Comment on Dickinson et al. (2002)

Dear Editor,

Dickinson et al. (1) found that lean, young South East Asian subjects had reduced insulin sensitivity and marked postprandial hyperglycemia after a carbohydrate challenge compared with matched Caucasian subjects. Visceral adiposity, age, body mass index or birth weight did not account for the differences in insulin sensitivity among these young adults. In the accompanying commentary, Ludwig et al. (2) suggested that intrauterine influences could be responsible for the finding.

High levels and impaired utilization of fatty acids are considered to be etiological factors in insulin resistance (3). Two hypotheses are available to explain the inappropriate release of fatty acids from adipose tissue. According to the first hypothesis, intracellular lipase develops resistance to insulin resulting in release of excess fatty acids from adipocytes (4). The molecular mechanism that leads to the appearance of insulin resistance is not known. According to the second hypothesis, fullness of fat storage capacity makes it difficult for adipocytes to accept more fatty acids. Fatty acids released by intravascular lipase in the vicinity of adipocytes enter the circulation instead (5). This hypothesis is applicable regardless of age, weight, ethnicity or location and size of fat depots of the affected individual. This hypothesis can also explain how a woman’s own birth weight can be strongly and inversely related to her risk of gestational diabetes, which strongly predicts the later development of type 2 diabetes (6).

Mature fat cells release paracrine growth factors to convert preadipocytes to triglyceride-storing fat cells (7). It is possible that the programming of genetic factors that determine fat storage capacity of adipocytes and the availability and rate of conversion of preadipocytes to adipocytes is influenced by intrauterine nutritional status. Insufficient adipose tissue to accommodate the postprandial flux of glucose could be responsible for the finding by Dickinson et al. The finding that insufficient adipose tissue to absorb the postprandial flux in fatty acids in lipodystrophy results in insulin resistance supports this possibility (8).

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