Blood Pressure, Body Composition, and Fat Tissue Cellularity in Adults

ROGER M. SIERVOGEL, PH.D., ALEX F. ROCHE, M.D., PH.D., D. SC.,
WILLIAM CAMERON CHUMLEA, PH.D., JAMES G. MORRIS, M. STAT.,
P AUL WEBB, M.D., AND JEROME L. KNITTLE, M.D.

SUMMARY While total body mass has been recognized for many years as having a strong positive association with blood pressure, it is not known whether body mass per se is important in this association or if a component of total mass, such as lean body mass or total body fat, is responsible for the relationship. To determine the relative importance of these components, 217 healthy adults unselected with respect to blood pressure or body composition, who were 18 to 49 years of age (X± SD = 33.1 ± 8.9 years) had body density measured by underwater weighing. Body density was used to estimate percent body fat from which lean body mass and total body fat mass were determined. In addition, an adipose tissue biopsy from the gluteal area was made, and average fat cell size was measured using the osmium tetroxide method; fat cell number was then estimated. In both males and females, after adjusting for age, there were highly significant, positive partial correlations (r = 0.2 to 0.3) between measures of blood pressure (systolic, diastolic, and mean arterial) and percent body fat, total body fat mass or fat cell number. No significant correlation existed between lean body mass or fat cell size and any measure of blood pressure. These findings suggest that fat mass as opposed to body mass, per se, may be an important etiological component in elevated blood pressure in adults. (Hypertension 4: 382-386, 1982)

KEY WORDS • lean body mass • total body fat • fat cells • adipose tissue • essential hypertension • blood pressure

NUMEROUS variables are related to blood pressure, but associations are particularly strong with body size and fatness.1-4 The separate influences of these factors and the relationships of blood pressure to fat cell size and number are unknown. It is well established, however, that elevated blood pressure alone is an important risk factor for coronary heart disease,5 and there are strong, positive associations of obesity with hypertension and mortality from cardiovascular disease in adults.6,7 A positive correlation between blood pressure and body weight has long been recognized in adults,1, 2, 4, 6 and studies involving overweight hypertensive patients have shown that weight reduction is commonly associated with a decrease in blood pressure.8-11 Moreover, age, weight, stature, various indices combining weight and stature, and other measures of body fatness are determinants of blood pressure in children.12-16 Effects of age on blood pressure have been demonstrated in adults, also,7, 17 but stature in adults has little or no influence on blood pressure.1, 11, 17, 18

Few previous studies of the determinants of blood pressure have been based on measures of body composition that allow a separation of total body mass into lean body mass and total body fat. Consequently, the possible effects of these two factors on blood pressure have not been separated. In the present study, body weight was partitioned into lean body mass (LBM) and total body fat mass (FM). The relationship between each of these factors and blood pressure was investigated, in addition to the relationships between blood pressure and measurements of fat cell size (FCS) and estimates of fat cell number (FCN).
Materials and Methods

The participants in this research are members of the Fels Longitudinal Study. In general, participants in the Fels Longitudinal Study are white and live in Southwestern Ohio; they were born between 1929, when the study began, and 1974, when the last participant was enrolled. About 15 participants each year were enrolled in the study while in utero, that is, while the mother of the participant was pregnant. Selection was based principally on willingness to participate in the serial study, which, in general, involved semiannual examinations through age 18 years, and less frequent visits thereafter.

About 35% of the participants live in cities of medium size (population 30,000 to 60,000) and most of the rest live in smaller communities (population 500 to 10,000). About 15% of the participants' fathers are professionals or major executives, 35% businessmen, 35% tradesmen or white collar workers, and the remaining 15% skilled or semiskilled workers. About 60% of the participants' parents attended college for 1 year or more, and about 60% of them were born in Ohio. The present analyses are based on recent cross-sectional data from 217 of the participants 18 to 49 years of age (X ± SD = 33.1 ± 8.9 years).

Body density was determined by hydrostatic weighing using body weight in air, body weight in water, water density, and residual lung volume.19 Residual lung volume was measured to the nearest 0.001 liter by closed circuit oxygen dilution.20 Body density was used to estimate percent body fat by applying the equation of Siri;21 then FM and LBM were computed from percent body fat and total body weight. Adipose tissue was collected from the gluteal area by needle aspiration,22 and FCS (average lipid weight in micrograms) was collected from the gluteal area by needle aspiration.23 Fat cell number (FCN) was estimated by dividing FM by FCS.

Systolic blood pressure (SBP) and fifth phase diastolic blood pressure (DBP) were measured in seated subjects by trained observers. A rigorously standardized protocol similar to that used in the Multiple Risk Factor Intervention Trial was followed.14 As part of the procedures, arm sizes were measured and appropriate cuffs used; the cuffs were long enough to completely encircle the arm and wide enough to cover two-thirds the length of the upper arm. The values used in the present study are the means of the last two of three measurements made with a standard mercury sphygmomanometer during a single examination visit. Pulse pressure (PP = SBP - DBP) and mean arterial blood pressure (MAP = 0.33 SBP + 0.67 DBP) were calculated.

Results

Effects of age on blood pressure have been identified;2, 8, 17 therefore, it is important to investigate and remove age effects before attempting to determine the relationships between body composition and blood pressure. Sex-specific multiple regression analyses of the body composition and blood pressure variables on age, age2, and age3 were performed to determine the effects of age on these variables. Quadratic and cubic functions of age were included to allow for all possible age effects that could reasonably be expected.

As shown in table 1, the coefficient of determination (R2) indicated that an appreciable proportion of the total variation in some variables was "accounted for" by age effects. The R2 values may be interpreted as indicating the proportion of the total variance of the outcome measure (SBP, DBP, etc., in this case) that is explained by age. Therefore, it is a measure of the importance of age in determining the value of the measure. For SBP, DBP, and MAP, there were

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (n = 111)</th>
<th>Women (n = 106)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td>114.8 ± 11.6</td>
<td>105.1 ± 11.1</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>67.9 ± 10.1</td>
<td>63.9 ± 9.2</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>83.6 ± 9.2</td>
<td>77.7 ± 9.0</td>
</tr>
<tr>
<td>PP (mm Hg)</td>
<td>46.9 ± 11.0</td>
<td>41.2 ± 8.6</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>19.4 ± 5.8</td>
<td>27.8 ± 7.2</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>15.7 ± 6.5</td>
<td>17.4 ± 7.0</td>
</tr>
<tr>
<td>LBM (kg)</td>
<td>61.7 ± 7.4</td>
<td>43.7 ± 4.9</td>
</tr>
<tr>
<td>FCS (µg)</td>
<td>0.43 ± 0.15</td>
<td>0.47 ± 0.16</td>
</tr>
<tr>
<td>FCN (X 109)</td>
<td>39.1 ± 17.5</td>
<td>39.6 ± 16.5</td>
</tr>
</tbody>
</table>

Values are given as means (X) and standard deviations (SD) of the variables; R2 is the coefficient of determination and represents the proportion of the total variation "accounted for" by age. Significance levels (p) are for the t test of the null hypothesis of no sex difference between the means of the age-adjusted variables.

Abbreviations: SBP = systolic blood pressure; DBP = fifth phase diastolic blood pressure; MAP = mean arterial blood pressure; PP = pulse pressure; FM = total body fat mass; LBM = lean body mass; FCS = fat cell size; FCN = fat cell number.
significant age effects in each sex, being slightly greater in men. Pulse pressure (PP) showed little age effect in either sex. It is interesting that percent body fat, FM, and FCN showed little or no age effects in women, but substantial effects in men. LBM and FCS, however, showed only small age effects in each sex.

Also shown in table 1 are the sex-specific means and standard deviations of the age-adjusted variables. Generally small but highly significant differences between the means occurred for all variables except FM, FCS, and FCN. Men tended to have higher blood pressures and larger LBM, while women tended to have a larger percent body fat, associated with greater FM and FCS.

Partial correlations (age effects removed) between blood pressure and body composition variables were performed sex specifically. The relationship between body composition and blood pressure is apparent from the partial correlations shown in table 2. In men, SBP, MAP, and PP were significantly positively correlated with percent body fat. In women the picture was similar, except that SBP, MAP, and DBP were significantly correlated with percent body fat. In each sex, the pattern of correlations of the blood pressure variables with FM was identical to that with percent body fat; however, the correlations with FM tended to be slightly greater. The significant positive correlations between percent body fat and blood pressure reflected the positive association between percent body fat and FM. There was no evidence of an association between LBM and any measure of blood pressure in either sex.

FCS, which was measured directly, showed no association with blood pressure in men or women. FCN, however, showed a pattern of relationship to blood pressure variables in both sexes that tended to parallel that of percent body fat or FM, but at a somewhat lower level of correlation. FCN was significantly positively correlated with SBP in men, and with DBP and MAP in women.

**Discussion**

Blood pressure in adults is related to various indices of body build and obesity; most of these indices are functions of stature and weight. Because weight is positively associated with blood pressure, and stature shows little relationship with it, associations between blood pressure and body build indices often reflect the way in which weight is used in the index. For example, positive correlations exist between blood pressure and weight/stature, weight/stature squared, or weight/stature cubed. Another index of body build positively related to blood pressure is relative weight, that is, weight expressed as a percentage of a standard weight for age, stature, sex, and race.

Skinfold thicknesses are more direct measures of body fatness than weight or indices of body build because they are measures of subcutaneous fat at specific sites on the body. There is considerable site-to-site variation in skinfold thicknesses, and their measurements are subject to relatively more measurement error than weight or stature. However, positive correlations between skinfold thicknesses from a variety of sites and blood pressure have been reported. It has been hypothesized that the distribution of subcutaneous fat within an individual, as measured by skinfold thickness, may be related to blood pressure, such that persons with more trunk fat, as opposed to limb fat, have higher blood pressure; however, little evidence supporting this hypothesis has been reported.

There is evidence that increased body weight in adults is predominantly due to an increase in the amount of fat. The association of blood pressure with body weight could be due to the increased total body mass or to some special underlying relationship between blood pressure and body fat (e.g., resulting from differences in microcirculation in fat tissue). Blood pressure–body fat relationships have been demonstrated only indirectly by showing that associations exist between blood pressure and skinfold thickness and between blood pressure and various indices of body build that are related to body fat.

In our present study, there is a clear and unequivocal positive relationship in women between FM and each of the blood pressure variables, SBP, DBP, and MAP. In men, FM is strongly correlated with SBP, and to a lesser degree with MAP and PP. These observations, coupled with the complete lack of relationship between measures of blood pressure and
LBM, provide a strong indication that increased fatness, rather than increased mass per se, may be responsible for the association between blood pressure and weight or indices of body build. Although there is considerable variation in both FM and LBM in this study, the sample is unselected with respect to these variables and contains few individuals at the extremes of the FM or LBM distributions. Therefore, any extrapolation of the relationships identified to individuals with extreme obesity or musclearity should be made with caution.

In the present data, as well as in other studies of adults, FM is not correlated with FCS. In general, therefore, an increase in FM implies an increase in FCM. Estimation of FCM from FM and FCS in the present study assumes that FCM determined from a gluteal sample is representative of FCM in all other sites. This is not entirely true; gluteal fat cells tend to be among the larger adipocytes in the body. Therefore, use of gluteal cells to estimate FCM probably results in an underestimation of FCM. While there is site-to-site variation in mean FCS, measurements of FCS at various sites are correlated. This implies that, while the use of single site FCS to estimate FCM is associated with more error than the use of an average FCS from several sites, a single site provides at least a reasonable estimate of FCM.

Results of our study indicate that increased fatness (either total body fat or percent body fat) is associated with an increased number of fat cells, but not with an increase in fat cell size. Furthermore, the more fat cells an individual has the more likely he or she is to have increased blood pressure; however, the size of fat cells is not related to blood pressure level.

Fat cells are reported to reach their adult number by about 18 years of age. Normal changes in body fat during adulthood are believed to be primarily due to changes in FCS rather than FCM. There is some controversy about the FCN hypothesis, which states that FCM is fixed early in life and an individual is predestined toward leanness or obesity depending on whether this number is great or small. The relevant literature has been reviewed by Roche, who concludes there are considerable data that tend to contradict such a hypothesis.

Whether the FCN hypothesis is correct or not, our present findings in adults have important health implications. They suggest that it is important to study longitudinal relationships among these variables and contains few individuals at the extremes of the FM or LBM distributions. Therefore, any extrapolation of the relationships identified to individuals with extreme obesity or musclearity should be made with caution.

In the present data, as well as in other studies of adults, FM is not correlated with FCS. In general, therefore, an increase in FM implies an increase in FCM. Estimation of FCM from FM and FCS in the present study assumes that FCM determined from a gluteal sample is representative of FCM in all other sites. This is not entirely true; gluteal fat cells tend to be among the larger adipocytes in the body. Therefore, use of gluteal cells to estimate FCM probably results in an underestimation of FCM. While there is site-to-site variation in mean FCS, measurements of FCS at various sites are correlated. This implies that, while the use of single site FCS to estimate FCM is associated with more error than the use of an average FCS from several sites, a single site provides at least a reasonable estimate of FCM.

Results of our study indicate that increased fatness (either total body fat or percent body fat) is associated with an increased number of fat cells, but not with an increase in fat cell size. Furthermore, the more fat cells an individual has the more likely he or she is to have increased blood pressure; however, the size of fat cells is not related to blood pressure level.

Fat cells are reported to reach their adult number by about 18 years of age. Normal changes in body fat during adulthood are believed to be primarily due to changes in FCS rather than FCM. There is some controversy about the FCN hypothesis, which states that FCM is fixed early in life and an individual is predestined toward leanness or obesity depending on whether this number is great or small. The relevant literature has been reviewed by Roche, who concludes there are considerable data that tend to contradict such a hypothesis.

Whether the FCN hypothesis is correct or not, our present findings in adults have important health implications. They suggest that it is important to study longitudinal relationships among these variables and contains few individuals at the extremes of the FM or LBM distributions. Therefore, any extrapolation of the relationships identified to individuals with extreme obesity or musclearity should be made with caution.

In the present data, as well as in other studies of adults, FM is not correlated with FCS. In general, therefore, an increase in FM implies an increase in FCM. Estimation of FCM from FM and FCS in the present study assumes that FCM determined from a gluteal sample is representative of FCM in all other sites. This is not entirely true; gluteal fat cells tend to be among the larger adipocytes in the body. Therefore, use of gluteal cells to estimate FCM probably results in an underestimation of FCM. While there is site-to-site variation in mean FCS, measurements of FCS at various sites are correlated. This implies that, while the use of single site FCS to estimate FCM is associated with more error than the use of an average FCS from several sites, a single site provides at least a reasonable estimate of FCM.

Results of our study indicate that increased fatness (either total body fat or percent body fat) is associated with an increased number of fat cells, but not with an increase in fat cell size. Furthermore, the more fat cells an individual has the more likely he or she is to have increased blood pressure; however, the size of fat cells is not related to blood pressure level.

Fat cells are reported to reach their adult number by about 18 years of age. Normal changes in body fat during adulthood are believed to be primarily due to changes in FCS rather than FCM. There is some controversy about the FCN hypothesis, which states that FCM is fixed early in life and an individual is predestined toward leanness or obesity depending on whether this number is great or small. The relevant literature has been reviewed by Roche, who concludes there are considerable data that tend to contradict such a hypothesis.

Whether the FCN hypothesis is correct or not, our present findings in adults have important health implications. They suggest that it is important to study longitudinal relationships among these variables and contains few individuals at the extremes of the FM or LBM distributions. Therefore, any extrapolation of the relationships identified to individuals with extreme obesity or musclearity should be made with caution.
Blood pressure, body composition, and fat tissue cellularity in adults.
R M Siervogel, A F Roche, W C Chumlea, J G Morris, P Webb and J L Knittle

Hypertension. 1982;4:382-386
doi: 10.1161/01.HYP.4.3.382

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://hyper.ahajournals.org/content/4/3/382

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in
Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial
Office. Once the online version of the published article for which permission is being requested is located, click
Request Permissions in the middle column of the Web page under Services. Further information about this
process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/