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Response to "Cortisol in Human Milk: The Good, the Bad, or the Ugly?"

Jennifer Hahn-Holbrook
Chapman University, hahnholb@chapman.edu

Elysia P. Davis
University of California, Irvine

Laura M. Glynn
Chapman University, lglynn@chapman.edu

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Milk Cortisol and Childhood Obesity: Clarifications and Future Directions

Jennifer Hahn-Holbrook*, **Elysia P. Davis**, and **Laura M. Glynn**

We are thankful for the interest with which Finken and colleagues read our paper and appreciate the opportunity to discuss the important ideas they present. Finken et al. express concern that we assessed milk cortisol at one time-point to predict changes in infant BMIP because cortisol concentrations in milk vary across the day, reaching their apex shortly after waking and their nadir at night.[1,2] While we agree that collecting 24-hour milk samples will constitute an important methodological advance in future research, we regard our current findings to be reliable for several reasons. First, we included the time-of-day of sample collection in our multivariate growth curve models. By statistically adjusting for temporal variation, we isolated the contribution of milk cortisol on infant BMIP. Second, although it is true that the morning apex and nightly nadir of milk cortisol differ by ~500%,[1] cortisol variation in the timeframe relevant to our study (1100 – 1700) only differs by ~30%.[1] Consistent with this limited variation, the correlation between time-of-day and milk cortisol in our study was relatively modest and only marginally significant ($\beta = .27$, $p = .050$). Given these complementary considerations, our study most likely captured between-mother variation in milk cortisol, as intended, rather than within-mother variation in diurnal milk cortisol. Furthermore, the vast majority of prior research on early glucocorticoid exposure and later obesity risk derives from samples taken at one time-point.[3]

Finken and colleagues also express skepticism regarding our (admittedly speculative) hypothesis that milk cortisol may facilitate maturation of the infant gut, enabling greater nutrient absorption and thereby potentially explaining the observed relationship between higher milk cortisol and reduced infant BMIP gains over the first two years of life. Finken et al. counter that greater nutrient absorption would be related to ‘catch-up growth’, or rapid weight gain in the early months of life known to be a risk factor in later obesity, against our finding that high milk cortisol appeared protective against obesity.[4] Crucially, however, nutrient absorption and caloric consumption should not be conflated; the former is protective against obesity while the latter is a risk factor.[5] Indeed, many obese individuals are overfed yet undernourished.[5] As noted in our paper, the mechanisms by which milk glucocorticoids impact infant body composition have yet to be elucidated. We encourage researchers to test Finken and colleagues’ hypothesis that dysregulation of diurnal milk cortisol patterns disrupts infants’ intestinal immune and microbiomal function, thus increasing obesity risk.

*Correspondence should be addressed to Jennifer Hahn-Holbrook, Chapman University, Crean School of Health and Behavioral Sciences, 1 University Drive, Orange, California, 92866. hahnholb@chapman.edu.

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From our perspective, their proposal is both plausible and orthogonal to our suggestion regarding a possible pathway by which high milk cortisol might enhance infant nutrient absorption.

Once again, we thank Finken et al. for their engagement with our work. Ideally, independent groups will attempt to replicate and extend this research by conducting longitudinal studies of 24-hour milk cortisol patterns on multiple days, while including assessments of potential metabolic, immune and microbiomal mechanisms.

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