Type 1 diabetes (T1D) is associated with the development of debilitating, life-shortening complications related to chronic hyperglycemia. In 1993, the landmark Diabetes Control and Complications Trial (DCCT) demonstrated that carefully supervised, intensive basal-bolus insulin therapy that led to the chronic reduction of HbA1c to normal or near-normal levels could prevent the occurrence and progression of microvascular complications. The DCCT findings led to the use of HbA1c as the main clinical indicator of treatment effectiveness, recommendations for patient HbA1c treatment goals in the normal/near normal range, and advocacy of intensive insulin management to achieve those goals. In the 2 decades since the DCCT, there have been many technical advances in insulin formulations, insulin delivery systems, and blood glucose monitoring to facilitate the achievement of HbA1c outcome goals. Despite these changes, the T1D Exchange Clinic Network found that the majority of pediatric patients in their survey still did not reach recommended HbA1c goals and thus remain at potentially higher risk for developing complications.

In an important follow-up study in this issue of Pediatrics, the T1D Exchange Clinic Network further analyzed treatment patterns and HbA1c outcomes from 10,704 pediatric patients in 60 participating clinical centers across the United States. Data were statistically adjusted to take into account potential variation due to family socioeconomic status (SES), patient gender, age, and method of insulin delivery. Particularly perplexing and worrisome is the confirmation by this new survey that black children have higher HbA1c than white children. The underlying cause of this persistent racial disparity in HbA1c is unclear. The data indicate that regardless of age and SES, black youth were far less likely to be using insulin pumps. However, regardless of age and whether using pumps, multiple daily injections, or fixed-dose mixed insulin, black patients still had higher HbA1c levels than white patients. This suggests that factors besides the insulin-delivery method account for HbA1c differences. As yet unrecognized differences related to management or factors such as education, cultural sensitivity, or follow-up care between blacks and whites might contribute to higher HbA1c in black patients.

The possibility of factors unrelated to mean blood glucose (MBG) level as a cause of higher HbA1c in black patients should also be considered. In a study by Kamps comparing black and white children with T1D, blacks were found to have both higher HbA1c and MBGs compared with whites. However, when HbA1c was statistically adjusted for the level of MBG, the adjusted mean HbA1c was still higher in blacks by 0.8. This indicates that even at identical MBG levels, blacks will have higher HbA1c than whites. Additional evidence for a nonglucose contribution to differences in HbA1c between the races has been reported from other populations. Although glucose meter MBG from a subset of the T1D Exchange Clinic patients was higher in blacks than whites, the HbA1c data were not further statistically
adjusted to examine the possibility of racial disparity not attributable to MBG. A component of HbA1c that is independent of MBG may not be modifiable by changes in insulin dosing. If such non-MBG factors contribute to higher HbA1c in blacks, then attempts to drive HbA1c down to target by lowering MBG through more aggressive insulin dosing would cause greater occurrence of hypoglycemia. Indeed, blacks had higher reported occurrence of hypoglycemia than whites in the T1D Exchange Clinic survey. Clarification of the impact of factors besides MBG as a cause of higher HbA1c is important because such factors may require new approaches to patient monitoring and innovative interventions for safe and effective prevention of complications.

Besides racial disparity in HbA1c, the wide range in mean HbA1c (7.8%–9.9%) between participating clinics also catches the eye. This degree of between-clinic variation in HbA1c echoes the findings of the earlier multicenter Hvidore study.9 Between-clinic differences in HbA1c may be due to SES, cultural, and ethnic composition of the local populations served. However, factors at each clinic such as patient to staff ratio, staff composition, patient accessibility to staff, level of staff training, experience and motivation, incorporation of best management practices, and financial support for management services provided may also account for wide variation in HbA1c outcomes. The T1D Exchange Clinic Network report challenges us to identify and confront the sources of ongoing disparities in our young patients with T1D. Additional work is a priority to develop and implement safe and more effective solutions to achieve better long-term health outcomes.

REFERENCES

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