A review of maturity onset diabetes of the young (MODY) and challenges in the management of glucokinase-MODY

In reply: We thank Flack and colleagues for their detailed comments on our review.[1] The prevalence of gestational diabetes mellitus (GDM) of 5–10% was based on a prospective study by Moses and colleagues using the Australasian Diabetes in Pregnancy Society criteria, which found a prevalence in Wollongong, New South Wales, of 8.6–10.5% (9.6% overall).[2] Using the more recently adopted International Association of Diabetes and Pregnancy Study Groups diagnostic definition in the same study, the prevalence of GDM was 13.0%. However, the cohort primarily comprised Australian women of European descent and the data were likely influenced by the slightly older maternal age; therefore, it may not reflect the national prevalence of GDM. Although other studies have produced a similar figure (eg, 13.6%),[3] data from the National Perinatal Data Collection provided by the Australian Institute of Health and Welfare indicated a prevalence of 4.8%.[4] Therefore, a range of 5–10% was deemed an adequate approximation. We agree with Flack and colleagues that certain multi-ethnic populations (eg, Bankstown-Lidcombe) will have higher GDM prevalence rates.

The second comment related to the number of pregnant women with slim body habitus (body mass index, < 25 kg/m^2) and isolated fasting hyperglycaemia (≥ 5.5 mmol/L) needed to test to yield one diagnosis of maturity onset diabetes of the young due to a glucokinase mutation (GCK-MODY). The figure of 2.7 derived from a study of primarily European women with a small (7%) ethnic minority.[5] Although not representative of the multicultural Australian population, this study remains one of the largest population studies addressing GCK-MODY prevalence in pregnancy ($n = 5500$). Further, it is clear that the presence of a predominantly ethnic population, as noted from the authors' cited studies (eg, Asian, Middle Eastern, Indian and Afro-Caribbean), may diminish the relative proportion of GDM secondary to *GCK* mutations, given the higher likelihood of insulin resistance in these populations. We agree that, in this context, the number needed to test may be much higher than 2.7; however, no large population-based Australian studies with confirmatory genetic testing were available at the time of writing.

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References are available online at www.mja.com.au.

1 Bishay RH, Greenfield JR. A review of maturity onset diabetes of the young (MODY) and challenges in the management of glucokinase-MODY. *Med J Aust* 2016; 205: 480-485. https://www.mja.com.au/journal/2016/205/10/review-maturity-onset-diabetes-young-mody-and-challenges-management-glucokinase

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3 Sacks DA, Hadden DR, Maresh M, et al. Frequency of gestational diabetes mellitus (GDM) at collaborating centres based on IADPSG consensus panel recommended criteria: the Hyperglycaemia and Adverse Pregnancy Outcome (HAPO) study. *Diabetes Care* 2012; 35: 526-528.

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5 Chakera AJ, Spyer G, Vincent N, et al. The 0.1% of the population with glucokinase monogenic diabetes can be recognized by clinical characteristics in pregnancy: the Atlantic Diabetes in Pregnancy cohort. *Diabetes Care* 2014; 37: 1230-1236. ■