



Cardiovascular Dysfunction in Childhood Obesity and CVD Risk Factors in the Paediatric Population

Simmons AB*

Division of Biological and Biomedical Sciences, Emory University, Georgia

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Introduction

Dylipidemia, hypertension, and insulin resistance are some of the cardiovascular consequences of childhood obesity. Although the evidence directly connecting obesity as an independent risk factor to CVD in children is not currently as robust as it is in adults, there is increasing evidence that obesity may be an independent risk factor.

Dylipidemia

Children who are overweight or obese are more likely to have an aberrant lipid profile, particularly if their body fat percentage or waist circumference are higher. High triglyceride levels and low high-density lipoprotein cholesterol levels are common in the lipid profile. Although low-density lipoprotein cholesterol levels are typically normal, there may be qualitative changes in the particles, causing them to become smaller, more dense, and more atherogenic [1,2].

Hypertension

Obese kids are three times as likely than nonobese kids to develop hypertension. Because conventional office BP readings may miss it, this is more likely to be noticed during ambulatory blood pressure monitoring. Ambulatory BP monitoring, rather than clinical BP monitoring, is thought to be more reliable because it describes BP variations more precisely throughout the day [3].

Insulin resistance and the metabolic syndrome

The metabolic syndrome is a term used to describe a grouping of metabolic risk factors in adults, such as hypertension, dyslipidemia, poor glucose tolerance, and abdominal obesity. Youngsters are similarly affected by this clustering of numerous risk factors, with obese children being the most vulnerable. The metabolic syndrome has been linked to an increased risk of cardiovascular disease in adults. Although being overweight as a kid is linked to high levels of insulin, lipids, and blood pressure in early adulthood, the long-term impact on children is unclear [4,5].

A nonlinear connection was found between BMI and aberrant lipid levels, hyperinsulinemia, and hypertension in children in the Bogalusa Heart Research (29), a long-term population-based study that monitored the natural history of CAD in Bogalusa, Louisiana (USA). At very high BMI levels, the prevalence of these risk factors increased substantially among the five to 17-year-old children investigated. BMI was most significantly related with insulin levels in the Bogalusa Heart Study, with 40 percent of children with a BMI greater than the 99th percentile having a high insulin level. Type 2 diabetes may be preceded by insulin resistance and hyperinsulinemia. The frequency of type 2 diabetes in children and adolescents has risen substantially, and the majority of those diagnosed in childhood are obese.

Other CVD risk factors linked to childhood obesity include left ventricular hypertrophy and elevated homocysteine, C-reactive protein, and lipoprotein levels. CVD risk may also be predicted by

certain phenotypes. Youth who were overweight had elevated CVD risks, and those who were obese had even larger risks. compared to youth who were of normal weight. Left ventricular mass index, cardiac output, nightly sodium excretion, and ambulatory and nocturnal blood pressure data were all used to predict CVD risk.

Mechanisms of CV Dysfunction in childhood obesity

Obesity may alter early stages of atherosclerosis (endothelial function, arterial stiffness, adhesion molecule expression, foam cell formation, and smooth muscle proliferation) as well as later changes in atherosclerosis (endothelial function, arterial stiffness, adhesion molecule expression, foam cell formation, and smooth muscle proliferation) (plaque rupture and thrombosis). Recent research has focused on new mediators of CV dysfunction in obese people. VAT dysfunction (as part of 'adiposopathy') may be a significant source of inflammatory, oxidative stress, and angiogenesis mediators. Adipocytokines are a type of mediator that can operate locally in an autocrine or paracrine manner or more distantly in an endocrine manner [6].

Physiological levels of adiponectin inhibit monocyte adhesion to human aortic endothelial cells, down-regulate the expression of adhesion molecules on these endothelial cells, suppress human aortic smooth muscle cell proliferation and migration, inhibit macrophage foam cell transformation, and increase the production of tissue inhibitor of metalloproteinase-1 (an important regulator of extracellular matrix degradation). Control Apo-E deficient animals treated with recombinant adenovirus that expresses human adiponectin have much less atherosclerosis than Apo-E deficient mice treated with recombinant adenovirus that expresses human adiponectin. Low adiponectin levels have been linked to higher levels of hs-CRP, endothelial dysfunction in adults and children, increased coronary artery calcification progression, 65 thicker carotid IMT in adolescents, and women in human studies. Adiponectin's activities have regularly been found to be anti-atherosclerotic in several trials.

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*Corresponding author: Simmons AB, Division of Biological and Biomedical Sciences, Emory University, Georgia; E-mail: SimmonsAB@emory.edu

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