bined curve fitting with assessment of horizontal and vertical shift, sensitivity, and positive predictive value for detecting hypoglycemia.

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# Breast-Feeding and Risk for Childhood Obesity

## Response to Mayer-Davis et al.

The study by Mayer-Davis et al. (1) reflects the fact that maternal nutrition plays an important role in the pathogenesis of childhood obesity. Breast milk contains linoleic acid (of the n-6 polyunsatured fatty acids [PUFA] series) and α linolenic acid (of the n-3 PUFA series) as well as longer chain derivatives, such as arachidonic acid (of the n-6 PUFA series) and docosahexanoic acid (of the n-3 PUFA series). Maternal intake determines content of breast milk, which ultimately affects the infant's future health. Childhood obesity is probably an immune inflammatory response to a faulty diet of the mother (before and during gestation and lactation) consisting of high n-6 PUFAs, low n-3 PUFAs, and deranged n-6–to–n-3 ratio (2). In those who are breast-fed, breast milk provides longer-chain n-3 PUFAs, which prevent ectopic accumulation of fatty acids in muscle and liver (3,4). Formula feeding does not provide this benefit. Cow's milk content depends on whether it is pasture fed (more n-3 PUFAs) or given commercial feeds (more n-6 PUFAs). Breast-fed infants have a muscle membrane fatty acid composition similar to insulin-sensitive adults, and formula-fed infants have a muscle membrane fatty acid composition similar to insulin-resistant adults (5). Correcting n-6 and n-3 PUFAs in the diet is currently needed for changing global health for one and all.

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# Breast-Feeding and Risk for Childhood Obesity

## Response to Mayer-Davis et al.

We read with great interest the recent study by Mayer-Davis et al. (1) on the impact of breast-feeding on childhood obesity risk in the presence of maternal diabetes or obesity. The authors drew conclusions that seem to directly oppose previous observations from our group (2,3). However, we would like to deliver three arguments suggesting that the presented data can also be interpreted in a completely different manner and in no way exclude, but rather support, a potentially negative dose-depending effect of early neonatal breast-feeding on overweight risk in offspring of diabetic/overweight mothers, as observed by us.

First, the majority of fully adjusted estimates for the effect of maternal diabetes have 95% CIs that include decreased as well as increased odds ratios over a wide range (e.g., odds ratio 0.79 [0.29–2.16] for breast milk only vs. formula only). By statistical definition, one therefore cannot exclude the possibility that the true effect of breast-feeding on overweight risk in the presence of maternal diabetes/obesity is not beneficial but deleterious, at least in a considerable number of cases.

Second, breast-feeding during the 1st month by diabetic mothers increased overweight risk compared with formula feeding. This, in fact, confirms rather than rejects our observations. Moreover, this is unlikely to be accounted for by reverse causation, since no dose response–like relation between duration of breast-feeding and risk of overweight was observed in offspring of diabetic mothers. These data may even support our hypothesis of a crucial and probably even deleterious impact of breast-feeding by diabetic mothers during the early neonatal period.

Finally, the authors stated that our observations might reflect "appropriate" growth rather than untoward effects. This, however, does not correspond with increased prevalence of overweight in the highest tertile of early neonatal intake of diabetic breast milk, using the symmetry index (2) additionally validated against BMI (4). Most importantly, this interpretation completely ignores deleterious ef-