors influencing progression of type 2 diabetes, independent of large variations in adiposity, in a very at-risk population.

Several limitations of our study should be considered. Breastfeeding information was obtained retrospectively from the mother at the year 1 visit, which would have been 8–13 years after the birth of the child, and is therefore subject to recall bias. However, other investigators have shown maternal recall of infant-feeding data to be a valid and reliable measure (36,37), even after 20 years (38). Another limitation is that we did not collect child-feeding data, such as introduction of solids and concurrent formula feeding. This supplementation of solids or formula may negate or mask the potential beneficial effects of the breastfeeding. Yet, some researchers have shown that these child-feeding factors are not or only marginally related to childhood obesity (4,9,12) and that breastfeeding appears to play a more integral role. Another limitation is the moderating effect of GDM and birth weight on certain outcome variables at baseline and across puberty. The significant moderating effects of GDM and birth weight could have altered the relationship between breastfeeding and certain outcome variables (i.e., DI, AIR, and subcutaneous visceral fat). However, when we ran the stratified analyses with GDM (yes vs. no GDM status) and birth weight (divided by a median split), we found that breastfeeding did not have a differential effect on the outcomes variables; thus we feel that including GDM and birth weight as covariates in our analyses is acceptable. We also did not collect the rate of weight gain during infancy. An additional limitation is that the current study used a relatively small sample size of children (n = 240) compared with prior epidemiological studies. When breastfeeding was broken down into categories, there were only eight subjects who had been breastfed 6–11.99 months. However, when breastfeeding was run as a continuous variable and as shorter and longer duration categories, identical nonsignificant results were found. The limitation of a small sample size is somewhat offset by use of precise measures of body composition (DEXA and MRI), as well as glucose and insulin dynamics (OGTT and FSIVGT test), control of various covariates, and the longitudinal design.

In conclusion, to our knowledge, this is the first study to examine the influence of breastfeeding on detailed and precise measures of body composition, fat distribution, and insulin dynamics in overweight Latino youth at prepubescent stages and longitudinal changes across puberty. Even though we did not observe any protective effects of breastfeeding on measures of adiposity and type 2 diabetes, continued longitudinal analyses using more precise and valid measures are warranted, especially in populations that are extremely susceptible to becoming obese and developing type 2 diabetes.

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