Reply to DN Polesel et al

Dear Sir:

We thank Polesel et al for their comments on our study (1) and agree that the relation between obesity and cardiometabolic risk factors is modulated by comorbidities and/or by medications for treating chronic diseases. In this context, as rightly pointed out, sleep disorders are known to be associated with cardiometabolic abnormalities, and because sleep-disordered breathing is more common in overweight/obese individuals, it will certainly influence the association of obesity indexes and cardiometabolic risk factors. Unfortunately, when our study was performed, we did not collect information on diagnoses of sleep disorders, perform polysomnography, or determine by questionnaires whether or not the individuals were at risk of having obstructive sleep apnea (OSA). However, since then, our research group has become interested in these issues; and along with Dr Alice Liu, we are actively investigating associations between OSA, obesity, and cardiometabolic risk factors, with an emphasis on how differences in insulin-mediated glucose disposal influence these relations in nondiabetic individuals. OSA is well established as a comorbidity in obese individuals, yet the breadth of the obesity epidemic and cost of polysomnography are barriers to universal testing. In an attempt to address this issue, we recently published data showing that insulin-resistant, nondiabetic individuals without known OSA were more likely to be at high risk of OSA, as determined by screening questionnaires, than insulin-sensitive individuals of similar adiposity (2). We proposed that administering questionnaires to the insulin-resistant subset of obese individuals would be a clinically useful and a cost-effective way to identify those at the greatest risk of having OSA who will benefit the most from referral for polysomnography. Short sleep duration has also been reported to be associated with obesity and dysglycemia (3, 4). We extended these findings by showing in an obese group of subjects with a high prevalence of impaired fasting glucose and glucose intolerance that insulin resistance was independently associated with habitual shortened sleep, defined as fewer than 7 h of sleep per night (5). Thus, our findings are consistent with the notion that disordered sleep may play an important role in modulating cardiometabolic diseases. That said, it is doubtful that accounting for OSA diagnoses in our present study population would have altered our primary results—namely, that BMI and waist circumference were similar in their associations with markers of increased cardiometabolic risk. In both studies referenced above (2, 5), BMI and waist circumference did not differ between insulin-resistant and insulin-sensitive groups, suggesting that the differences seen in OSA risk and sleep duration were attributed to insulin resistance rather than obesity per se. Nonetheless, further studies are necessary to characterize these relations.

Polesel et al’s comments with regard to age and the visceral adiposity index (VAI) are also of interest. The median (IQR) age of our study participants was 51 (44–57) y, and 85% of the participants were younger than 60 y, indicating that our sample did not include a large proportion of older individuals who are more prone to developing age-related diseases. The VAI is based on measurements of waist circumference, BMI, and triglyceride and HDL-cholesterol concentrations (6). We have been interested in the utility of the plasma concentration ratio of triglyceride:HDL-cholesterol to identify apparently healthy individuals at increased risk of cardiovascular disease (7–10), and the comments of Polesel et al have stimulated us to the point that we are initiating efforts to compare the relative abilities of the VAI and the triglyceride:HDL-cholesterol ratio to identify individuals at increased cardiometabolic risk.

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REFERENCES
