

COMMENTS AND RESPONSES

Response to Comment on: Selvin et al. No Racial Differences in the Association of Glycated Hemoglobin With Kidney Disease and Cardiovascular Outcomes. Diabetes Care 2013; 36:2995–3001

We thank Drs. Kilpatrick and Bloomgarden (1) for their interest in our study. While we agree that the higher mean HbA_{1c} in blacks compared with whites is well established, the collective evidence suggests that risk associations are similar. The goal of our recent study (2) was to compare the prognostic value of HbA_{1c} to fasting glucose for risk of major complications in black and white adults. The primary conclusion of our study was that we did not observe major differences between blacks and whites in association with HbA_{1c} and long-term clinical outcomes. First, our results suggest that HbA_{1c} is stronger than fasting glucose as a predictor of outcomes in both whites and blacks. Second, in the only outcome (mortality) where we observed a significant difference between whites and blacks in the risk association, the interaction was present for both HbA_{1c} and fasting glucose; the hazard ratios for all-cause mortality and undiagnosed diabetes in whites were 1.74 (95% CI

1.38–2.18) for HbA_{1c} \geq 6.5% and 1.62 (1.34–1.96) for fasting glucose \geq 126 mg/dL. The corresponding weaker hazard ratios for undiagnosed diabetes in blacks were 1.38 (1.05–1.81) for HbA_{1c} \geq 6.5% and 0.99 (0.73–1.34) for fasting glucose \geq 126 mg/dL.

We think that Kilpatrick and Bloomgarden's focus on only the prediabetic group (HbA_{1c} of 5.7–6.4%) is inappropriate since power in this group is more limited. They point out that HbA_{1c} in this range is often not associated with outcomes in blacks, but it is important to note that the prediabetic group defined by fasting glucose (100–125 mg/dL) is also not significantly associated with any of the outcomes either in blacks or whites in this study population. The group with HbA_{1c} \geq 6.5% is of greatest clinical importance, particularly when making statements about diagnosis. Furthermore, the continuous analysis provides greater power and the figure in our article strongly supports our original conclusions. Our results support the contention that the processes that lead to the higher HbA_{1c} in blacks compared with whites reflect glycaemic factors. Indeed, blacks are known to be at higher risk for diabetes and its complications (3,4). It is likely that the racial differences in HbA_{1c} reflect differences in diet, lifestyle, and possibly chronic exposure to higher postprandial glucose levels (5). Retinopathy, which is highly specific to diabetes, occurs at a higher prevalence in blacks compared with whites, even at the same HbA_{1c} level (6). We find no evidence that there is anything "false" about elevations of HbA_{1c} in blacks.

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