COMMENTS AND RESPONSES

Comment on: Riddle et al. Contributions of Basal and Postprandial Hyperglycemia Over a Wide Range of A1C Levels Before and After Treatment Intensification in Type 2 Diabetes. Diabetes Care 2011; 34:2508–2514

The studies devoted to contributions of postprandial (PPG) and fasting/basal glucose to overall hyperglycemia remain limited to non–insulin-using diabetic patients, and most reports indicate that PPG makes the highest contribution in patients with satisfactory control (1). Recently, Riddle et al. (2) have challenged this concept and extended the analysis to type 2 diabetic patients treated with different insulin regimens. From a general point of view, this study contains both expected and unexpected results. The analysis after treatment intensification with insulin confirms that the contribution of basal hyperglycemia drops from ~80% to <50%. However, the basal glucose contribution to overall hyperglycemia remains higher in patients with the smallest improvement in HbA_{1c} and still accounts for about one-third of the additional hyperglycemic exposure (2). Our opinion is that these findings are not surprising because it has been demonstrated that a substantial percentage of patients did not attain the objectives with a treat-to-target basal insulin regimen (3). As a consequence, no one can deny that more attention should be paid to both basal and prandial hyperglycemia in order to achieve optimal glycemic control. Such an approach is also in agreement with the data obtained at the 3rd year of the Treating To Target in Type 2 Diabetes (4-T) study (4). Another point of the article by Riddle et al. (2) concerns the PPG contributions to overall hyperglycemia, which appear much lower (20–24%) than those observed in our previous landmark study (1). In addition, despite a tendency toward a greater contribution of postprandial hyperglycemia at lower ranges and of basal hyperglycemia at higher ranges of HbA_{1c} at baseline, these authors do not confirm that contributions of postprandial and basal hyperglycemia are steadily decreasing or increasing, respectively, with worsening diabetic control (1). These results, obtained from a 7-point self-measured glycemic profile, highlight the difficulty of reaching a consensus in terms of calculation of prandial and basal contributions and seem to indicate that the calculation used by Riddle et al. (2) has probably under and overestimated the postprandial and basal contributions, respectively. For instance, postprandial excursions should be quantified by measuring each postprandial increment in excess of preprandial values at breakfast, lunch, and dinner, rather than by taking a single fasting value as reference. As a consequence, the continuous glucose monitoring is at present a unique tool that permits the capture of the entire glycemic profile over 24 h and more specifically during postprandial periods (5). For instance, the use of a discontinuous glucose profile with determination of 2-h postmeal glucose values has certainly failed to record the peak value of postmeal excursions, which usually occurs 60 to 90 min after the beginning of the meal (5). Without sufficient considerations to the above arguments, it is not surprising that the contributions of postprandial and basal hyperglycemia were under or overestimated, respectively. However, the study by Riddle et al. has the merit to point out again that the remnant hyperglycemia, which is frequently observed with insulin regimens using basal strategies or premixed insulins, should be combated by subtly combining basal with prandial insulins.

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