The Journal of Clinical Endocrinology & Metabolism, 2021, Vol. 106, No. 3, e1458–e1459
doi:10.1210/clinem/dgaa910
Commentary

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Further Evidence that Breast Milk Lipids Control Adiposity

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Abbreviation: 12,13-diHOME, 12,13-dihydroxy-9Z-octadecenoic acid.

Received: 9 November 2020; Editorial Decision: 2 December 2020; First Published Online: 7 December 2020; Corrected and Typeset: 5 January 2021.

Key Words: metabolism, neonate physiology, lipid mediators, obesity; breastfeeding

Adipose tissue development during infancy determines body adiposity in childhood and hence might have a lifelong impact on the probability of developing obesity (1, 2). Adipose tissue can store lipids as triacylglycerols or burn them off as heat and energy, and these 2 disparate functions are observed to varying degrees in the anatomically distinct fat depots. Obesity develops when energy intake exceeds expenditure, which favors fat storage in adipose tissues (3). The heat-generating function of subcutaneous adipose tissue is dominant just after birth but is lost in late infancy when the heat-generating cells transform into fat-storing cells, with the exception of some specific fat depots. Breast milk lipid signaling appears to control the onset of this transformation, and hence can determine adiposity in the infant (4). The underlying signal is provided by breast milk–specific lipid mediators—alkylglycerols—which directly control adipose tissue development in the infant, and likely have a lasting impact on childhood obesity. Indeed, insufficient breastfeeding promotes childhood obesity and increases the risk of inflammatory diseases and diabetes later in life (1, 2, 4).

In the current obesity pandemic, a better understanding of the roles of breast milk lipid mediators in adipose tissue development would seem timely and important. In this line, a recent study by Wolfs et al from the Elvira Isganaitis laboratory at the Joslin Diabetes Center in Boston (5) has addressed an intriguing question: do heat-generating adipose tissue-derived metabolites of the mother maintain heat-generating fat mass in the infant, and hence protect against obesity? The authors studied a linoleic acid metabolite, 12,13-dihydroxy-9Z-octadecenoic acid (12,13-diHOME), also known as isoleukotoxin, which was recently identified as a circulating lipokine released by heat-generating adipose cells in response to exercise and cold exposure (6). They showed that a bout of moderate exercise at 1 month postpartum increases the levels of 12,13-diHOME in human breast milk. They also found that greater breast milk abundance of 12,13-diHOME at 1 month postpartum was associated with lower subcutaneous fat mass in the infant, and a reduced gain in body mass index in the first 6 months of infancy (5). Thus, 12,13-diHOME and its related metabolites appear to protect against adiposity in infancy, which is similar to what has been reported in adults (7). Unlike breast milk–specific alkylglycerols, 12,13-diHOME is detectable in commercially available cow milk–based infant formula (5). Moreover, it is also produced by the gut microbiota and can be found on the surface of neonatal skin (5). A recent study demonstrated a correlation between increased microbiota-derived 12,13-diHOME levels and asthma in infancy (8), revealing its potential as a possible immune regulator and raising the possibility that it may also influence adipose tissue immune cell function in the infant. Although 12,13-diHOME levels and adiposity appear to be inversely correlated, it remains to be determined whether 12,13-diHOME reduces adiposity by increasing thermogenic fat differentiation. The

ISSN Print 0021-972X ISSN Online 1945-7197
Printed in USA

study by Wolds et al nevertheless further strengthens the novel concept that breast milk lipids constitute a unique molecular axis of communication between the mother and the child, and that they might be powerful determinants of adipose tissue development.

Additional Information

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Data Availability: Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Disclosures: The author has nothing to disclose.

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