COMMENTS AND RESPONSES

Insulin Resistance and Hyperinsulinemia Are Related to Plasma Aldosterone Levels in Hypertensive Patients

Response to Perciaccante et al.

Perciaccante et al. (1) commented on our article (2) and underlined the relevance that an activated autonomic nervous system might have as a potential link between insulin resistance and hypertension. The possibility that sympathetic hyperactivity contributes to the prohypertensive action of insulin resistance and hyperinsulinemia was suggested more than 25 years ago (3). Initial studies had shown that acute insulin infusion increases plasma norepinephrine (NE) levels even when plasma glucose concentration is maintained constant (4) and that overfeeding-induced hyperinsulinemia increases NE turnover (5). Additionally, muscle sympathetic nerve activity had been shown to increase in a dose-dependent manner during a hyperinsulinemic-euglycemic clamp (6), and, more recently, an association between a nondipper status as a reflection of an unbalanced autonomic nervous system and more prominent insulin resistance has been reported in essential hypertension (7).

These findings were challenged by large population studies in which no significant association was found between plasma NE and insulin sensitivity (8). We investigated the relationship between the sympathetic nervous system and parameters of glucose metabolism in 77 patients with essential hypertension; however, we did not observe a relationship between urinary NE and both fasting and postglucose load plasma insulin and *C*-peptide (9).

Thus, we agree that multiple pathophysiological mechanisms, possibly including sympathetic hyperactivity, might be involved in the development and maintenance of hypertension in subjects with insulin resistance (10).

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