COMMENTS AND RESPONSES

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 read with interest the article by Black et al. (1) and the associated commentary by Ryan (2) and wish to provide some comments. Firstly, calculations by Black et al. of population attributable fractions (PAFs) of large-for-gestational-age (LGA) babies attributable to overweight/obesity and gestational diabetes mellitus (GDM) are, as correctly noted, largely influenced by the high prevalence of obesity (28%) and overweight (32%) in their sample. By contrast, in the Hyperglycemia and Adverse Pregnancy Outcome study (3), conducted in 15 centers worldwide, the prevalence of obesity (14%) and overweight (22%) was much lower. Therefore, in the global context, PAFs relating to maternal BMI would be substantially lower. The inclusion by Black et al. of underweight women in the referent group and exclusion of women treated for GDM (25% of all GDM women by the International Association of the Diabetes and Pregnancy Study Groups criteria, including those with more marked hyperglycemia) may also have contributed to overestimation of the relative importance of obesity compared with GDM.

Further, the PAF calculations (conventionally) assume that all women in the overweight and obese groups, even the most markedly obese, could revert to the normal weight category. In reality, this would be nothing short of a miracle and overstates the real world therapeutic potential. Further, as noted by Ryan (2), there is no evidence that current interventions during pregnancy in obese women actually reduce fetal overgrowth. We concur with Ryan and others that achievement of a healthy body weight prior to a planned pregnancy is a worthwhile research objective that needs further evaluation. Maternal prepregnancy BMI has a stronger relationship with fetal fat accretion compared with gestational weight gain in overweight and obese women. In contrast, in lean or normal BMI women, gestational weight gain has a stronger relationship with fetal fat accretion compared with maternal prepregnancy BMI (4). Hence the approach to gestational weight gain needs to be individualized.

In contrast to the dearth of benefits demonstrated for obesity treatment during pregnancy, a recent systematic review (5) has confirmed benefits of treatment of GDM including reduced frequency of LGA, preeclampsia, hypertensive disorders of pregnancy, and shoulder dystocia. Although the statement by Ryan (2) that "glucose is playing a minimal role in LGA" may have some limited veracity in terms of theoretical PAFs, it ignores the strong body of evidence showing that GDM is a modifiable risk factor for LGA and that GDM treatment provides the other clinically important benefits noted above. Treatment of GDM also provides the possibility of limiting excessive gestational weight gain during pregnancy. This may decrease postpartum weight retention and thus prepregnancy overweight and obesity in subsequent pregnancies. Our aim should not be the "elimination" of LGA, but rather overall health improvements. Women with GDM should receive proven interventions involving lifestyle modification and judicious use of pharmacotherapy during pregnancy. In the postpartum period, this should be followed by energetic attempts to prevent them progressing to frank obesity and related metabolic dysfunction, including the potentially devastating risks of diabetes and cardiovascular disease.

HAROLD DAVID MCINTYRE, MD[1]
PATRICK M. CATALANO, MD[2]

From [1]Mater Clinical School, The University of Queensland and Mater Health Services, South Brisbane, Queensland, Australia; and [2]Reproductive Biology, Case Western Reserve University at MetroHealth Medical Center, Cleveland, Ohio.

Corresponding author: Harold David McIntyre, david.mcintyre@mater.org.au.

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