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Does Breastfeeding Protect Against Childhood Obesity? Moving Beyond Observational Evidence

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Abstract Human milk is the optimal feeding choice for infants, as it dynamically provides the nutrients, immunity support, and other bioactive factors needed for infants at specific stages during development. Observational studies and several meta-analyses have suggested that breastfeeding is protective against development of obesity in childhood and beyond. However, these findings are not without significant controversy. This review includes an overview of observational findings to date, then focuses on three specific pathways that connect human milk and infant physiology: maternal obesity, microbiome development in the infant, and the development of taste preference and diet quality. Each of these pathways involves complex interactions between mother and infant, includes both biologic and non-biologic factors, and may have both direct and indirect effects on obesity risk in the offspring. This type of integrated approach to examining breastfeeding and childhood obesity is necessary to advance research in this area beyond observational findings.

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Introduction

Obesity is a major public health concern, with over one third of adults in the USA considered obese (with a body mass index, BMI, ≥30), and a growing number of children and adolescents considered obese during the past 30 years [1, 2]. Because treatment of established obesity is notoriously difficult, research has increasingly turned to potential prevention strategies. Evidence supports intervention during the earliest years of life to prevent obesity and obesogenic lifestyle habits, such as low physical activity, overeating, and poor diet quality. Increasingly, research has focused on infancy as a critical period for the development of these lifestyle habits, and infant feeding practices have been identified as one potentially modifiable aspect that could alter obesity risk.

The health benefits of breastfeeding over infant formula feeding are well-recognized, including protection from respiratory and diarrheal illness, optimal growth, and longer-term impacts such as higher IQ, lower cardiovascular risk factors, and lower obesity rates as the children get older [3], with some effects spanning decades after weaning. This review will focus on one of these relationships, that between breastfeeding and reduced risk of obesity later in life. Establishing this relationship between breastfeeding and obesity protection has been notably difficult for many reasons, including imprecise recall of breastfeeding timing and intensity, incomplete accounting for covariates that may affect both the decision to breastfeed and obesity propensity, and long intervals between the exposure (human milk or breastfeeding) and outcome (obesity development).

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Existing Observational Evidence

To date, six systematic reviews and meta-analyses have been conducted to examine the relationships between breastfeeding and obesity risk or protection (Table 1) [4••, 5–9]. Together, these meta-analyses have included results from 81 individual studies, and each has concluded that the odds of developing obesity is reduced between 12–24 % (odds ratios from 0.76 to 0.88) in those who were breastfed as infants compared to those who were not. Longer duration of breastfeeding is associated with greater reduction of the odds of obesity development (estimated as a 4 % decreased odds of obesity per additional month of breastfeeding) [8]. Many of these results were attenuated when adjusting for a variety of covariates, but remained significant.

On the surface, these results would appear to settle the question of whether breastfeeding is associated with protection from obesity. However, the debate continues for many reasons. First, at least one of the meta-analyses has been criticized for analytic decisions that may bias interpretation [10]. Also, several, but not all, of the meta-analyses found evidence of significant publication bias in the literature [4••, 6, 7], with positive studies more likely to be published, while negative ones are apparently missing. In addition, there are a number of potentially confounding relationships in the observational studies that were incompletely accounted for, resulting in heterogeneity of effects across studies [4••, 5–7]. Thus, in the face of significant confounding, researchers have noted that observational studies may never be able to definitively resolve the question of whether breastfeeding is *directly* associated with lower adiposity [11].

While individual assignment of mothers to breastfeeding versus formula feeding is not ethically possible, other study designs have been pursued to reduce confounding. The Promotion of Breastfeeding Intervention Trial (PROBIT) [12] was a cluster-based intervention conducted in 1996–1997, which randomized hospitals to standard maternal care or enhanced promotion of breastfeeding. The intervention group did achieve higher overall and exclusive breastfeeding (43.3 % vs. 6.4 % exclusive breastfeeding at 3 months for intervention and control groups, respectively, $p<0.001$), but the proportions of obesity did not differ between groups by age 6.5 years (5.9 and 5.0 %, respectively) [13] or at age 11.5 years [14]. Interestingly, this study compared discordant breastfeeding patterns (weaned at <1 month versus exclusive breastfeeding >6 months), and found that prolonged exclusive breastfeeding was associated with *higher* BMI and triceps skinfold thickness at 6.5 years [13], adjusting for a variety of cluster- and individual-level covariates. This study has been widely cited as evidence against a direct role of breastfeeding in childhood obesity development. However, this study has also been criticized for studying a population with a low overall prevalence of obesity and for primarily comparing obesity between the two intervention arms where over 60 % of both groups were breastfed to some degree at 3 months [12], limiting the power.

Other strategies to reduce major confounding have included studies of siblings who were breastfed to different degrees. Such studies have the benefit of eliminating the effect of genetics and reducing the effect of measured and unmeasured household factors, such as education, income, and potentially, maternal obesity. Six such studies have been published to date [15–20], with just one [18] noting evidence for a within-family effect of breastfeeding on obesity, adjusting for several covariates. Unfortunately, most of these studies used imprecise measures of breastfeeding duration, with little additional information about why siblings experienced discordant breastfeeding. In addition, only the small proportion of families (10–20 %) where breastfeeding experience differed between siblings was informative, making the generalizability unclear.

Biologic Models for how Breastfeeding May Associate with Childhood Obesity

The remainder of this review focuses on three potential biologic pathways between breastfeeding and human milk composition and risk for obesity later in life: the role of maternal obesity, development of the infant gut microbiome, and development of taste preferences and dietary quality. Research in

Table 1 Meta-analyses of breastfeeding and obesity development

First author, year [Reference number]	Number articles included, years	Odds ratio for overweight/obese	Adjusted odds ratio for overweight/obese	Evidence of effect heterogeneity	Evidence of publication bias
Horta BL, 2013 [4••]	71, <2011	0.76 [0.71, 0.81]	0.81 [0.75, 0.88]	Yes	Yes (Egger, $p=0.003$)
Weng SF, 2012 [5]	10, 2003–2006	NR	0.85 [0.74, 0.99]	Yes ($I^2=73.3$ %)	No (asymmetry test, $p=0.25$)
Horta B, 2007 [6]	33, <2006	0.78 [0.72, 0.84]	NR	Yes	Yes
Owen CG, 2005 [7]	28, <2004	0.87 [0.85–0.89]	0.93 [0.88, 0.99]	Yes (χ^2, $p<0.001$)	Mixed (Egger, $p<0.001$; Begg, $p=0.96$)
Harder T, 2005 [8]	17, <2004	0.96 [0.94, 0.98] per month of BF	NR	NR	No (Egger, $p=0.77$, and Begg, $p=0.64$)
Arenz S, 2004 [9]	9, <2004	0.78 [0.71, 0.85]	NR	No (Q test, $p>0.1$)	No (asymmetry test, $p=0.71$)

NR not reported

these areas may help refine our understanding of how breastfeeding or human milk may be biologically related to infant growth and development.

Maternal Obesity

Maternal obesity is frequently considered only as a confounder in the relationship between breastfeeding and childhood obesity. For example, a genetic link between maternal obesity and increased risk of childhood obesity has long been recognized [21–24]. In addition, offspring of obese women are often exposed to greater levels of glucose and insulin *in utero*, resulting in infant insulin resistance and hypothalamic and epigenetic changes [25–31]. Maternal obesity is also associated with lower breastfeeding rates [24, 32, 33], although intention to breastfeed does not differ significantly by group [34]. Thus, obesity in breastfed offspring may be confounded by maternal obesity. Attributing the relationship to confounding alone assumes that there are no biological differences between obese and lean mothers that may also alter the breastfeeding–obesity relationship.

However, there is likely a biologic basis for differences in breastfeeding behavior, as obese mothers have been shown to have delayed lactogenesis [35] and reduced lactation performance compared with normal-weight mothers [36]. In addition, the prolactin response to suckling is less in the first postpartum week in obese compared to normal weight mothers [37], and obese women may also experience greater physical problems in achieving successful infant latching to the breast [38]. Taken together, these results support a biologic role of maternal obesity in breastfeeding itself.

Furthermore, for obese mothers who successfully breastfeed their children, breastfeeding may be particularly important in modulating their child's obesity risk. In one study, infants of obese mothers who were breastfed >17 weeks experienced growth trajectories similar to infants of normal-weight mothers, while non-breastfed infants had accelerated BMI gains by mid-childhood. By contrast, breastfeeding had limited impact on growth trajectories in children of normal-weight mothers [39]. A second study also noted that lack of breastfeeding was most detrimental among children of overweight or obese mothers [40]. This is consistent with a recent study which showed that the impact of breastfeeding is significantly protective only at the higher levels of BMI [41]. It is possible that differences in milk composition in obese mothers may play a role. Indeed higher human milk leptin and insulin are associated with slower infant growth [42], and administration of leptin during suckling in mice reduces food intake and overweight in adulthood [43], suggesting it acts as a protective factor in infancy. Higher milk adiponectin is associated with slower infant growth during the first year [44], but increased growth in the second year of life [45, 46]. These findings, taken together, suggest that the relationship between breastfeeding and child obesity may differ between obese and normal-weight mothers. Other studies, reviewed below, report that the composition of human milk differs between obese and normal-weight mothers. Adjusting for maternal obesity in observational studies is likely insufficient to account for these complex relationships, but may be critical to understanding whether breastfeeding is beneficial in high-risk populations.

Gut Microbiome, Disease Protection, and Antibiotic Use

Humans as well as many other living systems are the host to a diverse species of microorganisms. These microorganisms (collectively known as the microbiome) can develop symbiotic relationships with the host and ultimately influence host health. The human gut hosts over a thousand of different bacterial species by the first year of life and these species play a major role in nutrition and metabolism [47, 48]. Not surprisingly, obesity has been associated with alterations in the gut microbiome [49–51]. Importantly, by changing the gut microbiome, energy harvest and consequentially body fat can be altered [52] thereby supporting a potential causal effect of the microbiome on obesity.

Establishment of the gut microbiome begins perinatally and continues postnatally [53], with children first exposed to the microorganisms that colonize the gut during delivery [54, 55]. After birth, diet during the first year of life is a major contributor to the gut microbiome [55]. It has long been recognized that breastfeeding is associated with differential colonization of several microorganisms in the infant gut [56, 57•] compared to formula feeding, such as a relative abundance of *Bifidobacteria* strains in breastfed infants [56]. Human milk includes a variety of bacterial species whose relative proportions change over the first 6 months of lactation and differ by maternal pre-pregnancy obesity status [58•]. Interestingly, the milk of obese mothers has been shown to have a lower concentration of *Bifidobacteria* [58•, 59], but a higher total bacterial count and higher concentration of *Staphylococcus* and *Lactobaccilis* [58•] than normal-weight women. *Bifidobacteria* administration in animal models has been shown to counteract visceral fat accumulation and improve glucose utilization in response to a high-fat diet [60]. Human milk may also alter the infant's microbiome through the action of human milk oligosaccharides and secretory antibodies [48, 61, 62]. For example, human milk oligosaccharides are a major substrate for *Bifidobacteria* in the gut, promoting their abundance [63•, 64].

In addition to this potential direct pathway between human milk and gut microbiome, an indirect pathway may also play a role. Human milk contains a variety of immune molecules, which protect the newborn while its own immune system matures. Notably, breastfed infants have lower incidence of both respiratory and diarrheal diseases [12], and breastfed

infants are 50 % less likely to be given antimicrobial treatments in the first year of life [65]. A growing body of literature has implicated the early use of antibiotics [66–69] and prenatal exposure to antibiotics [70] in increased childhood overweight and obesity. Antibiotic exposure is associated with gut gene expression, obesity and alterations in the microbiome [71••, 72, 73], particularly lowering levels of *Bifidobacteria* [64]. Importantly, these studies suggest that antibiotics act through alterations in the microbiome to influence obesity risk [71••]. Thus, human milk directly and indirectly impacts both intestinal development and the health of the intestinal microbiome in the infant, likely leading to differential utilization of food and later obesity risk. Observational studies examining breastfeeding duration are not able to account for differences in human milk quality or physiological impact in the infant, leading to oversimplified conclusions about the impact of human milk on obesity risk.

Taste Preference and Diet Quality

A third possible biological mechanism by which breastfeeding may influence the risk of later childhood obesity is through alterations in taste preferences and diet. Early food experiences set the stage for later food choices and are important in establishing life-long food habits and may ultimately affect later health [74, 75]. Indeed, being fed breast milk is associated with more acceptance of novel flavors in infancy [76, 77] and openness to healthy food options in childhood [78•]. Importantly, the flavor profile of human milk is modified by maternal diet [78•, 79], while the taste of any given formula does not change, and controlled trials demonstrate that infants exposed to specific tastes through human milk or formulas prefer those tastes later [80, 81].

This higher acceptability for novel foods may enhance infants' likelihood of establishing healthy eating habits. Breastfeeding is associated with higher dietary diversity during preschool years [82–84], and higher vegetable intake at age 4–5 [85•, 86–88], compared with formula-feeding, suggesting impacts of breastfeeding on establishment of longer-term healthy eating habits. It is important to note that mothers who decide to breastfeed are also more likely to adhere to pediatric nutrition guidelines, resulting in better complementary food choices for young children [89], including being more likely to introduce complementary foods later, less likely to give sugary drinks, and more likely to give fruits and vegetables. Maternal diet quality is also positively associated with her child's diet quality during preschool ages [83, 85•], perhaps reinforcing the effect of breastfeeding on healthy dietary patterns.

Whether or not healthy diet establishment is protective of later obesity remains surprisingly unclear, however. In general, unhealthy eating behaviors [90] and reduced dietary diversity [91, 92] have been associated with increased likelihood of obesity in cross-sectional studies. However, one study noted that although infants breastfed >16 weeks were significantly more likely to eat fruit and vegetables at age 7 than those not breastfed, this diet difference did not explain the differences in obesity between the two infant feeding groups [88]. Additional studies are needed to determine whether lower obesity rates among children who were breastfed are mediated by improved diet quality or taste preferences in childhood.

Other Factors

The present review has focused on some selected biologic mechanisms that may help explain lower obesity rates among breastfed individuals. However, a number of other biologic and non-biologic, and even confounding or effect modifying, relationships are likely to play an important role, which we will here mention briefly.

Race/ethnicity and Socioeconomic Status In the USA, race and socio economic status are two factors that are strongly associated with both obesity prevalence and likelihood of breastfeeding, in ways that likely confound even carefully-designed or carefully-controlled studies of breastfeeding. Obesity and unhealthy eating behaviors are more prevalent among African-Americans and among lower-income social strata [93–97]; African-Americans are more likely to also be low-income; and these same racial and socioeconomic groups also have a lower prevalence of breastfeeding [98–101]. Some evidence suggests low socioeconomic status may have a larger detrimental impact on breastfeeding rates among black women than other racial or ethnic groups [100, 102]. Furthermore, most research on breastfeeding is conducted among predominantly white populations, so within-race or within-low social stratum analyses are frequently underpowered. However, racial/ethnic differences in the relationship between breastfeeding and obesity development have been noted in some [103, 104] but not all [105] studies, but there remains controversy about which racial/ethnic groups may be more protected, even if true racial/ethnic differences exist. Thus, race/ethnicity and social stratum, combined with breastfeeding prevalence differences among these groups and even potential differences in the relationship between breastfeeding and obesity development, may hamper any efforts to disentangle this complicated set of relationships.

Behavioral Aspects of Breastfeeding Behavioral differences between breastfeeding and formula feeding have also been postulated to impact risk for obesity, beyond the nutritional composition of human milk versus formula. Breastfeeding mothers cannot monitor infant intake directly, and must therefore rely on infants' feeding cues (e.g., turning head away) rather than external cues (e.g., empty bottle). In mothers, early encouragement of finishing a bottle is associated with encouraging children to eat all the food on their plate in childhood

[106], and mothers who exclusively breastfed are less likely to have food restrictions at one year of age [107]. With respect to children, being breastfed is associated with increases in satiety and appetite regulation [108–111]. However, while persistent behavioral differences between breastfed and formula-fed groups are seen among both mothers and children, neither lower maternal food restriction [112] nor greater child appetite regulation [110] actually accounts for differences in obesity risk seen among breastfed children compared with formula-fed children. Taken together, these results support that breastfeeding can have long term implications for healthier feeding behaviors, but these changes may not be sufficient for reducing childhood obesity risk.

Much of the controversy surrounding breastfeeding's relationship with obesity protection relates to the difficulty in assessing the association of breastfeeding itself, independent of the factors that may lead to the mother's decision to breastfeed in the first place, and/or her decision to stop breastfeeding. Breastfeeding is not a decision that is made only once, but a behavior whose continuation is reconsidered continually throughout the course of infant feeding, based on a variety of factors that vary over time, ranging from mother or infant health, to family/social supports or expectations, to job pressures or inconvenience. Mothers also do not decide to breastfeed in a vacuum, as many mothers rely on the advice and support of their partners, parents, doctors, and peers to make and maintain a breastfeeding decision. Even among women with a desire to breastfeed, some women are not physically or socially able to start or maintain breastfeeding. Thus, additional research is needed to understand the specific nature of the biases introduced by these external and internal decision-making factors, and the extent to which each of these and other factors are applicable in higher-risk subgroups, such as obese mothers, racial/ethnic minorities, or low-income households.

Discussion

Breastfeeding has unmistakable benefits for both mothers and infants, both during infancy and beyond, and human milk is the optimal first food. Human milk is a dynamic, complex bioactive fluid that not only sustains infant nutrition, but provides a variety of important hormones, cytokines, and cells that promote the development of the infant's intestinal tract and immune system, and provide awareness of the food tastes in the mother's diet, among many other functions. Thus, the act of breastfeeding and the composition of human milk impact multiple pathways involved with infant health, growth, and development, likely impacting the risk for obesity later in life. In this review, we have shown how three biologic pathways related to breastfeeding may serve to influence later obesity.

Specifically, maternal obesity is increasingly being viewed as a biologic rather than simply a confounding factor in the relationship between breastfeeding and offspring obesity development. Many human milk components differ between obese and lean mothers, potentially offering different protection against obesity development in the context of maternal obesity. Secondly, the importance of the gut microbiome in obesity protection and factors influencing its development in infancy has been elucidated rapidly, with the promise of integrating findings from human milk studies, work in probiotics, and the impact of maternal characteristics. Thirdly, the development of infant taste preference is clearly rooted in breastfeeding and early food experiences, and is associated with healthier food choices later on. It is possible that the microbiome also influences both our preference for and utilization of specific types of foods. Surprisingly, the path from a healthy diet to a healthy weight in childhood is not as strong as expected, suggesting that there may be other factors involved, or complementary pathways between healthy diet and healthy weight that could and should be explored. These results suggest that there may be an underlying biologic basis for the protective effect of breastfeeding on later obesity identified by epidemiologic studies.

One assumption of this review is that a relationship between breastfeeding and obesity development in the offspring is real, and even plausibly causal. However, what if breastfeeding and human milk are actually not the drivers of differential obesity development in offspring? This is also possible and would shape a different set of questions relating to the relationship between breastfeeding and obesity risk. In this instance, breastfeeding behavior needs to be considered as embedded in a fabric of other positive attributes. For example in the USA, the decision to breastfeed may be a marker for mothers otherwise pursuing a healthy lifestyle, with knowledge about healthy choices, and the time, income and social support to translate that knowledge into the non-trivial choice to initiate and continue breastfeeding, in order to accrue the many known benefits for their infant's and their own health. Thus, encouraging breastfeeding among those not otherwise inclined, without improvement in maternal diet, physical activity and other healthy lifestyle factors, may have limited impact on childhood obesity, as the often-cited PROBIT study suggests [13]. Similarly, the benefits of having a healthy mother may already reduce obesity rates in the offspring, and additional effects of increasing the duration or exclusivity of breastfeeding, or improving the quality of mother's milk, may be minor. In short, we may need to look "upstream" of breastfeeding to improving maternal health habits in order to appreciably reduce obesity in children, which would benefit mothers' health as well.

Pathways toward the Future

Recently, a set of recommended next steps regarding research in lactation and neonatal nutrition was published [113••],

which we suggest as a guidepost. In that review, 11 specific research priorities were identified, several of which are directly relevant to the understanding of human milk composition (recommendation 1), infant gut development (recommendation 2), infant growth (recommendation 5), and impacts of maternal obesity (recommendation 8). In addition to these research priorities, we add some specific suggestions that pertain to the relationships between breastfeeding and human milk with obesity development.

Epidemiologic studies, to the extent that they continue to explore this relationship, should focus on refined questions in homogeneous groups of participants. It is not sufficient to continue to ask whether obesity is associated with breastfeeding. Future research should refine the question to "under what circumstances does feeding human milk affect growth patterns, or body composition, or metabolic processes in the short, medium and longer term".

Epidemiologic studies also need to significantly refine assessment of the breastfeeding behavior (the exposure) itself. Each woman who breastfeeds her infant may have a unique pattern of breastfeeding intensity (e.g., number of feeds, duration of feeds, and exclusivity of human milk) that may differ almost-daily over the course of infant feeding. These complex breastfeeding patterns are often only ascertained retrospectively and analyzed using broad categories such as "breastfed to any degree for≥[3, 4, or 6] months" versus "breastfed to any degree for<[3, 4, or 6] months." These broad categories likely result in significant within-category variation of exposures to human milk. The implications of this residual confounding may also vary across studies depending on locally-prevalent distribution of breastfeeding behaviors underlying the categorization. Ideally, epidemiologic studies of breastfeeding populations should also collect human milk samples and ascertain aspects of human milk composition that may impact infant development, further refining the concept of exposure. The lack of precision related to exposure to human milk upon which observational study results are based may help explain the widely divergent results seen in epidemiologic studies.

Beyond epidemiologic studies, cross-disciplinary research will be critical moving forward. The abundance of new information regarding human milk composition, gut microbiome ecology, and social and cultural factors begs the question of how these factors interrelate to impact adiposity development in infancy and beyond. Studies relating human milk and infant development are increasingly complex, and the potential benefit of targeting strategies for primordial obesity prevention are significant, provided we can move beyond observational evidence.

Conclusions

Because the decision to breastfeed is strongly personal and also closely linked with specific socioeconomic and lifestyle patterns, definitive epidemiologic evidence about the role of breastfeeding, or the feeding of human milk, on obesity prevention may never be truly attainable. The best observational evidence to date, compiled across over 80 separate studies conducted over at least 20 years, suggests that breastfeeding, especially for longer durations or more exclusively, is associated with a 10–20 % reduction in obesity prevalence in childhood. However, the complex nature of the relationships between breastfeeding and obesity, including the fact that human milk is variable between women and breastfeeding may have differing effects in specific population subgroups, suggests that the concept of promotion of breastfeeding as a front-line strategy for the primordial prevention of obesity is not supported by the literature.

In lieu of such a recommendation, where does the current state of the literature leave us? Research conducted recently, using methods and tools from multiple disciplines, suggests that by understanding the mechanisms of how breastfeeding and/or the composition of human milk impacts infant development, we may be able to generate a more nuanced view of the connection between breastfeeding and obesity risk. This review has focused on three potential pathways, but many other mechanisms have been suggested, and still others likely are being developed. By leveraging experimental, epidemiologic, and molecular methods, we can move beyond observational evidence to identify specific biologic pathways and refine the role of human milk in obesity prevention.

Compliance with Ethics Guidelines

Conflict of Interest Jessica G. Woo reports grants from Mead Johnson Pediatric Research Institute, Inc.

Lisa J. Martin has a patent issued on Adiponectin for Treatment of Various Disorders, Patent # 8,314,061.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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This paper provides a significant recent summary and roadmap concerning research priorities in human lactation and neonatal nutrition.