COMMENTS AND RESPONSES

Response to Comment on: Black et al. The Relative Contribution of Prepregnancy Overweight and Obesity, Gestational Weight Gain, and IADPSG-Defined Gestational Diabetes Mellitus to Fetal Overgrowth. Diabetes Care 2013;36:56–62

We thank McIntyre and Catalano (1) for their interest in our article (2). In this study, we examined the relative effects of prepregnancy overweight/obesity, excess gestational weight gain, and IADPSG-defined gestational diabetes mellitus (GDM) on fetal overgrowth and other adverse outcomes, in a sample of 9,835 women who had a 2-h 75-g oral glucose tolerance test and were not treated with diet, exercise, or antidiabetic medications during pregnancy. Our analysis revealed that the majority (75%) of GDM women were overweight or obese at the start of pregnancy, and 21.6% of large-for-gestational-age (LGA) infants were attributable to overweight/obesity among women who never developed GDM. We also found that the effects of overweight/obesity and GDM were additive, jointly accounting for 23.3% of LGA, and that excess weight gain exacerbated these effects. Despite the fact that GDM among normal weight women accounted for <3% of LGA infants, we never suggested that GDM should not be treated.

In their letter, McIntyre and Catalano question the relevance of our findings due to the observation that the rates of overweight (32%) and obesity (28%) in our sample exceeded those in Hyperglycemia and Adverse Pregnancy Outcome study (22% and 14%, respectively). While we made no attempt to generalize our findings to the worldwide population, it is worth noting that the prevalence of overweight/obesity in our sample is nearly identical to that of U.S. women of reproductive age (3). The authors also suggest that including underweight women and excluding treated women may have biased our inferences on the importance of obesity relative to GDM. Underweight women (BMI <18.5 kg/m^2) comprised <2% of our sample and had a mean BMI approximately 1 SD from the normal weight threshold (17.6 ± 0.8 kg/m^2). Results did not change after excluding them from analysis. Results were also similar after including treated women; estimates changed slightly only for overweight GDM (population attributable fraction: 7.3 vs. 6.2%) and obese GDM women (population attributable fraction: 20.8 vs. 17.1%). This is because all treated women excluded from our analysis had GDM, and 87% were overweight or obese, which further illustrates the strong correlation between maternal adiposity and elevated glucose levels in pregnancy.

To date, interventions aimed at controlling weight gain in pregnant women have predominantly been of poor quality, with inconsistent findings (4,5). Many have been successful in limiting weight gain, the primary outcome for which they were powered; birth weight, LGA, and cesarean delivery, if assessed, were secondary outcomes for which they were not powered. Moreover, most studies included normal weight women (4,5), and thus power to detect significant intervention effects among overweight/obese women was further diminished by small sample size. This highlights the critical need for large-scale, well-designed, adequately powered intervention trials focused on reducing prepregnancy weight and controlling gestational weight gain in overweight and obese women.

Prepregnancy overweight/obesity and excess weight gain are also strongly associated with GDM development. Therefore, interventions that effectively help overweight/obese women lose weight before pregnancy and/or control weight gain during pregnancy may reduce adverse outcomes in overweight/obese women who never develop GDM, as well as the occurrence of GDM and the adverse outcomes attributable to it.

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