

Maternal, Paternal, and Societal Efforts Are Needed to “Cure” Childhood Obesity

To the Editor: In the January 2015 issue of *Mayo Clinic Proceedings*, Archer¹ presented the maternal resource hypothesis (MRH) as a nongenetic mechanism responsible for childhood obesity. We commend him for providing a thought-provoking narrative that integrates many disciplines, and we support several of his assertions, including the necessity of regular physical activity for optimal metabolic health. However, we believe a few issues deserve attention.

First, the “paradigm shifting” nature of the MRH may be an overstatement to those actively exploring this research area. It may be more appropriately described as a sociobiological extension of the truly groundbreaking work of the late Sir David Barker’s developmental origins of health and disease concept and the systems approach to energy regulation offered by the UK Foresight initiative.² Further, although it was encouraging to see the inclusion of epigenetics in the hypothesis, the implied contribution of epigenetics as the mechanism driving the intergenerational cycle of obesity is complicated and largely oversimplified. Indeed, novel research exploring epigenetic inheritance of obesity has questioned the validity of many contentions suggesting that epigenetic markings (eg, methylation) persist over time. It has been suggested that obesity may be the result of epigenetically mediated transgenerational recapitulation of learned behaviors.³ Clearly, disentangling the relative contributions of recapitulation of environmental factors with genes that promote positive energy balance and increased body habitus poses a challenge. This issue alone warrants consideration in such a comprehensive theory.

The additional message implicit in Archer’s narrative is the notion that mom is largely responsible for child outcomes whether optimal or poor,

and he fails to consider the emerging evidence supporting the independent paternal contributions to obesity. Indeed, paternal obesity alters sex hormones and sperm form and function, which can subsequently impair embryo development and increase offspring disease risk.⁴ We would be interested in commentary regarding the vital sociobiological implications that paternal support in the prenatal and postpartum periods has for child health.

Consideration for gestational weight gain (GWG), in addition to maternal obesity, would surely supplement his discussion of the MRH. Many would argue that it is imperative to recognize GWG as an important modifiable risk factor of child size at birth. Big does not always beget big because normal-weight moms who exceed GWG recommendations have an increased likelihood of large for gestational age neonates, and the majority of women with obesity give birth to appropriate for gestational age babies,⁵ outcomes independent of maternal physical activity. A surplus of maternal resources in the form of excess macronutrient consumption coupled with pregnancy-induced maternal insulin resistance may drive substrate delivery to the fetus. However, one cannot discount the role of the placenta when theorizing the volume of energy presented to baby. In fact, placental adaptations appear to resist deviations in nutrient delivery in efforts to regulate and optimize fetal size.

Overall, one’s genetic predisposition to obesity and small deviations from what constitutes “normal behavior” should not dissuade efforts to improve maternal health. Healthy outcomes are not categorically all or nothing. Imperfect behaviors do not constitute irreversible lifetime risk; susceptibilities can be attenuated by a healthful postnatal environment. Every little bit counts, and small changes can have powerful effects on maternal-fetal health. So let’s stop blaming moms and empower them, give them the tools to facilitate healthful

decision making, and institute societal-level changes that will help in this process. After all, making a baby takes two, but as the African proverb posits, “it takes a village to raise a child.”

Zachary M. Ferraro, PhD

The Ottawa Hospital
Ottawa, Canada

Kristi B. Adamo, PhD

HALO Research Group, Children’s Hospital of
Eastern Ontario Research Institute
Ottawa, Canada

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In reply—Maternal, Paternal, and Societal Efforts Are Needed to “Cure” Childhood Obesity

I welcome Drs Ferraro and Adamo’s discourse and readily admit that whenever educated readers wholly fail to comprehend the main thrust of a theory, the fault frequently lies with the author (ie, me). Given this possibility, I now seek to remove whatever opacity led to their miscomprehension.

Science is the pursuit of lawful relations, not mere correlations, and the only test of a scientific theory is how well it explains *all* the available evidence, not isolated findings. With these premises as a foundation, my theory links the socioenvironmental