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## Reply



### To the Editor:

We thank Schikler for his comments. We did not collect data about sex maturity ratings or joint hypermobility, which could be done in future studies, but we do not believe that these influence our results. In fact, there is no clear effect of sport on sex maturity ratings, and a large number of papers conclude that only highly challenging sports, such as artistic and rhythmic gymnastics but not swimming, have an impact on growth during puberty.<sup>1,2</sup> Some studies have demonstrated a delayed age of menarche in competitive swimmers,<sup>3,4</sup> and others have found no differences,<sup>5</sup> or attributed the differences to familial characteristics more than the effect of training.<sup>6</sup> It is possible that athletes achieve premature maturation, but we do interpret that evidence to be consistently true. With regards to joint hypermobility, a correlation with low back pain during adolescence has not been found, but identified only with other pain localization.<sup>7</sup> The work of Kim et al does not generalize to our population because it included only young adult males and not adolescents.<sup>8</sup> Joint hypermobility in swimming has been demonstrated to be due partially to inherent factors and partially to training,<sup>9</sup> but this feature is also present in many patients suffering adolescent idiopathic scoliosis.<sup>10</sup> These findings could relate to common features, explaining the association found in our study between swimming and trunk asymmetries. It is also possible that our study included a self-selected population, but we recruited only adolescents performing competitive swimming, not elite athletes.

In conclusion, we do not think that the points you raise affect our results. Our study aimed to determine associations and not causal effects, because of its cross-sectional design.

Fabio Zaina, MD  
Sabrina Donzelli, MD  
Monia Lusini, MD  
Salvatore Minnella, MD  
ISICO, Italian Scientific Spine Institute  
Milan

Stefano Negrini, MD  
Physical and Rehabilitation Medicine  
Department of Clinical and Experimental Sciences  
University of Brescia  
Brescia, Italy

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## Anthropometric indices and metabolic co-morbidities



### To the Editor:

Morandi et al<sup>1</sup> have demonstrated that commonly used anthropometric indices, Z-score of the body mass index (BMI), waist circumference, and waist-to-height ratio, are not satisfactory markers of metabolic disturbances among obese children and adolescents. Certainly, children with the same level of adiposity vary regarding the presence and level of cardiometabolic risk factors. The duration of obesity and the speed of weight gain could be important determinants of metabolic alterations, regardless of the present weight status. Factors beyond anthropometry (ie, lifestyle and family history of type 2 diabetes mellitus) also could modulate metabolic measures.<sup>1-3</sup> From this perspective, based on a longitudinal population-based study,<sup>4</sup> we tested the hypothesis that the degree of cardiometabolic disturbance associated with obesity varies depending on the timing of excessive weight gain.

Overweight or obese children at 12 years of age (BMI  $\geq$ 85th percentile: 44 of 280 children in the cohort) were divided into 2 groups depending on the timing of the adiposity rebound: before 4 years of age ( $n = 30$ ) and after 5 years of age ( $n = 14$ ). We then compared cardiometabolic risk factors between the groups. Although the mean BMI at 12 years of age was not different ( $27.4 \pm 3.7$  vs  $26.3 \pm 2.2$  kg/m<sup>2</sup>,  $P = .315$ ), overweight/obese children who developed adiposity earlier ( $\leq 4$  years of age) showed more atherogenic lipid patterns compared with overweight/