The long-term metabolic health outcomes of breast-fed v. formula-fed infants have received considerable attention and, although still controversial\(^1\), there have been some suggestions that breast-feeding may be protective against the development of obesity\(^2\). Although it is challenging in humans to separate the effects of breast-feeding per se from those of other socio-economic/lifestyle factors with which extended breast-feeding is closely entwined, animal data support the concept that there are biological links between being reared on artificial milk and being reared on natural suckling\(^3\).

The question then becomes – what are the drivers? The principal hypothesis, to date, has been related to differences in the composition of breast milk v. infant formula, particularly in its fatty acid content\(^4\). Specifically, it has been suggested that the higher \(n-3\) polyunsaturated fat (PUFA) and lower \(n-6\) PUFA contents in breast milk compared with the contents in infant formula are associated with lower rates of adipogenesis/lipogenesis in the developing neonate, giving rise to lower numbers of adipocytes with a lower capacity for fat storage.

The paper by Oosting et al.\(^5\) published in this issue, however, suggests that there is another factor that needs to be considered – namely, the physical structure of the lipids in human milk compared with that of the lipids in infant formula.

Oosting et al.\(^5\) prepared two diets with the same fatty acid composition, which had lipid droplets resembling either those in human breast milk (large lipid droplets surrounded by a biological membrane, Concept IMF) or those in infant formula (smaller lipid droplets with no biological membrane, Control IMF)\(^5\). They then fed these diets to rat offspring from postnatal day 16 until young adulthood. All offspring were then fed a Western-style diet until postnatal day 98. They found that rats fed the Concept IMF in early life gained less weight and less fat on the Western-style diet than the controls – indicating that they were less susceptible to diet-induced obesity. Importantly, this was driven almost exclusively by a reduction in lipogenesis, as indicated by the lower expression of lipogenic transcription factors and reduced size of the adipocytes.

Although changes in the fatty acid composition of the early-life diet have been shown to programme the function of adipocytes, this is the first time that it has been reported that this is associated with differences in the structural properties of lipids consumed in early life. This adds a new element to the growing body of literature linking early nutritional exposures to increased obesity risk and highlights the sensitivity of the developing adipocytes to the manipulations of the infant diet.

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