Breastfeeding as Obesity Prevention in the United States: A Sibling Difference Model

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ABSTRACT In light of the growing prevalence of obesity in the United States, and the health risks associated with childhood obesity in particular, it is critical to identify avenues for obesity prevention. This study tests the hypothesis that breastfeeding serves as one protective factor against children’s subsequent development of obesity. We used linear-, logistic-, and sibling fixed-effects regression models to evaluate the association between infant feeding history and body mass index (BMI) in late childhood or adolescence (9–19 years, mean = 14 years). Complete data were available for 976 participants (488 sibling pairs) in the 2002 Child Development Supplement of the Panel Study of Income Dynamics, a nationally representative survey of families in the United States. In sibling pairs in which only one sibling was breastfed, the breastfed sibling had an adolescent BMI that was 0.39 standard deviations lower than his or her sibling, controlling for child-specific factors that may have influenced parents’ feeding decisions. This effect is equivalent to a difference of more than 13 pounds for a 14-year-old child of average height. Furthermore, fixed-effects logistic regressions predicting overweight and obese status showed that breastfed siblings were less likely to reach those BMI thresholds. We therefore conclude that breastfeeding in infancy may be an important protective factor against the development of obesity in the United States. The application of a sibling fixed-effects model provides stronger evidence of a causal relationship than prior research reporting similar patterns of association. Am. J. Hum. Biol. 22:291–296, 2010. © 2009 Wiley-Liss, Inc.

Obesity across the life course has become a major public health concern. Focusing on adolescence, recent estimates in the United States classify more than 15% of the adolescent population as “obese” and more than one-third as “overweight” (Ogden et al., 2008). Early overweight and obesity may lead to later health problems, such as hypertension, type II diabetes, and obesity in adulthood (Bao et al., 1995; Freedman et al., 2001). Weight problems also affect near-term quality of life by presenting psychosocial and cardiovascular risks (Ogden et al., 2002). It is, therefore, critical for obesity prevention efforts to identify and target the factors involved in the early development of obesity (Koplan et al., 2005). In that vein, the present study tests the hypothesis that breastfeeding in infancy plays a protective role against the development of obesity in childhood and adolescence.

A substantial body of research has demonstrated a link between breastfeeding and specific health outcomes for both infants and mothers. Summarizing this research in their policy statement on the practice of breastfeeding, the American Academy of Pediatrics suggests a variety of health benefits, including, but not limited to protection against diarrhea, lower respiratory infections, and blood infections in infants (American Academy of Pediatrics, 2005). There is also evidence to suggest benefits to infants’ cognitive development and protective effects against type II diabetes and SIDS (Bernshaw, 1991; Caspi et al., 2007). For nursing mothers, immediate benefits include the stimulation of oxytocin production, which results in decreased postpartum bleeding and more rapid return of the uterus to its prepregnancy size (Chua et al., 1994). Furthermore, breastfeeding may aid with postpartum weight loss and reduce the risk of ovarian cancer and premenopausal breast cancers (Labock, 2001). Needless to say, breastfeeding is widely regarded as the most health-promoting form of infant feeding. Can prevention of obesity be added to this list of benefits of breastfeeding?

Recent meta-analyses have suggested that the adjusted odds ratio (AOR) of becoming obese after having been breastfed ranges from 0.78 to 0.93, controlling for other risk factors (Arenz et al., 2004; Owen et al., 2005). A third meta-analysis suggests a dose-dependent effect, with a longer duration of breastfeeding associated with healthier body weight later in life (Harder et al., 2005). However, these meta-analyses utilize samples across numerous countries, raising the question as to whether breastfeeding plays a protective role against obesity in the particularly “obesogenic” context of the United States (Swinburn et al., 1999). Recent studies in the United States present somewhat discrepant results. One study associated breastfeeding with overweight, but not obesity, in early childhood (Hediger et al., 2001), while another reported a statistically significant AOR of 0.78 for overweight in adolescents who had been “mostly or exclusively” breastfed (Gillman et al., 2001). More recently, Michels et al. (2007) examined breastfeeding and body mass index (BMI) outcomes in adult women who were breastfed as infants, finding no significant effects.

A major limitation of the research summarized above is its reliance on standard ordinary least squares (OLS) or logistic regression models. Though these studies differ in the extent to which they control for potential confounding factors, there always remains the possibility of an omitted variable driving both mothers’ feeding choices and children’s later body weight outcomes. The present study is the one of the first to use a sibling difference model to test the hypothesis that

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breastfeeding in infancy reduces the risk of obesity in childhood and adolescence. As hard-to-measure characteristics of mothers may affect both breastfeeding and child health, it is difficult to estimate the effects of breastfeeding without bias. Sibling difference models, a form of fixed-effects modeling, use differences between siblings as the dependent and independent variables in an OLS or logistic regression (Duncan et al., 2004). Any characteristics, observable or unobservable, that are shared by siblings are differenced out of the model. A sibling fixed-effects model is particularly useful for obesity research because it implicitly controls for some of the major predictors of childhood obesity, which happen to be shared by siblings: parental obesity, household income, and family eating habits (Whitaker et al., 1997). The model also implicitly removes the influence of any characteristics that are shared by siblings and implicated in the complex processes by which families decide how to feed their infant, such as race and ethnicity, socioeconomic status, and maternal education.

**METHODS**

**Sample**

The data for this study come from the Panel Study of Income Dynamics (PSID), a longitudinal, nationally representative survey of US families that began in 1968. We drew on Waves 1 and 2 of the PSID's Child Development Supplement (CDS), in which rich developmental data were collected on children in 1997 and 2002, respectively. Additionally, certain measures of maternal characteristics were collected on children in 1997 and 2002, respectively. Certain measures of maternal characteristics were drawn from the general PSID survey and linked to specific children via the caregivers' identifiers.

In Wave 1, 2,394 families participated in the CDS, with families reporting on either one or two children per family (N = 3,563 children). Children's ages ranged from age 0 to 13 years in Wave 1. In Wave 2, 5 years later, the sample size dropped to 2,019 families (N = 2,907 children) due to attrition. Children without a sibling in the sample were dropped from the family fixed-effects analysis, which requires sibling differences, bringing the analytic sample size to 1,857. Furthermore, due to the interest in adolescence and the relative instability of BMI at younger ages, children born after 1993 were dropped from analysis. This left a final analytic sample of 976 children (488 sibling pairs) aged 9 through 19 (mean = 13.7 years), with available data on both breastfeeding history and BMI at Wave 2.

**Analytic strategy**

In spite of the use of statistical controls, the possibility remains that an omitted variable drives parents' decisions to breastfeed one sibling and not the other. If this omitted variable also affects children's BMI in adolescence, then the model has not successfully isolated the effects of breastfeeding. Accordingly, the present analyses begin with descriptive statistics that compare breastfed and never-breastfed children using both the full sample and the sample of differently fed (“discordant”) sibling pairs. Following work in economics by Joseph Altonji et al., we assumed that if selection into breastfeeding were not based on observable characteristics, it would be less likely to be based on unobservable characteristics (Altonji et al., 2005). In other words, observed similarities between differently fed siblings would further substantiate the fixed-effects approach.

Next, we ran a set of regressions predicting a continuous measure of BMI. Standard OLS regressions were performed first on the entire age-eligible sample and then on the sibling sample, in order to check the comparability of the two samples. We then ran a sibling fixed-effects regression, controlling only for characteristics that may differ between siblings. For each regression model, we also report the bivariate relationship, without controls, between breastfeeding and BMI, in order to examine how this association might then be attenuated by the set of statistical controls.

Finally, in the interest of predicting overweight and obesity in addition to the continuous measure of BMI, we replicated the previous models, but using logistic regression to predict dichotomous measures of BMI set at the 50th, 85th, and 95th percentiles (standardized by age and gender).

**BMI and breastfeeding measurement**

In Wave 2 of the CDS, children's weight and height were measured directly by the interviewer. In the rare instances in which the child refused to be measured, the primary caregiver reported the child's height and weight from the time of the most recent doctor visit. The PSID reported BMI as kg/m² units, which were converted to age- and sex-specific z-scores based on the 1978 norms from the CDC, using the Stata program “zanthro” (Vidmar et al., 2004).

Children's primary caregivers—biological mothers for 92% of the participants—were asked whether their children were "ever breastfed." These data were collected retrospectively at both Wave 1 and Wave 2, and those cases with data at both waves show high reliability across time (95.5% agreement). The present analyses use the breastfeeding responses from Wave 1.

**Additional covariates**

For the fixed-effects models, covariates were chosen in an attempt to model the selection factors that might lead a family to breastfeed one sibling but not the other. The first additional covariate was a dummy code representing whether or not a child was born premature, defined as less than 37 weeks of gestation. Children born early are less likely to be breastfed (Callen et al., 2001; Hediger et al., 2001). Data on gestational age at birth were not missing on any case within the 488 pairs of siblings.

The second additional covariate was a measure of the biological mother's employment status preceding her child's birth. This covariate was included to account for the fact that breastfeeding requires a substantial commitment on the part of mothers; women who expected to return to work after the birth of a child were hypothesized to be less likely to initiate breastfeeding (Chatterji and Frick, 2005). The maternal employment variable was created using data from the general PSID from 1983 through 1993, incorporating the values of the maternal employment status from the year before a given child was born. The year prior to birth was used to ensure that a mother's decision to work was not endogenous to her decision regarding breastfeeding. As with gestational age,
data on maternal employment at birth were complete for all cases.

The remaining covariates all included some amount of missing data, which was addressed using multiple imputation with Stata’s ICE procedure (m = 5 imputed data-sets). The third covariate represented whether or not the mother was married or cohabitating with a partner in the year the child was born, which has been associated with a greater likelihood of breastfeeding (Gibson-Davis and Brooks-Gunn, 2006). This variable was missing for 54 cases. The fourth covariate was teen birth, computed as the child’s birth year minus the mother’s birth year, recoded as “1” for ages 13 through 19. Giving birth prior to age 20 has been identified elsewhere as a crucial cutpoint in rates of breastfeeding (Centers for Disease Control and Prevention, 2008). As with the previous covariate, about 6% of the analytic sample was missing data on the mother’s age at birth.

Dummy variables for race/ethnicity and family income-to-needs ratio were included in the first OLS and logistic models, but not the fixed-effects models, as these characteristics did not differ between siblings. Conversely, only the fixed-effects models included a dummy variable representing whether or not the child was the older child within the sibling pair.

The descriptive statistics and standard OLS and logistic models utilize the child-level sample weights provided by the PSID, due to the fact that the individual child was the unit of analysis in those models. However, all of the weighted fixed-effects models employ the PSID’s household-level sample weights. The standard errors for all of the standard (not fixed-effects) OLS models are Huber-White adjusted for clustering within families.

It is important to note the limitations of the fixed-effects models as currently specified. First, for a variety of reasons—including not having a sibling in the study, sample attrition, missing data, and age ineligibility—the original sample size of 3,563 children at Wave 1 was reduced to 976 for the fixed effects analyses. Though sample weights were employed, the reductions compromised the power and generalizability of the sample. Second, the PSID does not ask whether “breastfed” children were exclusively breastfed, as opposed to being fed a combination of breastmilk and formula. It also does not ask about mothers’ use of breast pumps. Some researchers emphasize the importance of exclusive breastfeeding for later health benefits (Gillman et al., 2001), but those effects cannot be tested using these data. Third, the dependent variable of BMI does not provide information regarding the location of fat on the body. There is evidence suggesting that central adiposity confers the greatest risk in terms of development of the metabolic syndrome (Okosun et al., 2000), but the relatively blunt instrument of BMI cannot speak to this issue. Fourth and finally, though selection bias is arguably reduced by the use of fixed effects, its potential is not eliminated. Why would a mother breastfeed one child but not the other? The fixed-effects results are unbiased only if unmeasured child-specific determinants of breastfeeding are uncorrelated with child body mass.

### RESULTS

**Descriptives**

The descriptive statistics in Table 1 show that in a national sample of more than 3,500 children (“Full Sample”), breastfed and never-breastfed children differ far beyond how they were first fed. On average, children who were not breastfed had lower birth weights, were more likely to be premature, and were slightly more likely to be male compared to children who were breastfed. Mothers who did not breastfeed were more likely to have been a teenager at the time of the child’s birth, and their household income relative to family size was lower than that of breastfeeding mothers. Additionally, these mothers were less likely to be working or to be married or cohabiting with a partner at the child’s birth. When examining the full sample, all of these differences were statistically significant (P < 0.001). These descriptive statistics utilized the child-level sample weights from the PSID, such that the results estimate a national sample of children ages 9–19 in 2002.

Comparing the children in the discordant pairs of the sibling sample (N = 118), breastfed and never-breastfed children appear much more similar to one another. In this subsample, none of the previously mentioned differences (e.g., birth weight, maternal marital/cohabitation status) is statistically significant. Furthermore, the magnitude of the difference between breastfed and never-breastfed children is almost always reduced in the sample of discordant pairs, when compared with differences within the full sample. The one exception is maternal employment: Among the differently fed pairs, 59% of the breastfed siblings’ mothers had worked prior to the birth.

**TABLE 1. Child-specific characteristics by feeding history**

<table>
<thead>
<tr>
<th>Child characteristics</th>
<th>Breastfed</th>
<th>Never breastfed</th>
<th>P-value for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI z-score, W2</td>
<td>.46</td>
<td>.64</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Birthweight (lbs.)</td>
<td>7.12</td>
<td>6.78</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Premature</td>
<td>9%</td>
<td>13%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Female</td>
<td>51%</td>
<td>48%</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Maternal characteristics at time of child’s birth</th>
<th>Breastfed</th>
<th>Never breastfed</th>
<th>P-value for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under age 20</td>
<td>5%</td>
<td>11%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Employed</td>
<td>48%</td>
<td>39%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Married or cohabiting</td>
<td>65%</td>
<td>44%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Income-to-needs ratio</td>
<td>3.66</td>
<td>2.34</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

| N        | 1,570 | 1,968 |

Values are taken from Wave 1 data, unless otherwise specified. BMI = body mass index; W2 = Wave 2 of the Child Development Supplement.
compared with 47% of never-breastfed siblings. Although this difference is not statistically significant, it is larger in magnitude than the difference observed in the full sample. Overall, though, the mothers in the sibling sample did not tend to choose to breastfeed based on the observable characteristics that differed between siblings, adding support for the use of a fixed-effects model (Altonji et al., 2005).

Table 2 provides additional information on the discordant pairs in the sibling subsample, who drive the fixed-effects estimates of the effects of breastfeeding. In terms of gender composition, roughly half of the discordant pairs were male-female, a quarter were male-male, and a quarter female-female. Among the male-female pairs, the breastfed sibling was male exactly 50% of the time. Table 2 also shows that breastfed and nonbreastfed children were roughly the same age, on average, at the time the outcome was measured. Furthermore, there was no significant difference in BMI z-score according to birth order. Despite these apparent similarities between older and younger siblings, all fixed-effects models nonetheless control for birth order.

**OLS and logistic models**

The models without fixed effects predicted both continuous BMI (Table 3) and three specific BMI thresholds (see Fig. 1). Consistent with prior research, OLS and logistic models without fixed effects demonstrated a relatively small, negative association between breastfeeding and subsequent BMI when controlling for some of the potential confounding variables. When examining the continuous measure of BMI, OLS models suggest that breastfeeding is associated with a BMI difference of less than 0.2 standard deviations in the hypothesized direction ($P < 0.05$ for the full sample and $ns$ for the sibling sample; Table 3, Columns 1–4). Notably, the magnitude of these estimates changed very little when run on the full CDS sample with complete feeding and BMI data ($n = 2,591$) versus the smaller sibling sample that was used for the fixed-effects analyses ($n = 976$). This consistency of results, together with the similarity in descriptive statistics between the two samples, suggested that there were not glaring differences between the sibling sample and the full sample.

The logistic regressions without fixed effects (see Fig. 1) suggested that the effects observed in the OLS models were driven by the upper end of the BMI distribution. When predicting the 85th percentile, breastfeeding was associated with an AOR of 0.59 ($P < 0.01$), suggesting that breastfed infants are 41% less likely than nonbreastfed infants to become overweight or obese. Although the AOR for breastfeeding was not statistically significant when predicting obesity (the 95th percentile), it was less than the null value (AOR = 0.69, $ns$), following the hypothesized trend. Also as hypothesized, the model predicting the 50th percentile produced an AOR for breastfeeding that was noticeably closer to the null value (AOR = 1.03, $ns$).

**Fixed-effects models**

The fixed-effects results in Table 3 (Column 6) are appreciably larger than the previous OLS results. The predictor of interest—the practice of breastfeeding was associated with a four-tenths of a standard deviation decrease in BMI net of the other predictors ($P < 0.05$). For a 14 year old of average height, this effect amounts to a difference of 13.49 pounds for a girl or 14.06 pounds for a boy (Centers for Disease Control and Prevention, 2005).

Among the control variables, premature birth was also a strong predictor of later body composition, associated with a 0.45 decrease in BMI z-score ($P < 0.05$). We conducted sensitivity analyses (not shown) to examine the effect of including either prematurity or a continuous measure of birth weight in the fixed-effects model. We found that the presence of either prematurity or birth weight in the model did not attenuate the association between breastfeeding and BMI, but rather increased the coefficient from 0.35 to 0.40. We also ran a fixed-effects model interacting prematurity and breastfeeding, but the coefficient on the interaction was small and not statistically significant, suggesting that the effects

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**Table 2. Additional characteristics of differently fed sibling pairs**

<table>
<thead>
<tr>
<th>Gender composition</th>
<th>%</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both male</td>
<td>25.4%</td>
<td></td>
</tr>
<tr>
<td>Both female</td>
<td>27.1%</td>
<td></td>
</tr>
<tr>
<td>One male, one female</td>
<td>47.5%</td>
<td></td>
</tr>
<tr>
<td><strong>BMI z-scores at Wave 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Older sibling</td>
<td>0.59 (0.14)</td>
<td></td>
</tr>
<tr>
<td>Younger sibling</td>
<td>0.61 (0.14)</td>
<td></td>
</tr>
<tr>
<td>Age at Wave 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breastfed sibling</td>
<td>13.57 (2.79)</td>
<td></td>
</tr>
<tr>
<td>Never-breastfed sibling</td>
<td>13.25 (2.39)</td>
<td></td>
</tr>
<tr>
<td>Age difference between siblings</td>
<td>1.53 (0.69)</td>
<td></td>
</tr>
</tbody>
</table>

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**Table 3. OLS and fixed effects models of breastfeeding and BMI**

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>(1) OLS, all adolescents</th>
<th>(2) OLS, all adolescents</th>
<th>(3) OLS, sibling sample</th>
<th>(4) OLS, sibling sample</th>
<th>(5) Fixed-effects, sibling sample</th>
<th>(6) Fixed-effects, sibling sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breastfed</td>
<td>$-0.166^{**}$ (0.059)</td>
<td>$-0.150^{**}$ (0.065)</td>
<td>$-0.190^{**}$ (0.095)</td>
<td>$-0.152$ (0.106)</td>
<td>$-0.340^{**}$ (0.177)</td>
<td>$-0.397^{**}$ (0.176)</td>
</tr>
<tr>
<td>Premature</td>
<td>$-0.109$ (0.112)</td>
<td>$-0.252$ (0.156)</td>
<td>$-0.063$ (0.101)</td>
<td>$-0.156$ (0.129)</td>
<td>$-0.467^{**}$ (0.227)</td>
<td>$-0.672^{**}$ (0.246)</td>
</tr>
<tr>
<td>Employed</td>
<td>$-0.052$ (0.070)</td>
<td>$-0.049$ (0.131)</td>
<td>$-0.002$ (0.237)</td>
<td>$-0.216$ (0.228)</td>
<td>$-0.095$ (0.122)</td>
<td>$-0.000$ (0.257)</td>
</tr>
<tr>
<td>Married/cohabiting</td>
<td>$-0.171^{**}$ (0.083)</td>
<td>$-0.163$ (0.131)</td>
<td>$-0.002$ (0.237)</td>
<td>$-0.216$ (0.228)</td>
<td>$-0.171^{**}$ (0.083)</td>
<td>$-0.002$ (0.237)</td>
</tr>
<tr>
<td>Teen mother</td>
<td>$-0.085$ (0.122)</td>
<td>$-0.049$ (0.131)</td>
<td>$-0.002$ (0.237)</td>
<td>$-0.216$ (0.228)</td>
<td>$-0.085$ (0.122)</td>
<td>$-0.002$ (0.237)</td>
</tr>
<tr>
<td>Older sibling</td>
<td>$-0.171^{**}$ (0.083)</td>
<td>$-0.163$ (0.131)</td>
<td>$-0.002$ (0.237)</td>
<td>$-0.216$ (0.228)</td>
<td>$-0.171^{**}$ (0.083)</td>
<td>$-0.002$ (0.237)</td>
</tr>
<tr>
<td>Child age &amp; gender</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Race &amp; family income</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>N</td>
<td>2591</td>
<td>2591</td>
<td>976</td>
<td>976</td>
<td>976</td>
<td>976</td>
</tr>
<tr>
<td>N, pairs</td>
<td>488</td>
<td>488</td>
<td>488</td>
<td>488</td>
<td>488</td>
<td>488</td>
</tr>
<tr>
<td>N, differently-fed pairs</td>
<td>59</td>
<td>59</td>
<td>59</td>
<td>59</td>
<td>59</td>
<td>59</td>
</tr>
</tbody>
</table>

* $^{**} P < 0.01$, $^{*} P < 0.05$, $P < 0.10$.
of breastfeeding and birth weight (or prematurity) are operating independently.

Finally, Figure 1 presents the AORs from fixed-effects logistic models in which the 50th, 85th, and 95th percentile scores are used as the dependent variables. As with the standard logistic models, the observed association of breastfeeding and BMI appears at the top end of the distribution, this time achieving statistical significance at both the 85th and 95th percentile thresholds. It is important to note that these models require differences in both feeding history and BMI status, which effectively reduces the sample sizes to between 30 and 44 for the fixed-effects logistic models. So while these coefficients should be interpreted with caution, it is notable that statistically significant effects still emerge, despite the decreased power resulting from the smaller sample sizes.

**DISCUSSION**

This set of analyses tests the association of breastfeeding with the development of overweight and obesity in childhood and adolescence, including models controlling for fixed factors shared by siblings (such as parental obesity), as well as some of the selection factors that might lead a mother to breastfeed one child but not the other (premature birth, recent employment status, and being a teen parent). With these controls in place, children who were breastfed were four-tenths of a standard deviation thinner, on average, than their counterparts who were not breastfed. To put this effect into more concrete terms, the difference would amount to 13.5 pounds for a 14-year-old girl or 14.0 pounds for a 14-year-old boy (Centers for Disease Control and Prevention, 2008). Furthermore, the logistic regressions suggest that breastfeeding specifically reduces the risk of reaching the upper end of the BMI distribution.

These findings support prior literature demonstrating statistically significant effects when looking at outcomes in adolescence (Gillman et al., 2001). However, by controlling for family-level fixed effects this model reduces the likelihood that an unobserved variable is driving the effect, thus strengthening the evidence for a causal relationship. These fixed-effects estimates account for all of the controls in the OLS models with the additional control for unmeasured characteristics shared by siblings, such as the biological characteristics of their mothers. The only other published study using sibling fixed effects to model breastfeeding and BMI found no significant effects associated with breastfeeding (Evenhouse and Reilly, 2005). However, that study did not control for maternal characteristics specific to each sibling’s birth year (e.g., maternal age and marital status at the birth, and employment status prior to the birth). Because those characteristics have shown associations with mothers’ feeding decisions, the fixed-effects estimates reported here provide a more unbiased estimate of the breastfeeding-BMI relationship than previous models.

In terms of biological mechanism, there are numerous hypotheses as to why breastfeeding might confer BMI benefits long after infancy. Some of these hypotheses emphasize the establishment of behavioral patterns, while others emphasize physiological programming effects. In terms of behavioral mechanisms, breastfeeding may promote a feeding style that is more responsive to infant hunger and satiety, setting children on a course to better regulate their own appetites at later ages (Fisher et al., 2000; Taveras et al., 2006). In terms of physiological programming, there are numerous differences between breast milk and infant formula in terms of nutrients and hormones (Hamosh, 2001). For instance, overall protein content is much higher in infant formula and the hormone leptin is found only in breast milk. Difference in early intake of these components potentially could lead to longer term effects via the programming of metabolic systems by changing the set points of appetite or metabolism (Locke, 2002; Singhal and Lanigan, 2007).

In closing, the most immediate causes of the childhood obesity epidemic are well-understood: children in the United States typically eat energy-dense diets and do not enjoy enough exercise. Public health efforts must continue to work on these fronts. However, if breastfeeding is implicated in the initial organization of children’s metabolic systems, then breastfeeding in infancy might protect children by setting them on positive trajectories with respect to appetite regulation. If children are better at registering the feeling of “fullness,” they will be better equipped to establish the healthy habits promoted by public health efforts.

Breastfeeding is widely regarded as the most beneficial form of early nutrition. Prior research has suggested that a healthier body composition might be added to the list of breastfeeding’s benefits, with hypothesized biological mechanisms emerging from an actively growing body of research. By employing the rigorous test of sibling fixed effects, this study adds to the positive evidence base on this question. We therefore conclude that breastfeeding may be characterized as a means of obesity prevention in the context of the United States. A number of approaches have been pursued in order to support mothers who wish to breastfeed, including state and federal legislation, supports in the workplace, public health campaigns, and...
improvements to the WIC program (Jacknowitz, 2006). The findings of this study provide additional support for the continued implementation and evaluation of policy levers such as these.

**LITERATURE CITED**


