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Association of body mass index with arterial stiffness and blood pressure components: A twin study

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ABSTRACT

Rationale: Obesity, blood pressure and arterial stiffness are heritable traits interconnected to each other but their possible common genetic and environmental etiologies are unknown.
Methods: We studied 228 monozygotic and 150 dizygotic twin pairs aged 18–82 years from Italy, Hungary and the United States, of which 45 monozygotic and 38 dizygotic pairs were discordant for body mass index (BMI; intrapair difference (Δ) in BMI $\geq$ 3 kg/m^2). Blood pressure components and arterial stiffness were measured by TensioMed Arteriograph.
Results: Hypertension was more prevalent among obese than non-obese individuals (55% vs. 29%, $p < 0.001$). Age-, sex- and country-adjusted heritability estimates were high for hemodynamic measures (45%–58%) and BMI (78%). According to bivariate Cholesky decomposition, phenotypic correlations between BMI and blood pressure components ($r = -0.15$ to 0.24, $p < 0.05$) were largely explained by additive genetic factors (65%–77%) with the remaining explained by the unique environment. When controlling for genetic factors within all monozygotic pairs, ΔBMI was significantly correlated with Δbrachial systolic blood pressure (SBP) and diastolic blood pressure (DBP), Δmean arterial pressure, and Δaortic SBP ($r = 0.15$–0.17, $p < 0.05$). For the same measures, heavier co-twins of BMI-discordant monozygotic pairs had significantly higher values than their leaner counterparts ($p < 0.05$).

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Conclusion: Blood pressure components are moderately correlated with BMI, largely because of shared genetic factors. However, for the association of BMI with brachial SBP and DBP, aortic SBP and mean arterial pressure, acquired, modifiable factors were also found to be important.

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1. Introduction

Obesity, a complex condition of excessive fat accumulation, caused by several genetic and non-genetic risk factors, reached epidemic proportions worldwide, with more than one billion overweight adults of which at least 300 million are clinically obese [1]. Obesity poses a major risk for cardiovascular disease, hypertension, and stroke [2]. Although weight gain has long been associated with increased blood pressure [3], pathophysiologic mechanisms underlying this link are not well understood. The relationship between increases in adiposity and elevations in blood pressure may involve sympathetic nervous system activity, alterations in renal handling of sodium and water, insulin sensitivity and fatty acid metabolism, with some of these alterations being induced by hormones, growth factors, and cytokines expressed by adipose tissue in response to its expansion [4]. It is also evident that body mass index (BMI) correlates with atherosclerotic phenotypes, and research has increasingly begun to explore pathways through which body composition might influence cardiovascular disease [5,6]. However, it is not clear why the association between obesity and cardiovascular risk is not as strong as it might be expected, and why many obese individuals remain normotensive.

Family, adoption, and twin studies provide strong evidence that genetic factors contribute substantially to individual differences of BMI [7–10]. The role of heredity of blood pressure and arterial compliance is also evident. Twin and nuclear family studies [11–13] have shown that genetic variance explains between 30% and 71% of the variability of brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) [14], while heritability of parameters related to arterial elastic properties, such as pulse pressure, central blood pressure and arterial stiffness range between 24% and 63% [15–18]. In fact, most previous studies estimated the heritability of brachial augmentation index (AIx), aortic AIx and aortic pulse wave velocity (PWV) between 37% and 53% [15,16,19–21].

As both obesity and hemodynamic variables are heritable, common genetic factors may explain associations between BMI and blood pressure components. In a large sample of Han Twins from the Chinese National Twin Registry, Wu et al. [22] recently reported that positive correlations of SBP and DBP with BMI are largely attributable to genetic factors. In the same study, however, similarly to another study conducted among adult African–American twins [23], BMI shared a minor fraction (<7%) of its genetic variance with DBP and SBP.

It is possible that acquired obesity, resulting from unhealthy lifestyle regardless of genetic predisposition, contributes to the development of hypertension. However, in a previous study utilizing young obesity-discordant monozygotic (MZ) twins, blood pressure was comparable in obese and non-obese co-twins with impairment of endothelial function being evident in acquired obesity only in conjunction with adiponectin deficiency [24]. To our knowledge, the genetic and environmental architecture of obesity and hemodynamic variables other than SBP and DBP has not yet been investigated.

The purpose of the present study was to estimate the extent to which genetic and environmental factors contribute to the association of obesity with blood pressure and parameters related to arterial compliance using an international twin sample. The second aim was to examine the effects of acquired obesity on these parameters after completely matching for the genetic background in weight-discordant MZ twin pairs. We used a broad panel of hemodynamic variables ranging from traditional parameters such as brachial SBP and DBP to more specific early markers of cardiovascular risk such as central blood pressure, pulse pressure, augmentation index and aortic pulse wave velocity [25].

2. Methods

2.1. Subjects and study design

Data from 378 twin pairs (228 MZ, 150 dizygotic, DZ (88 same-sex DZ); 156 Hungarian, 49 American and 173 Italian; age range: 18–82 years) were analyzed in this classical twin study. Hungarian twins were measured during two Hungarian twin festivals (Agfalva, Szigethalom) or at two large hospitals in Budapest enrolled by the Hungarian Twin Registry [26]. American twins were tested at the Twins Day Festival in Twinsburg, OH, United States. Italian twins were identified through the Italian Twin Registry [27] and were tested in Rome, Padua, and Perugia. Body composition, arterial stiffness and blood pressure measurements were facilitated by two of the authors (ADT and DLT) at all research sites by the same devices in order to reduce inter-observer variations. In the absence of genotyping data and in order to maximize the accuracy of zygosity classification, we used a multiple self-reported question approach to assess zygosity. The most likely zygosity was assigned based on the seven self-reported responses, which included questions on the similarities of twins during their childhood [28,29]. Exclusion criteria were race other than white (to exclude the influence of ethnicity), pregnancy, and arrhythmia. All subjects were restricted from smoking for 3 h, from eating for 1 h, and from drinking alcohol or coffee for 10 h prior to measurements. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and Title 45, U.S. Code of Federal Regulations, Part 46, Protection of Human Subjects, Revised November 13, 2001, effective December 13, 2001; furthermore, all procedures involving human subjects were approved by the local ethical committees. Written informed consent was obtained from all study subjects.

2.2. Assessment of body mass index, waist circumference smoking and disease status

Weight measurements were carried out by a clinically validated OMRON BF500 body consistency monitor (Omron Healthcare Ltd., Kyoto, Japan). Height was measured with a wall-mounted tape. BMI was derived from the formula weight(kg)/height(m)^2. The BMI cut-off for obesity was $\geq$30 kg/m^2. Waist circumference was measured by a tape at the level of the top of the hip bone by positioning the tape horizontally in standing position. Smoking was assessed by questionnaire. The group of non-smokers comprised never smokers or former smokers (84.1% of participants) and the group of smokers included current smokers (15.9%); 4.6% of study subjects reported previous myocardial infarction or coronary artery disease, while 0.9% and 1.8% had stroke and peripheral artery disease, respectively.

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