



BENEFITS OF BREASTFEEDING FOR EARLY GROWTH AND LONG TERM OBESITY: A SUMMARIZED REVIEW

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Background

Breastfeeding is one of the most important determinants of child survival, birth spacing, and prevention of childhood infections. Breastfeeding brings clear short-term benefits for child health by reducing mortality and morbidity from infectious diseases. In humans, the influence of early nutrition on long-term adiposity has focused on the possible protective role of breastfeeding. The importance of breastfeeding has been emphasized in various studies.¹ The importance of exclusive breastfeeding and the immunological and nutritional values of breast milk has been demonstrated.² The beneficial effects of breastfeeding depend on breastfeeding initiation, its duration, and the age at which the breast-fed child is weaned. Breastfeeding practices vary among different regions and communities. In India, breastfeeding in rural areas appears to be shaped by the beliefs of a community, which are further influenced by social, cultural, and economic factors. However, breast milk is the best source of nutrition for the newborn, whether breastfeeding has long-term health benefits remains controversial.³ At present mostly studies related to the same concept of breastfeeding are presented in a frame of

systematic review. This is a first review of summary designed to discuss the conclusion of the literature present on the protection of breastfeeding against later obesity and growth acceleration of a child.

Does breastfeeding protect against later obesity?

A case-control study by Kramer was one of the first studies to suggest breastfeeding protected against later obesity. Since then, as summarized recently in four systematic reviews⁴, many population-based studies have confirmed an association between breastfeeding and lower risk of later adiposity. These reviews were based on observational studies, and because of marked differences in demographic and socioeconomic characteristics between breastfed and formula-fed infants, they are unable to establish causation. Nevertheless, the association between breastfeeding and later obesity remained after adjustment for potential confounding factors. A long-term advantage of breastfeeding was further supported by a 'dose response' effect, that is, a longer duration of breastfeeding was associated with lower tendency to later obesity (for example, each month of breastfeeding was associated with a 4% reduction in obesity risk, 95% CI – 6% to – 2%). Interestingly, the definition of

breastfeeding appeared to be relevant to interpretation of data. For instance, confining the meta-analysis to studies comparing exclusive breastfeeding with exclusive formula feeding found a remarkably similar approximately 20% reduction in obesity risk in two independent systematic reviews⁵, an effect size important for populations. Exclusivity of breastfeeding may therefore be central to the mechanism by which breastfeeding protects later obesity.

Mechanisms

The potential mechanisms by which breastfeeding protects against later obesity can be broadly categorized as those that influence behaviour, and those related to the unique nutritional composition of human milk.

Behavioural explanations

One such explanation might be that breastfed children may be at less risk of later obesity because breastfeeding is more common in families that adopt healthier dietary and other lifestyle habits. Another behavioural explanation is that, because breastfed babies control the amount of milk they consume, they may learn to self-regulate their energy intake better than formula-fed babies, although whether this difference persists into adult life is unknown.

Nutritional explanations

These might include the possibility that the programming of adiposity by breastfeeding is because of a number of bioactive nutrients in human milk that are absent from some formulas (e.g. long-chain polyunsaturated fatty acids). Differences in early protein intake (up to 70% greater in formula-fed than breastfed infants could also affect later adiposity, possibly by mechanisms that involve an earlier age of adiposity rebound⁶. A higher protein intake in infancy has been suggested to promote later obesity by stimulation of insulin release and programming of higher long-term insulin concentrations. Consistent with this thesis, formula-fed infants were shown to have

higher plasma insulin concentrations than those breastfed from as early as 6 days of age⁷.

The growth acceleration hypothesis

Finally and most recently, we have suggested that the benefits of breastfeeding for long-term obesity may be due to a slower pattern of growth in breastfed compared with formula-fed infants the growth acceleration hypothesis.

This proposes that faster post-natal growth (upward centile crossing particularly in infancy) programmes several components of the metabolic syndrome, including insulin resistance, higher low-density lipoprotein cholesterol concentration, higher blood pressure and obesity⁸. Consistent with this hypothesis, acceleration in both weight and length in the first 2 weeks of life was associated with later endothelial dysfunction, a measure of the earliest physiological changes associated with the development of atherosclerosis.

The size of the effect was substantial. Adolescents with the greatest weight gain in their first 2 weeks had 4% lower flow-mediated dilatation of the brachial artery (a measure of endothelial function) than those with the lowest weight gain. This effect is similar to that of type 1 diabetes mellitus (4%) and smoking (6%) in adults⁹. Similarly, for cholesterol concentration, slower neonatal weight gain was associated with 20% lower cholesterol concentration compared with 10% lowering of cholesterol concentration associated with breastfeeding rather than formula feeding¹⁰.

Growth acceleration and later obesity

The growth acceleration concept may be particularly important for programming of obesity, as shown in two recent systematic reviews¹¹ in which upward-centile crossing for weight and length in infancy was associated with later obesity risk, with odds ratios ranging from 1.2 to 5.7¹². In fact, infant growth appears to make a major contribution to later adiposity. Stettler has estimated that 20% of the risk of obesity at age 7 years could be attributed to having a rate of weight gain in the highest

quintile in the first 4 months of life¹³. The effects of growth acceleration on later obesity are also not confined to formula-fed infants. For instance, faster weight gain in the first 2 months was associated with higher body mass index (BMI) at 10 years in a large, prospective and predominantly breastfed cohort from the UK (the Avon Longitudinal Study of Pregnancy and Childhood) [mean BMI of children who were in the highest quartile of the population for weight gain in the first two post-natal months (mean, SD: 18.6, 0.2 kg m⁻²) was greater than those in the lowest quartile (18.1, 0.1; $P = 0.01$) Charakida, personal communication]. Programming effects of faster early growth therefore appear to be applicable to diverse populations. When is the critical window? Growth is fastest in the first few weeks after birth, which, as in animal models, may therefore be a key programming window. Consistent with this hypothesis, greater weight gain in the first week of life was shown to programme obesity in adulthood¹⁴, a finding analogous to programming of insulin resistance and endothelial function by faster early growth in preterm infants. Again, the size of the effect was substantial; each 100 g increase in absolute weight gain during this period was associated with a 28% increase in the risk of becoming overweight (95% CI 8–52%). Emerging evidence therefore strongly supports the first few post-natal weeks as a critical window for programming long-term health in both humans and animals. The growth acceleration hypothesis could also explain the influence of other factors in infancy on later obesity. For example, an earlier age of adiposity rebound, suggested to be a key risk factor for later adiposity, may simply identify children whose BMI centile is high and/or crossing upwards (i.e. children with a faster growth rate)¹⁵. Similarly, previous associations between an earlier age at weaning (complementary feeding) and a greater risk of later obesity may reflect the influence of greater nutrient (and particularly protein) intake with weaning on growth rate in infancy. However, in humans and animals, it is not possible to separate the programming effects of nutrition from those of

growth as, clearly, these two factors are interdependent.

How does growth acceleration influence obesity?

Animal studies have helped shed light on the mechanisms that link early growth and nutrition with long-term obesity risk. Of particular interest is the programming of appetite and the underlying hormonal mechanisms involved¹⁶. Mechanisms favouring up-regulation of appetite may be advantageous in the short term, driving growth and improving the likelihood of survival to reproductive age. However, in the longer term, this programmed increase in appetite could contribute to obesity. In support of this hypothesis, breastfeeding has been associated with lower concentration of the appetite-regulating hormone, leptin (relative to fat mass), in adolescents¹⁷, raising the possibility that relative under nutrition and slower growth associated with breastfeeding in the first few weeks permanently programmes a lower appetite. In contrast, formula fed infants may have up-regulated appetite, which leads to obesity when faced with a highly palatable, energy-dense western diet (analogous to greater obesity in rats growing fast before weaning and fed a highly palatable ‘cafeteria’ diet after weaning).

Does breastfeeding protect against growth acceleration?

Central to the hypothesis that relative under nutrition associated with breastfeeding protects against later obesity is evidence that breastfed infants grow more slowly than those that are formula-fed¹⁸. While data from large epidemiological studies have confirmed the growth-accelerating effects of formula over the first 6 months, growth differences between breastfed and formula-fed infants may be greatest in the first few post-natal weeks, a time when breastfed infants often lose weight, while formula-fed infants tend to put on weight. As suggested by studies in animals and humans, this difference may be critical for the programming of obesity.

Future perspective

Overall, there is now good evidence to support a benefit of breastfeeding for long-term obesity risk, an effect possibly related to the slower growth and relative under nutrition associated with breastfeeding compared with formula feeding. Breastfeeding is therefore a preventative strategy,

which is both evidence-based and has large potential benefits for public health. Programming of obesity by infant nutrition has two major implications for health care. First, more resources have to be targeted to encourage exclusive breastfeeding by the high proportion of mothers in the UK still choosing to formula-feed. Breastfeeding promotion may be particularly important for reducing inequalities in health, as populations of low socioeconomic status tend to both formula feed and have lifestyle risk factors predisposing to obesity. Second, with the increasing evidence that growing too fast has detrimental long-term effects on health, mothers and healthcare professionals have to be aware of the harmful effects of over nutrition and growth acceleration and not focus exclusively on identifying suboptimal growth and under nutrition in infants. Further research is required to identify nutritional requirements in early infancy that facilitate an optimal pattern of growth for long-term health.

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