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REPLY

We thank Dr. Iacobellis for his kind comments and the concerns he raised regarding our study on the effects of obesity on left ventricular (LV) structure and function in young and otherwise healthy women (1). He raises some criticisms that are mostly related to the health status of our subjects. We believe our obese study subjects were "otherwise healthy" for the following reasons: 1) none of them had impaired fasting glucose levels (mean ± SD glucose: 84 ± 9 mg/dl); 2) their mean 2-h glucose level during an oral glucose tolerance test was 114 ± 22 mg/dl within normal limits; 3) none had low-density lipoprotein (LDL) or total cholesterol levels that would be considered "high" per Adult Treatment Panel (ATP) III guidelines (LDL: 117 ± 24 mg/dl; total cholesterol: 184 ± 28 mg/dl); 4) their mean high-density lipoprotein (HDL) (46 ± 11 mg/dl) was similar to that of the insulin-sensitive subjects in Dr. Iacobellis's study (42.3 ± 7.6 mg/dl) (2); 5) their mean triglyceride levels were normal (104 ± 63 mg/dl); only two subjects had high triglyceride levels, and excluding data from these two subjects did not significantly change our results; and 6) none had any nonlipid risk factors for coronary heart disease listed by the ATP III, such as increased age, male gender, family history of premature coronary heart disease, diabetes, thrombogenic state, hypertension, or cigarette smoking. Thus, we are hopeful that after this clarification, Dr. Iacobellis will agree that these subjects can be considered "otherwise healthy," and it is unlikely that the presence of underevaluated co-morbidities could explain the findings of our study.

Iacobellis et al. (2) found that among obese subjects, a subset of those who were insulin resistant exhibited eccentric LV hypertrophy, and in another study obesity was associated with hyperkinetic systole (3). In contrast, we found that the obese but otherwise healthy women exhibited concentric LV remodeling and subtle abnormalities in LV function (decreased diastolic and systolic function by tissue Doppler imaging). Some of the apparent discrepancies between the results of our studies may be attributable to differences in echocardiographic equipment used; methods of quantification of structure and function; and/or differences in gender, age, and duration of obesity of the subjects (1–3). We agree with Dr. Iacobellis that insulin resistance may affect LV structure and function. One plausible scenario is that, as is the case with hypertension, there is a continuum of obesity-related heart disease, with concentric remodeling preceding eccentric hypertrophy and diastolic function preceding systolic dysfunction. If this hypothesis is true, the findings of our study and those of Iacobellis et al. (2,3) may in fact be complementary.

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