The global epidemic of obesity has generated a lot of interest in the mechanisms resulting in the epidemic and its effects on population health. Towards this end, the effect of maternal nutrition (over- and undernutrition) on fetal and neonatal outcomes is being studied. It is understood that the maternal nutritional status is not simply a question of supply but also the effects of confounding factors such as infections and workload. Maternal nutrition before, during and after birth can all influence the long-term outcomes of the infant.

In this supplement issue of *Annals of Nutrition and Metabolism*, Dr. Yajnik elegantly examines the transmission of obesity-adiposity and noncommunicable diseases from the mother to the baby. He reminds us that ‘we sometimes forget that an individual is born at conception and not at delivery’. Citing multiple studies including his own, he describes the mechanisms of methylation of DNA, chemical modifications of histones and transcription and miRNAs that interfere with the translation of mRNA. Dr. Yajnik discusses a modifiable component of the intergenerational transmission of health and disease traits and suggests that the model of ‘primordial’ prevention offers an opportunity to reduce the burden of noncommunicable diseases, obesity, diabetes and so on by influencing the lifestyle of young women in the preconceptual and periconceptional window.

In their paper entitled ‘Fast growth of infants of overweight mothers: can it be slowed down?’, Haschke et al. then analyze observational studies demonstrating that breastfed infants are protected from obesity in later life. They further describe the nutritional content of human breast milk and its changes with lactation using data from recent studies attempted to answer two questions: first, is there an effect of maternal obesity/overweight on infant growth? And second, does feeding a formula with a lower-than-traditional content of protein decrease the growth trajectory of the infant and, therefore, confer a benefit? From the pooled data, the authors found that infants born to overweight/obese mothers demonstrate accelerated growth during the first year of life even when they were breastfed. Further, they argue that reducing the protein content of follow-on formulas should be a goal for the formula industry by demonstrating that infants fed formulas with a lower protein content (closer to that of breast milk) while supporting adequate growth can ‘slow down fast growth of infants of overweight/obese mothers’. This decrease in growth acceleration may reduce the risk for future obesity.

Dr. Vickers from New Zealand addresses the issue of developmental programming and transgenerational transmission of obesity. Arguing that little attention has been paid to the role of developmental plasticity and alterations in phenotypic outcomes resulting from environmental perturbations in the early-life period, he elegantly describes current evidence suggesting that developmental programming is a transgenerational phenomenon. Data
from both human and animal studies as well as the mechanisms involved are presented. Transgenerational epigenetic transmission of traits allows future generations to be maximally competitive in their environment; however, evidence suggests that environmental exposures such as malnutrition in early life result in maladaptive parental responses that can be passed on to the offspring. Dr. Vickers concludes that ‘understanding the mechanisms of transgenerational inheritance is essential for the development of future intervention strategies to modulate not only that of the immediate adult phenotype but also that of the offspring, grandoffspring and beyond’.

Lastly, Michael G. Ross and Mina Desai continue on the theme with a review entitled ‘Developmental programming of appetite/satiety’ by using data from studies of maternal/fetal under- and overnutrition demonstrating the increased risk for obesity caused by hyperphagia. Further, infants small for gestational age or those born to obese mothers who consume a high-fat diet are also at an increased risk for adult obesity. The authors provide epidemiological evidence for perinatal appetite programming and discuss appetite regulation and mechanisms. Through complex interplay of multiple factors, they conclude that ‘the maternal environment can program fetal/newborn appetite/satiety pathways resulting in newborn and adult hyperphagia’.

In summary, this issue of *Annals of Nutrition and Metabolism* brings to light new information of transgenerational programming, mechanisms altering appetite and the epigenome as well as the effect of overnutrition in the offspring through postnatal feeding and offers a possible prevention strategy aimed at the future generation of mothers.

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