Association of parental obesity with infant birth weight: weighing the evidence

Suboptimal birth weight is a risk factor for obesity and cardiometabolic diseases later in life, highlighting the need for a transgenerational and life-course approach to disease prevention. A wide variety of perinatal exposures, which can be conceptualized as both biological and societal determinants, are associated with birth weight in the progeny. However, most of the research on this topic has focused on exposures in pregnant persons with little to no consideration of their partners. This is despite the mounting evidence that many risk factors for health outcomes tend to aggregate within families, potentially imposing a synergistic effect.

We, therefore, applaud McPherson et al. (1) for their article on birth weight associations with parental obesity in this issue of *F&S Reports*. As of July 30, 2021, a PubMed search query using "maternal obesity" and "birth weight/birthweight/infant weight" yields 545 results, whereas using "paternal obesity" and "birth weight/birthweight/infant weight" yields 19 results, making evident a notable disparity. McPherson et al. (1) contribute to addressing this gap in evidence by evaluating the additional role of paternal body mass index (BMI) on the association of infant birth weight with maternal prepregnancy BMI in a retrospective analysis of 1778 singleton term births. Contrary to their hypothesis and in contrast to some previous studies, the investigators find that increases in the mean infant birth weight associated with maternal obesity are attenuated in the presence of paternal obesity.

This valuable and interesting research has led us to reflect on the selection and handling of both neonatal and parental anthropometric variables that may be best suited for illuminating the implications of such work. The investigators' careful treatment of parental BMI including their evaluation of continuous BMI and World Health Organization categories, and their modeling of continuous BMI using linear and nonlinear terms are commendable. They also rightly discuss that birth weight can be suboptimal at either end of the spectrum. Nevertheless, the present study did not evaluate clinically relevant neonatal variables such as low birth weight, macrosomia, small for gestational age, and large for gestational age. Direct evaluations of these categorical outcomes could provide further insight into the implications of the results, such as the clinical significance of the reported approximately 290-g higher birth weight associated with maternal obesity.

Future studies should also consider incorporating paternal height—an often-overlooked anthropometric measure that can capture genetic influences on stature and in some contexts, social class. The investigators' focus on paternal BMI as the primary exposure is, of course, justified given its modifiable nature compared to height. However, paternal BMI has shown independent associations with infant birth weight even after accounting for paternal height (2). It is even conceivable that the lack of adjustment for paternal height may attenuate the expected positive association between paternal BMI and infant birth weight, potentially contributing (however minimally) to the null linear association of paternal BMI with infant birth weight reported in the present article. To be sure, the same argument can be applied to maternal height and BMI. Therefore, their finding of a higher contribution of maternal BMI to infant birth weight than paternal BMI may likely still hold even when accounting for parental height.

The attenuation of infant birth weight and maternal obesity associations in the presence of paternal obesity highlight the need for future work that evaluates potential mediators in order to understand the mechanisms of these associations. For example, gestational weight gain is an important predictor of infant birth weight even while accounting for prepregnancy BMI (3). Could the obesity status of a pregnant person's partner negatively or positively influence their behavior, and therefore their success in meeting the recommended gestational weight gain goals? Or, as the investigators suggest, is the paternal contribution to infant birth weight driven by biological mechanisms such as obesity-related sperm epigenetic changes, thereby limiting its role to the preconception period? Examining potential mediators such as gestational weight gain may help move the needle forward on understanding the relative contributions of social/behavioral and biological mechanisms of association. Elucidating these potential mechanisms can also help direct the often-limited public health resources toward prevention efforts that prioritize the preconception or gestation period.

Despite the counterintuitive results for the additional role of parental obesity in this particular study, the investigators still conclude with an emphasis on the importance of a couple's based approach to preconception care. We echo their sentiment, but we do so with a call for further research into potential mechanisms and comprehensive and clinically meaningful measures of parental and neonatal anthropometry to inform evidence-based care.

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