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The relative impact of different measures of adiposity on markers of early atherosclerosis

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Abstract

Background: Although there are several methods available to assess adiposity, there is still controversy on the relative clinical utility of each of these methods. This study examines the relative impact of different measures of adiposity on markers of early atherosclerosis. In particular weight changes over time have been poorly assessed in this setting.
Methods: Eighty-six healthy individuals (31 men, age 36.5±8.9 years) with a wide range of body-mass index (28.7±7.0, 18.9–57.9 kg/m^2) without hypertension, diabetes or smoking were examined. In addition to waist circumference and waist-to-hip ratio self-reported weight change since adolescence was also calculated. Ultrasonography was used to measure abdominal fat layers and their ratio. Flow-mediated dilatation of the brachial artery, serum levels of intercellular adhesion molecule (sICAM-1) and mean intima-media thickness of the carotid artery were measured as markers of early atherosclerosis.
Results: Stepwise multivariate regression analysis showed waist circumference and waist-to-hip ratio as the only independent predictor of flow-mediated dilatation. Waist circumference and weight change but not current body-mass index were independent predictors of intima-media thickness. These correlations were not influenced by ultrasonographically measured fat layers, C-reactive protein and basal insulin resistance. Body-mass index and weight gain were associated with sICAM-1 but not independently of basal insulin resistance and C-reactive protein.
Conclusions: Waist circumference and weight gain were the strongest predictors of early atherosclerosis in a population of apparently healthy adults. The ultrasonographically measured fat layers did not provide additional information in this population.
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Keywords: Obesity; Measures of adiposity; Endothelial function; Intima-media thickness; Endothelial activation

1. Introduction

Increased body-mass index is associated with increased risk for development of hypertension and diabetes as well as cardiovascular morbidity and mortality [1–5]. Other markers of obesity have also been related to high cardiovascular risk irrespective of body-mass index. Healthy adults with central body fat distribution are at higher risk for cardiovascular mortality and development of clinical diabetes [6,7]. Modest long-term increases in body weight are also associated with increased incidence of hypertension as well as increased cardiovascular mortality and morbidity in healthy and diabetic adults [8–10].

Increased cardiovascular risk in subjects with increased fatness may be attributed to accelerated atherosclerosis via endothelial dysfunction. Obesity may lead to endothelial dysfunction through development of hypertension, diabetes mellitus and other early metabolic abnormalities like insulin resistance and dyslipidemia [11–14]. Furthermore, obesity may also be directly related to atherosclerosis possibly

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through increased expression of various proinflammatory cytokines like tissue necrosis factor-a and interleukin-6 [15–17]. These factors, which are mostly associated with regional rather than general obesity [18,19] are directly related to endothelial dysfunction [20], an early stage of atherosclerosis [21], as well as to upregulation of endothelial adhesion molecules and C-reactive protein [22]. The latter may be of particular significance given the well established role of C-reactive protein in cardiovascular disease [23] and that of intercellular adhesion molecule-1 (ICAM-1) and vascular adhesion molecule-1 (VCAM-1) as markers of endothelial activation and important mediators in atherogenesis [22].

Although several markers of obesity have been used to assess the severity of adiposity, there is still controversy as to which best predicts cardiovascular risk; it is therefore not clear which of the current simple or more sophisticated and complex markers should be used during clinical counseling for cardiovascular disease prevention. In some but not all studies, body-mass index, a marker of general adiposity, has been related to both endothelial dysfunction and increased carotid intima-media thickness, a structural surrogate marker of atherosclerosis [13,24–29]. However, in these studies, a co-existing metabolic disorder was almost always present in subjects with increased body-mass index [13,25] while other indices of adiposity related to body fat distribution showed more potent and independent associations with premature atherosclerosis[26–28,30]. Moreover, even though several expensive and time-consuming imaging techniques have been used to accurately measure regional adiposity, no clear superiority has been shown over a simple and easy to take measurement of waist and hip circumference and their ratio [26,28,30,31]. Finally, not much information is available on the impact of the long-term history of weight change on markers of premature atherosclerosis, nor are there data on its utility compared to markers of current adiposity [32]. This cross-sectional study examines the relative contribution of different indices of adiposity on surrogate markers of atherosclerosis with established clinical value in cardiovascular disease in a population of healthy young and middle aged adults with different grades of obesity without other cardiovascular risk factors. Assessment of this sample would allow identification of markers of adiposity associated with early cardiovascular damage coming from obesity per se prior to development of other associated abnormalities.

2. Materials and methods

2.1. Patients

All subjects gave informed consent before entering the study and the local ethics committee approved this protocol. Eighty-six young and middle aged Caucasian volunteers were recruited from our local hospital and clinic support staff and nurses with a wide range of body-mass index (18.9 to 57.9 kg/m^2) without overt cardiovascular disease, were enrolled. Only pre-menopausal women were enrolled in the study. In order to avoid bias during the assessment of the impact of adiposity on cardiovascular system, subjects with other cardiovascular risk factors were excluded; i.e. hypertension, defined as systolic and diastolic blood pressure of more than 139 mmHg and 89 mmHg, respectively; current and recently ceased smoking, defined as having ceased for less than 6 months; and diabetes mellitus, defined as fasting blood glucose levels of more than 126 mg/dl. Mild hypercholesterolemia, defined as total blood cholesterol of less than 240 mg/dl was allowed for enrolment in order to include subjects with morbid obesity which is often accompanied by dyslipidemia.

Baseline characteristics are shown in Tables 1 and 2. None of the volunteers was under any medication. Blood sampling and vascular studies were performed at the same time of the day (9 to 12 am). All subjects abstained from food, caffeine or alcohol for at least 8 h. Women were examined during any day of their menstrual cycle except from the M phase, during which endothelial function is significantly impaired compared to the other phases [32].

2.2. Anthropometric measures

Weight change since adolescence and body-mass index were used as measures of general adiposity. Weight change was calculated from the difference of current weight in kilograms minus self-reported weight at age 18. Long-term recalled weight has been previously shown to correlate well (r=0.82 to 0.87) with measured weight at that time [34,35].

Table 1
Characteristics of the population

Variables	
Age (yrs)	36.5±8.9
Men/women	31/55
Weight (kg)	82±23.4
Body-mass index (kg/m^2)	28.6±7.0
Waist circumference (cm)	94.0±18.2
Waist to hip ratio	0.86±0.08
Family history for coronary heart disease (%)	18.5%
Weight gain (kg)	15.5±15.9
Weight change (n, lost/stable/gained)	2/17/67
Systolic blood pressure (mmHg)	109.8±15.4
Diastolic blood pressure (mmHg)	70.4±12.3
Pre-peritoneal fat thickness (mm)	15.5±5.5
Subcutaneous fat thickness (mm)	12.1±7.3
Abdominal fat index	1.86±1.85
Total cholesterol (mg/dl)	188.3±33.9
Triglycerides (mg/dl)	91±44.9
HDL cholesterol (mg/dl)	55.8±14.8
LDL cholesterol (mg/dl)	131.4±35.9
Glucose (mg/dl)	84.5±10.7
Fibrinogen (mg/dl)	2.9±0.6
C-reactive protein (mg/l)	1.31±1.75
Insulin (IU/ml)	9.25±6.3
Homeostasis Model Assessment (IU mg/ml)	1.99±1.6

Values are expressed as mean±SD in continuous variables. Weight change was defined as follows: lost<−3 kg, stable=−3 to +4 kg, gained>4 kg.

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