Letters to the Editor

RE: “WHY EVIDENCE FOR THE FETAL ORIGINS OF ADULT DISEASE MIGHT BE A STATISTICAL ARTIFACT: THE ‘REVERSAL PARADOX’ FOR THE RELATION BETWEEN BIRTH WEIGHT AND BLOOD PRESSURE IN LATER LIFE”

Regarding the interchange between Tu et al. and Weinberg (1–3) about the “fetal origins” hypothesis, I fear that both sets of authors come to the wrong conclusions. As Weinberg (2) observes, Tu et al. (1) are simplistic in their arguments. In perinatology, birth weight itself—or more accurately, fetal size at birth or length of gestation—has intrinsic meaning. Avoiding restricted fetal growth or increasing length of gestation can prevent neonatal morbidity and mortality. In the “fetal origins” paradigm, however, birth weight itself is not a causal factor. One only has to recognize that higher birth weight is related to higher body mass index, and higher body mass index predicts higher blood pressure, to understand that the factors underlying that pathway must be different from those that explain any possible inverse association of birth weight with later blood pressure. Thus, trying to estimate the overall “effect” of birth weight on blood pressure is nonsensical.

Weinberg’s clarification touches on these issues (2), but she concludes, inexplicably, that we should investigate whether dizygotic twins have adult blood pressures that are different from those of singletons, because twins and singletons have different birth weights. This suggestion just returns us to the imprudent emphasis on birth weight, this time in the much more complicated physiologic system of twin pregnancy (4). The better way forward in fetal/developmental origins research is to examine putative prenatal determinants directly, through both observational and experimental designs (5–7). Birth weight got us into fetal origins research, but it cannot lead us forward.

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References


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