Response to Letters Regarding Article, "Pulmonary and Systemic Vascular Dysfunction in Young Offspring of Mothers With Preeclampsia"

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We thank Lazdam et al and Yuan et al for their interesting comments on our study.[1] Lazdam et al point out that in our study, birth weight in offspring of mothers with preeclampsia was significantly lower than in controls, and suggest that the vascular impairment in offspring was caused by a combination of the independent effects of preeclampsia and intrauterine growth restriction rather than by preeclampsia per se. To test this hypothesis, we performed a subgroup analysis of 15 offspring of mothers with preeclampsia and 15 controls matched for birth weight (3080±291 versus 3077±398 g; $P=0.98$). We found that the difference in flow-mediated dilation between birth-weight-matched offspring of preeclampsia and controls (6.4%±1.3% versus 8.9%±1.2%; $P<0.0001$) tended to be even larger than the one we reported in the entire groups (6.3%±1.2% versus 8.3%±1.6%; $P<0.0001$). This additional analysis, together with the already reported lack of any significant relationship between birth weight and flow-mediated dilation, indicates that vascular dysfunction in these young offspring is related to preeclampsia alone, and that low birth weight does not contribute to this problem. We therefore do not overestimate the impact of preeclampsia on vascular function in these term offspring.

Lazdam et al also ask for information on other aspects of vascular function and are surprised that we found no significant differences in peripheral blood pressure between these young offspring of mothers with preeclampsia and controls. First, we wish to note here that it took large epidemiological studies to demonstrate small, albeit statistically significant, differences in peripheral blood pressure between offspring of preeclampsia and controls.[2] Most importantly, in 24 offspring of preeclampsia and 27 controls we measured central blood pressure by tonometry (SphygmoCor, AtCor Medical, Sydney, Australia) and found that systolic (97.2±6.8 versus 97.8±9.3 mm Hg; $P=0.79$) and diastolic (76.3±7.1 versus 73.0±7.1 mm Hg; $P=0.74$) central blood pressure was comparable. We also assessed carotid-femoral pulse wave velocity (Complior Device, Alam Medical, Vincennes, France) in these subgroups and found that it was similar in offspring of preeclampsia and controls (6.6±1.1 versus 6.2±1.2 m/s; $P=0.25$). These findings indicate that, in contrast to flow-mediated dilation, peripheral and central blood pressure and pulse wave velocity are still normal in young offspring of preeclampsia. These important additional data suggest that endothelial dysfunction represents the very first step in the development of arteriosclerosis[3] predisposing to premature cardiovascular disease in offspring of preeclampsia.

Yuan et al suggest that vascular dysfunction in offspring of preeclampsia is related to low physical activity. Although we did not directly assess physical activity in our study, we do not have any evidence that could be consistent with this hypothesis. For example, low physical activity would be expected to be associated with an increased body mass index in offspring of preeclampsia, the opposite of what we found in our study. Siblings often have comparable physical activity; in our study, siblings of offspring of mothers with preeclampsia who were born after normal pregnancy had perfectly normal vascular function. Finally, resting heart rate was comparable in offspring and controls. We are very confident that the large differences in vascular function between offspring of preeclampsia and controls are not related to differences in physical activity, but to preeclampsia alone.

Disclosures

None.

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