COMMENTS AND RESPONSES

Comment on: Jeffery et al. Age Before Stage: Insulin Resistance Rises Before the Onset of Puberty: A 9-Year Longitudinal Study (EarlyBird 26). Diabetes Care 2012;35:536–541

We were very enthused to read an interesting article in *Diabetes Care* (1) that shows that increased insulin resistance in puberty actually starts as many as 3 to 4 years prepubertally, and more than 50% of this variance is unaccounted for by fat percentage, IGF-1, or age of the individual. We would also like to point out a study by Bavdekar et al. (2) that found increased insulin resistance in 8-year-old children from India; most of them were prepubertal and had increased insulin resistance, which correlated with low birthweight profiles. Also children with short parents had more insulin resistance. The thrifty gene hypothesis surrounding fetal growth aberration has been proposed as an explanation for increased insulin resistance in Indians (3). We would like it if the authors could provide data on the birthweight profiles of the study subjects.

The observation that the BMI standard deviation scores (BMIsds) contributed only 12% of variance in insulin resistance, even though adiposity contributed much more to the variance, highlights the fact that BMI and BMIsds in children are far from accurate, as they fail to differentiate between lean mass and fat mass (4). The readers would like to know which reference standards were used to compute the BMIsds.

The fact that skin-fold thickness, dual-energy X-ray absorptiometry, percent fat, insulin resistance by homeostasis model assessment, IGF-1, and leptin had positively skewed distributions and were log transformed for analysis and children were included in the analyses if they had insulin resistance by homeostasis model assessment measures on at least 5 out of the 10 possible time points (1), leaves us with a significant chance of being erroneous in our overall figures. We would like the authors to tell us what percentage of patients had complete 10-visit data and what percentage of data were missing and was adjusted using statistical tools.

The authors of the study acknowledge that adrenarche could have been a possible explanation for variance in prepubertal insulin resistance; appearance of axillary hair and dehydroepiandrosterone sulfate measurements, which would have been wonderful to look at in longitudinal data, were not done in this study.

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