Cardiotrophin-1 in Adolescents: Impact of Obesity and Blood Pressure

To the Editor:

With great interest we read the article by Malavazos et al discussing the possible pathophysiological role for cardiotrophin-1 (CT-1) in left ventricular growth. CT-1 is a member of the interleukin-6 superfamily, which induces cardiomyocyte growth. CT-1 provides prognostic information in patients with untreated essential hypertension and associates with the magnitude of left ventricular hypertrophy in these patients. In contrast, plasma CT-1 is decreased in hypertensive rats.

Recently, Natal et al identified adipose tissue as a source of CT-1. They found an elevated level of CT-1 in adult patients affected by the metabolic syndrome. This observation raised the possibility that CT-1 may play a pathophysiological role in metabolic syndrome, acting as a link between obesity-related complications and cardiovascular diseases. In vitro studies demonstrated that chronic administration of CT-1 to adipocytes resulted in the development of insulin resistance.

Overweight and related diseases are dramatically increasing problems in adolescents. Therefore, we investigated CT-1 concentrations in adolescents.

Seventy-two male, white adolescents (aged 13 to 17 years) were studied. Thirty seven (51%) were overweight according to the age-specific weight percentiles. Subjects and their parents gave informed consent, and protocols were approved by the university ethics committee in accordance with the Helsinki Declaration. The quantitative determination of human CT-1 (Antigenix America) and adiponectin (R&D) plasma concentration was performed using the ELISA technique. Data are expressed as means±SDs. Groups were compared by Mann–Whitney U test. For various risk factors, Pearson’s correlation coefficient was calculated. Statistical significance was assumed at P≤0.05. Statistical analysis was performed with SPSS 12.0 (SPSS Inc).

Overweight adolescents (0.43±1.78 ng/mL) and normal-weight (1.67±3.79 ng/mL) adolescents do not differ in their level of CT-1. Neither body mass index nor waist circumference correlate with plasma levels of CT-1. Systolic blood pressure at rest correlates inversely with CT-1 (P=0.002; R: −0.459). Diabetes mellitus in the families’ history correlates with higher CT-1 (P=0.028). CT-1 does not correlate with HbA1c or adiponectin.

Increased CT-1 levels could not be observed in obese teenagers compared with control subjects, in contrast to adults. It seems that CT-1 is not the link among fat tissue, insulin resistance, and cardiovascular disorders, because CT-1 does not correlate with HbA1c or adiponectin. However, there seems to be a hereditary disposition.

In contrast to adults, CT-1 is in inverse correlation with systolic blood pressure. In animal models with decreased CT-1 in hypertension, hormonal points of difference are discussed, which may also be the underlying mechanism in adolescents. This needs further investigation to reveal the mechanisms leading to morbidity in early life.

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Disclosures

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Christian Jung
Michael Fritzenwanger
Hans R. Figulla
Friedrich-Schiller-University
Clinic of Internal Medicine I
Jena, Germany

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Christian Jung, Michael Fritzenwanger and Hans R. Figulla

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