Modest Protective Effects of Breast-feeding on Obesity

Is the Evidence Truly Supportive?

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The consensus often reported is that there is a modest but significant protective effect of breast-feeding on obesity risk. Numerous investigators have reported that “breast-feeding reduces childhood adiposity.” This article provides a brief review of the existing literature on breast-feeding and obesity, highlighting a number of factors that may have led to overstatement of positive effects. These include publication bias, confounding variables, and lack of long-term effects on obesity risk. Although the literature does not support the existence of a protective effect of breast-feeding, a definitive conclusion of no protective effect of breast-feeding on obesity is premature, and more research is needed. Nutr Today. 2012;47(1):33–38

Beneficial associations of breast-feeding with health outcomes in children are well documented.1-3 The observation of an association between breast-feeding practices and weight gain trajectories dates to the early 1900s.4 However, the substantial increase in obesity among children has fueled interests to better understand and identify the potential contribution of breast-feeding to adiposity development. Over the past 3 decades, numerous evidence-based reports, editorials, and recommendations have focused on the association between breast-feeding and reduced adiposity in the offspring. Although the conclusions drawn from the published literature vary, it is often reported that there is a modest, but significant, protective effect of breast-feeding on obesity risk. For example, numerous investigators have reported that breast-feeding results in reductions in childhood adiposity,5-7 and Buyken et al8 suggested that “breast-feeding can offset the effects of maternal obesity.” However, careful inspection of supporting publications suggests that both the strength of the relationship and the evidence regarding the relationship between breast-feeding and obesity outcomes may be exaggerated.9 This article provides a brief review of the existing literature on breast-feeding and obesity, highlighting a number of factors that may have led to distorted conclusions. The development of obesity may be influenced by early childhood factors, including those related to diet. Optimization of infant growth and development is dependent on adequate amounts of essential nutrients and energy during the critical periods of rapid growth postnatally.8,10,11

As early as 1904, it was suggested that providing both human and cow’s milk to an infant contributed to excessive caloric intake, diminishing the beneficial effects of breast-feeding.4 In fact, Budin and Plachon4 suggested that overfeeding was more detrimental than underfeeding, although they did not specifically focus their attention on obesity itself. Recommendations were made to limit mixed (defined by Budin and Plachon as breast plus cow’s milk) feeding and encourage exclusive breast-feeding for optimal growth and beneficial health outcomes.4 Although the study by Kramer et al12 is often cited as the impetus for bringing this association to the forefront, it was in the early 1970s that researchers first reported formal analyses (eg, scientific studies with outcome evaluation) suggesting an inverse association between breast-feeding and obesity.13 Subsequently, many studies have analyzed this potential association. There are 2 main issues drawing attention by these early studies: initiation of breast-feeding itself and the timing of the introduction of mixed (formula in conjunction with breast milk) and/or complementary (breast milk plus solid foods) feeding. It may be that the association between breast-feeding and obesity risk is related to the timing of complementary feeding.14-16 Another issue is that the inadequacy of breast milk production may be the impetus for the introduction of complementary feeding for the prevention of growth faltering.1,4,11,17 Beyond the more associated link between initiation of breast-feeding and obesity risk, studies are slightly more consistent in suggesting a potential relationship between mixed introduction and obesity risk. Future research is
warranted because if a protective effect truly exists, it would have considerable public health importance. However, determination of the effect of breast-feeding independent of early introduction of mixed feeding or a potential later introduction of solid feeding will require a sophisticated study design.

The preponderance of the evidence suggesting breast-feeding’s protective effect against obesity risk stems from epidemiological observational studies that have evaluated the contribution of breast-feeding initiation and/or duration to overweight in early childhood and in adolescence.

EFFICACY VERSUS EFFECTIVENESS

One difficulty in analyzing these observational studies lies in the distinction between 2 quite different concepts: “efficacy” and “effectiveness.” Efficacy is the degree of desired effect produced by a given intervention implemented with fidelity (ie, a tightly controlled experiment), and effectiveness is the degree of success of intervention implementation in a realistic setting. Breast-feeding would be deemed efficacious when it proves superior to formula feeding (eg, fewer obese children) in terms of obesity prevention. Breast-feeding would be viewed as effective if efforts to increase the initiation or duration of breast-feeding in real life (ie, an educational program to increase breast-feeding) resulted in reduced risk of obesity. In general, testing the efficacy of breast-feeding (relative to formula-feeding) aims to establish a causal relationship. Investigating effectiveness aims to assess the usefulness of such advice in routine clinical practice. The distinction is clinically relevant. Efficacy studies may show that protective effects of breast-feeding on obesity/weight gain may vary according to contexts (eg, whether and when complementary feedings are introduced). Therefore, it may be wise to establish the efficacy of breast-feeding before attempting to assess its effectiveness.

PUBLICATION BIAS

Imagine for a moment that there is a small association between 2 variables, X and Y, in most populations and that people conduct many studies estimating and testing this association, each on a separate random sample of people from these populations with small associations. Imagine further that because of differences in sample size and possibly other factors, studies vary widely in precision of estimation and statistical power; some have very low power, and some have very high power (Figure). In such a situation, the sample estimates of the association, such as an odds ratio, all have small expected values, namely, the true odds ratios in the populations from which the samples are drawn. Of course, no single study will obtain exactly the true population odds ratio because of chance variations, but the bigger and the more powerful the study is, the closer its estimate of the true odds ratio can be expected to be on average. In other words, the sample estimates from the more powerful studies are more precise (ie, have small variances), whereas the sample estimates from less powerful studies will be less precise (more variable with larger variances). Now imagine further that when investigators conduct a study and obtain a statistically significant result, they always publish that study, but when they do not obtain a statistically significant result, they rarely do. What will happen if this is the case? The studies with very high power (probability) to detect even a very small association will often obtain statistically significant results and thus tend to be published regardless of the magnitude of association. In contrast, the low-power studies will obtain...
Publication bias is known to occur in many fields, and methods have been developed to test for it. The hypothetical situation in the preceding paragraph would produce a characteristic signature in the meta-analytic data such that there was an association between the precision or power of studies and their estimates of the odds ratio, with less powerful and precise studies producing evidence odds ratios indicating stronger associations and larger studies producing estimates of odds ratios indicating weaker associations. The authors of the World Health Organization (WHO) Report\(^1\) (as well as other authors\(^{28,29}\)) appropriately tested for evidence of publication bias in the literature on breast-feeding and found exactly this characteristic signature, strongly suggesting publication bias. If one examines the plots presented and extrapolates to the hypothetical studies that would produce unbiased results (ie, those with perfect power and precision), the plausible estimates one obtains simply by eye are remarkably close to zero. This finding further suggests that meta-analyses of published studies of the association between breast-feeding and obesity are likely to produce exaggerated estimates of the magnitude of the association. The WHO document states that because a statistically significant protective association was observed among those studies that controlled for confounders such as socioeconomic status (SES) and parental anthropometry, with sample sizes of 500 to 1500 as well as sample sizes greater than 1500, the protective “effect of breast-feeding on obesity was not likely to be due to publication bias or confounding,” yet this argument misses the mark because it does not take the publication bias that they themselves identified into account.

If we do take publication bias into account, the estimated association between breast-feeding and offspring obesity, even in childhood, is arguably very small, but not necessarily equal to zero. A well-established line of reasoning in the field of epidemiology is that when confounding is plausible in observational epidemiological studies, as is the case with breast-feeding and obesity, it is then quite plausible that a small observed association is due to confounding (see further discussion in next section) and therefore represents a zero real effect. This is because it is quite plausible that a small observed association could have been produced by an unrecognized and uncontrolled confounding variable, whereas a very large association could only be due to confounding by one or more uncontrolled confounding variables collectively of very large effect, and it is less plausible that such confounding variables of large effect went overlooked. This was pointed out most eloquently by Sir Austin Bradford Hill in his classic paper\(^30\) enumerating guidelines for assessing when an association is likely to represent causation and in fact is what he lists as his first guideline.

Given the above, identification of multiple large and powerful data sets in which within-family analyses could be conducted would arguably provide an opportunity for obtaining association estimates that could in turn serve as less biased estimates of causal effects.\(^{28}\) That being said, given that a randomized (not observational) study with more than 13,000 subjects did not detect a protective effect of breast-feeding on obesity suggests that, if any effect does exist, it is likely to be small indeed.

Curiously, despite the documented evidence of publication bias, major health organizations including WHO, American Academy of Pediatrics (2003), American Dietetic Association,\(^2\) and the Centers for Disease Control\(^{31}\) continue to support the promotion of breast-feeding for its purported “modest” effects in the protection from obesity later in life based on the apparently biased observational literature.

**MIGHT THE RELATIONSHIP BE MEDIATED BY CONFOUNDING VARIABLES?**

The finding of publication bias suggests that the true association between breast-feeding and later obesity may be much smaller than one would estimate by relying on the published literature. But is the association, if there is one, actually indicative of a causal relationship? We have all heard that correlation does not imply causation. This is because of the possibility of confounding.

Confounding exists when 2 variables are associated, but one does not necessarily cause the other. Perhaps the most commonly considered confounding situation involves 2 variables (eg, breast-feeding and obesity) being correlated because they are both influenced by a third variable. Are there variables that seem to have a causal effect (albeit perhaps through other intermediary variables) with both offspring obesity and breast-feeding? The answer is a clear yes. Notwithstanding, even when controlling for confounders, it must be recognized that (1) not all confounders can be measured well, (2) statistical modeling techniques for the inclusion of confounding may not function adequately, and (3) there is a relative impossibility of identifying and including all potential confounders in any study. Accordingly, controlling for confounders does not lead to cause-and-effect relationships but does improve our ability to establish a meaningful, statistically valid connection between the breast-feeding and obesity risk.
SOME POTENTIAL CONFOUNDERS

Mothers who choose to breast-feed are typically of higher SES, are more educated, and have a greater support system than those who do not breast-feed.32 These factors are also often related to healthier lifestyle habits (eg, healthier diet and increased physical activity levels) and lower prevalence of maternal obesity. In addition to the consistent reported observation indicating that maternal obesity is associated with child weight status, which could result in obesity in later life, women who are overweight or obese before pregnancy are less likely to initiate breast-feeding.14,33–35 Overweight/obese women also discontinue breast-feeding earlier than do normal-weight women.14,33–35 Furthermore, Buyken et al8 observed no relationship between breast-feeding and childhood adiposity levels among offspring of normal-weight mothers; however, overweight mothers who breast-fed for less than 2 months were more likely to have overweight children, at least among boys. Similarly, Baker and colleagues33 also observed that infant weight gain was associated with maternal prepregnancy body mass index (BMI) and with an interaction between the duration of breast-feeding and the timing of complementary food introduction.

Although most authors recognize the possibility of such confounding and in some cases try to include potentially confounding factors such as birth weight, maternal BMI, SES, sex, and smoking status, maternal behavioral factors are almost entirely neglected in analyses. Beyond the type of food an infant receives, mother-child social interactions also play a major role in current and future weight trajectory.33,36–38 Recent psychobiological research suggests that parental control rather than the infants’ ability to self-regulate intake may be driving differences (ie, mothers may encourage overconsumption of bottles because of their ability to visually assess and monitor intake).39,40 In addition, the prenatal environment has been shown to affect metabolism and appetite-regulating pathways, particularly in terms of maternal glucose control.33 Importantly, this is a limitation of all observational epidemiological research and not just that concerned with breast-feeding, no observational study can control for all potential confounding variables. They can control only for those variables of which the investigator can conceive, measure, and effectively statistically model.

Sibling Studies as an Attempt at Stronger Control of Confounding

One way to try to further decrease the effect of confounding variables is to conduct discordant sibling analyses (ie, siblings who have similar postnatal experiences, but vary in their obesity status). Although this does not completely eliminate the possibility of confounding, studying sibling pairs, who presumably experience many of the same confounding maternal factors but vary in the duration or initiation of breast-feeding, should radically reduce it. Findings from these studies have been equivocal.

Gillman et al41 analyzed 5614 sibling pairs and observed that, among siblings discordant for duration of breast-feeding, there was a protective association against overweight in adolescence with a longer duration of breast-feeding. Specifically, it was reported that the adjusted odds ratio for overweight among siblings with longer breast-feeding duration, compared with shorter duration, was 0.92, but it was not statistically significant (95% confidence interval, 0.76–1.11). Metzger and McDade3 reported that breast-fed siblings were less likely to reach BMI thresholds for overweight \((P < .05)\) and obesity \((P < .01)\) than their non-breast-fed siblings. Similarly, in a sibling analysis drawn from the Helsinki Birth Cohort, 84% of which had been breast-fed on average for 6 months, O’Tierney et al42 reported that longer periods of breast-feeding were associated with lower BMI at 1 year of age \((P < .05)\); however, this association with BMI had largely vanished and was not statistically significant by the age of 7 years \((P = .50)\). Furthermore, in this cohort, among siblings whose heights and weights had been measured, those who had been breast-fed for less than 2 months or greater than 8 months had the highest BMI and percentage body fat. In contrast, Nelson and colleagues41 reported that discordant breast-feeding of sibling pairs did not predict BMI \(Z\) score differences or discordant overweight status. Thus, the authors suggest that the effect of breast-feeding on overweight may be induced by confounding reflective of characteristics of mothers or families that who to breast-feed. Interestingly, the study by Evenhouse and Reilly43 observed that the breast-fed sibling is more likely to be overweight \((P < .10)\). Sibling analyses that control for many maternal variables (SES, employment status, teen mother, maternal BMI, smoking, race) have not generally produced supportive results. Although sibling analyses provide the capacity to control for effects of some of the maternal confounding factors, they do not account for child confounders (eg, prematurity, birth weight, suckling behavior) that may be related to the discordance in duration or initiation of breast-feeding. Clearly, no observational epidemiological study is capable of removing all confounding. The only method that can control for both known and unknown confounding variables is randomization.

Randomized Controlled Trials

In contrast to the observational epidemiological studies available, in a hypothetical world, the ideal way to determine a causal effect of breast-feeding on body fat outcomes independent of other factors is to randomly assign mother-child dyads into exclusive breast-feeding implemented with perfect fidelity or to no breast-feeding whatsoever. For obvious reasons, such trials would be impractical and unethical. However, one can randomly assign mothers to

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programs designed to promote breast-feeding and observe the effects of such assignment on adiposity. We know of only 1 such randomized controlled trial (RCT), an RCT conducted by Kramer et al. In this large RCT of a breast-feeding promotion intervention in the Republic of Belarus, no demonstrable beneficial (or adverse) long-term effects were observed on child adiposity up to 6.5 years of age. On the one hand, this study is disheartening in suggesting that encouraging women to breast-feed had no apparent effect on offspring adiposity. Yet it must be acknowledged that it is unclear whether this study shows that breast-feeding per se is ineffective in reducing childhood obesity or merely that attempting to get mothers to breast-feed is ineffective in reducing childhood obesity. Although the literature spans the continuum from weak, unadjusted observational epidemiological studies to randomized clinical trials, the majority of the “evidence” is based on observational, epidemiological studies. Breast-feeding practices are affected by biological and nonbiological factors that might confound the relationship between breast-feeding and childhood obesity.

CONCLUSIONS

Long-term obesity risk can be influenced by events in early postnatal life, and it is plausible that breast-feeding could play a key role in metabolic programming during this time. However, despite several decades of study, the actual empirical evidence supporting such an effect is weak at best, making it difficult to defend any strong statements about the benefits of breast-feeding on obesity. Although not discounting breast-feeding and its numerous health benefits, the mainstream discussions of clinical and public health approaches suggesting that encouraging breast-feeding may be a practical intervention for the prevention of childhood obesity may be misleading. The small odds ratio in the studies indicating a relationship could plausibly be explained not by a true effect but by publication bias or confounders. Furthermore, there is no convincing evidence of long-term effects of breast-feeding on obesity risk. That said, although the literature does not support the existence of a protective effect of breast-feeding against obesity, likewise, a definitive conclusion of no protective effect of breast-feeding on obesity is also premature.

REFERENCES

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