Response to “Low Serum Adiponectin Levels and Endothelial Dysfunction in Childhood Hypertension”

Paolo Brambilla, Laura Antolini, Maria Elisabeth Street, Marco Giussani, Sara Galbiati, Maria Grazia Valsecchi, Andrea Stella, Gian Vincenzo Zuccotti, Sergio Bernasconi, and Simonetta Genovesi

To the Editor: We recently published a paper showing an inverse relationship between adiponectin serum levels and hypertension, partially independent of obesity, fat distribution, and insulin resistance. Our results indicated that low adiponectin values in obese, but also in normal weight, children are associated with a higher probability of hypertension, suggesting that adiponectin might be responsible for a mechanism leading to hypertension, which would begin in childhood.

In his letter, “Low Serum Adiponectin Levels and Endothelial Dysfunction in Childhood Hypertension,” Tsuda suggested that one of the mechanisms underlying the cardiovascular protective effects of adiponectin may be the enhancement of nitric oxide (NO) production. The author supported this hypothesis on the basis of previous studies conducted in hypertensive and normotensive adults. According to these studies, it was supposed that adiponectin might have a beneficial effect on rheologic behavior and microcirculation through an NO-dependent mechanism.

We greatly appreciate the interest of Tsuda for our work. Unfortunately, in our study population we did not evaluate any marker of endothelial function or the endothelial-dependent brachial flow-mediated dilatation, and consequently we could not add any information on this topic.

Studies in adults have recently shown a direct vasculoprotective effect of adiponectin. However, studies evaluating the relationship between adiponectin and endothelial function in children are still controversial. Singhal et al. did not find a relationship between adiponectin and flow-mediated endothelial-dependent vasodilation or arterial distensibility in a cross-sectional study conducted in a healthy, nonobese population of adolescents. This finding seems to suggest that adiponectin does not exert an early influence on the development of endothelial dysfunction and the atherosclerotic process. On the other hand, Juonala et al. showed in a longitudinal cohort study in young adults that individuals showing a metabolic syndrome were more vulnerable to the proatherogenic effects of low adiponectin levels.

It has been shown that other circulating adipose-derived biomarkers (e.g., cystatin C) and markers of inflammation (e.g., resistin) are related to vascular alterations in obese children and could be implicated in obesity-related comorbidities. It is possible that interrelationships among all these factors and other adipose-derived cytokines may occur, leading to the development of early atherosclerosis in children and adolescents. We cannot exclude, however, that the first steps of the endothelial dysfunction process could be different in childhood in comparison with adulthood.

Only longitudinal, ad hoc–designed studies could demonstrate whether low adiponectin levels may predict alterations in endothelial function later in life.

DISCLOSURE

The authors declared no conflict of interest.

REFERENCES