In their letter, Rich-Edwards et al. (1) say that we misinterpreted our estimate for birth weight. They are right in stating that the parameter was –0.96 mmHg (95 percent confidence interval: –1.38, –0.54) for a 1 kg increase in birth weight (2). However, because the model included both later weight and birth weight, the effect of birth weight must be interpreted by holding later weight constant and examining changes in birth weight. Suppose two people are the same weight at age 7 years (say, a 1 standard deviation (SD) score) but have different birth weights (say, a 0 SD score and a –1 SD score); the predicted blood pressure for the person with the lower birth weight is 0.96 mmHg higher than that for the person with the higher birth weight. However, the person with the lower birth weight has grown more (a 2 SD score) than the person with the higher birth weight (a 1 SD score). Thus, the model should be interpreted in terms of growth or change in position on the weight distribution (centile change) rather than birth weight. Therefore, it is fair to say that a 1-SD-score increase in weight between birth and a later age is associated with an increase in systolic blood pressure of 0.96 mmHg (95 percent confidence interval: 0.54, 1.38). This interpretation is also plausible because increases in blood pressure have been shown to be related to increases in body size during adolescence and adult life (3–5). Furthermore, randomized trials of different forms of infant feeding and consequent blood pressure suggest that different patterns of weight gain in early childhood may be associated with later blood pressure (6).

Because models that include current size as well as birth size can be interpreted in terms of centile crossing or growth, Lucas et al. specified two other criteria that must be met for the fetal origins hypothesis to hold (7). The first was that the association between birth weight and blood pressure (the early model) should be statistically significant, which was not the case in our study. The second was that the interaction effect between birth weight and current weight, included to find out whether weight gain was more important for people with very low birth weights than it was for people whose birth weights were closer to the center of the distribution, should be statistically significant. This term was not statistically significant in our analysis. We therefore concluded that our study did not support the fetal origins hypothesis.

Rich-Edwards et al. (1) argue that we mistakenly chose to interpret the interaction effect rather than the birth-weight term in the “interaction model.” Because this model included terms for both later weight and birth weight, the latter would be interpreted in terms of growth. The blood pressure estimates based on the “main” and interaction effects provided by Rich-Edwards et al. are based on changes in “adult” weight for unspecified birth weights rather than on changes in birth weight for a fixed weight at a later age. Because estimates of blood pressure that include the interaction effect depend on both variables, the same pattern of findings will not apply for all levels of later size.

The estimates that our data provide are similar to those of many studies. A meta-analysis of the regression coefficients of 55 studies concluded that the effect of birth weight on blood pressure was small and that adjusting for current size exaggerated the effect of birth weight and may have produced spurious results (8).

REFERENCES


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