COMMENTS AND RESPONSES

Insulin Response in Relation to Insulin Sensitivity: An Appropriate β-Cell Response in Black South African Women

Response to Goedecke et al.

aving read the article by Goedecke et al. (1) with interest, a number of discrepancies inherent in their conclusions deserve comment. Many of the initial studies reporting reduced insulin secretion in black South Africans were performed several decades ago (2). Since then, considerable lifestyle changes have occurred in this segment of the South African population, including decreased physical activity and increased urbanization and westernization-all of which could promote insulin resistance and possibly improved β -cell development in utero. Indirect evidence of this is the rise in serum lipid levels (cholesterol and triglycerides) documented in black South African diabetic patients between 1976 and 1996 (3). Therefore, attempts to compare current insulin data with previous publications are fraught with difficulty.

Since the study by Goedecke et al. examined young normoglycemic women, extrapolation of their findings to diabetic individuals is speculative. It is conceivable that the compensatory hyperinsulinemia documented in black women will accelerate a decline in their (limited) β-cell functional reserve, as was previously documented in glucose-intolerant and frankly diabetic black subjects (4). Severe insulinopenia in type 2 diabetic black South Africans is supported by several clinical observations: frequent presentation with ketoacidosis; the large proportion of such patients requiring insulin therapy from an early stage to maintain glycemic control (unpublished data); and the rapid decline in fasting plasma C-peptide concentrations within a year of diagnosis (5).

The pathogenesis of type 2 diabetes in black South Africans cannot be inferred from limited studies of insulin responses in young normoglycemic women.

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