Total and Abdominal Adiposity Are Associated With Inflammation in Older Adults Using a Factor Analysis Approach

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Background. Obesity-related increases in multiple inflammatory markers may contribute to the persistent subclinical inflammation common with advancing age. However, it is unclear if a specific combination of markers reflects the underlying inflammatory state. We used factor analysis to identify inflammatory factor(s) and examine their associations with adiposity in older adults at risk for disability.

Methods. Adiponectin, CRP, IL-1ra, IL-1sRII, IL-2sRα, IL-6, IL-6sRα, IL-8, IL-15, sTNFRI, sTNFRII, and TNF-α were measured in 179 participants from the Lifestyle Interventions and Independence for Elders Pilot (Mean ± SD age 77 ± 4 years, 76% white, 70% women). Body mass index, waist circumference, and total fat mass were assessed by anthropometry and dual-energy x-ray absorptiometry.

Results. IL-2sRα, sTNFRI, and sTNFRII loaded highest on the first factor (factor 1). CRP, IL-1ra, and IL-6 loaded highest on the second factor (factor 2). Factor 2, but not factor 1, was positively associated with 1-SD increments in waist circumference (β = 0.160 ± 0.057, p = .005), body mass index (β = 0.132 ± 0.053, p = .01), and total fat mass (β = 0.126 ± 0.053, p = .02) after adjusting for age, gender, race/ethnicity, site, smoking, anti-inflammatory medications, comorbidity index, health-related quality of life, and physical function. These associations remained significant after further adjustment for grip strength, but only waist circumference remained associated with inflammation after adjusting for total lean mass. There were no significant interactions between adiposity and muscle mass or strength for either factor.

Conclusions. Greater total and abdominal adiposity are associated with higher levels of an inflammatory factor related to CRP, IL-1ra, and IL-6 in older adults, which may provide a clinically useful measure of inflammation in this population.

Key Words: Aging—Adiposity—Inflammation—Muscle impairment—Factor analysis.

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Activation of the immune system leads to large increases in the release of inflammatory mediators into the circulation (1). Although this critical physiological response typically progresses very rapidly, older persons are more likely to experience increased and prolonged inflammatory activity following acute infection (2). Moreover, persistent elevations in circulating markers of inflammation, even within the clinically normal range, are common with advancing age. Cross-sectional data show that compared with young and middle-aged adults, systemic levels of inflammatory markers, such as interleukin-6 (IL-6), IL-1 receptor antagonist (IL-1ra), tumor necrosis factor-alpha (TNF-α), soluble TNF receptor II (sTNFRII), and C-reactive protein (CRP), are twofold to fourfold higher in older persons (3,4). Elevated levels of these and other inflammatory markers are strongly associated with increased risk for diabetes, coronary heart disease, sarcopenia, physical disability, and mortality (5–9).

Several mechanisms for this age-related subclinical inflammation have been postulated, including increases in adipose tissue mass. Adipose tissue expresses and releases a number of inflammatory cytokines in direct proportion to the amount of adipose mass (10–12). In addition, circulating levels of inflammatory markers are elevated in obesity and
correlate with body mass index (BMI), total body fat, and abdominal fat (11,13). Given that cytokines may accelerate adverse changes in body composition that are typical of the aging process (14), older adults may be vulnerable to obesity-related increases in inflammation. In this regard, aging is associated with a geriatric syndrome characterized by the coexistence of obesity and muscle impairment, either defined by poor muscle strength or low muscle mass (15). Obese individuals with low muscle strength have higher CRP, IL-6, and IL-6 soluble receptor (IL-6sR) levels, a higher prevalence of walking limitations, and a higher risk of all-cause mortality compared with individuals who are nonobese and/or have high muscle strength (16–18). Similarly, obese individuals with low muscle mass are more likely to have balance and gait abnormalities, report falling in the previous year, and develop instrumental activities of daily living disability (19,20). As such, the imbalance between obesity and muscle impairment, which affects approximately 4%–12% of older persons, may have important negative health consequences for the aging population (15).

The inflammatory response is complex and involves numerous cytokines, soluble cytokine receptors, acute phase reactants, and other circulating factors. Despite the growing number of studies reporting an association between adiposity and inflammation, there is a paucity of studies integrating multiple inflammatory markers. As such, it is not clear if one particular inflammatory marker or a specific combination of markers best reflects the underlying inflammatory state. Therefore, the main objective of this study was to use factor analysis to identify summary inflammatory factor(s) from 12 markers and evaluate associations between the identified factors and measures of total and abdominal adiposity in community-dwelling older adults from the Lifestyle Interventions and Independence for Elders Pilot (LIFE-P) study. We also explored whether interactions between adiposity and muscle impairment are associated with the inflammatory factors.

**METHODS**

**Study Participants**

The LIFE-P study was a multicenter single-blind randomized clinical trial comparing a physical activity with a successful aging intervention in 424 elderly men and women at risk for physical disability. The study design and main trial results have been described previously (21,22). Participants were enrolled between April 2004 and February 2005 from four field centers (Cooper Institute, Stanford University, University of Pittsburgh, and Wake Forest University). The main inclusion criteria were ages 70–79 years, Short Physical Performance Battery (SPPB) score less than 10, sedentary lifestyle, and ability to complete a 400-m walk test within 15 minutes without sitting or using an assistive device. Individuals were excluded if they lived in a nursing home; had significant cognitive, hearing, or visual impairments; reported being unable to walk one mile; or had severe cardiac, pulmonary, neurologic, orthopedic, renal, or psychiatric disease. The study was approved by the Institutional Review Boards at each clinic site, and all study participants gave written informed consent.

In an ancillary study, circulating levels of 12 inflammatory markers, including 4 cytokines (IL-6, IL-8, IL-15, and TNF-α), 6 soluble cytokine receptors (IL-1ra, IL-1 soluble receptor type II [IL-1sRⅡ], IL-6sR, IL-2 soluble receptor alpha [IL-2sRα], sTNFRI, and sTNFRII), the acute phase reactant CRP, and the anti-inflammatory protein adiponectin, were measured in the 368 (87%) LIFE-P participants who had a sufficient blood sample collected at baseline (23). These markers were chosen based on their known association with age, disability, or physical activity; their ability to be readily detected in fasting blood samples of older persons; and their function as powerful stimulators of inflammation. Because of the short half-life of many cytokines, soluble cytokine receptors were also included, as they may be more representative of the inflammatory response (24). Importantly, most of these markers have been associated with adiposity and/or related outcomes (25,26). Since body composition was assessed at only three of the field centers, the present analysis uses baseline data from 179 LIFE-P participants with measures of both inflammation and body composition. Participants who were not included in this analysis had similar characteristics except for a higher BMI (31.1 vs 29.0 kg/m², p = .0003) and waist circumference (103.4 vs 100.3 cm, p = .04) and a greater number of prevalent conditions (1.85 vs 1.52, p = .003).

**Anthropometrics and Body Composition**

Height was measured to the nearest 0.1 cm, and weight was measured to the nearest 0.1 kg. Waist circumference was measured to the nearest 0.1 cm at the midpoint between the highest part of the iliac crest and the lowest part of the costal margin in the midaxillary line. Total fat mass and total lean mass were measured using dual-energy x-ray absorptiometry.

**Inflammatory Markers**

Blood samples were collected in the morning after a 12-hour overnight fast. In the event of an acute respiratory, urinary tract, or other infection, blood sampling was postponed until after resolution of all symptoms. Processed blood samples were divided into aliquots and shipped to the Biological Specimen Repository at Wake Forest School of Medicine, where samples were stored at −80°C until analysis. Adiponectin, IL1-ra, IL-1sRⅡ, IL-2sRα, IL-6, IL-6sR, IL-8, IL-15, sTNFRI, sTNFRII, and TNF-α were measured using Quantikine or QuantiGlo chemiluminescent enzyme-linked immunosorbent kits (R&D Systems, Minneapolis, MN). CRP was measured using an automated immunoanalyzer (IMMULITE; Diagnostics Products Corporation, Minneapolis, MN).
Los Angeles, CA). All samples were measured in duplicate, and the average of the two values was used for data analyses. Duplicate samples that did not provide a coefficient of variation less than 30% for TNF-α, 25% for sTNFRI, sTNFRII, IL-2sRα, IL-6sR, IL-1sRII, IL-8, and IL-15, and 20% for adiponectin, IL-6, and CRP were reanalyzed. The intra-assay coefficients of variation for the 12 inflammatory markers ranged from 2.4% to 7.1%, while the inter-assay coefficients of variation ranged from 6.7% to 15.6% (23). We previously reported a high degree of interrelatedness between the inflammatory markers (23).

**Clinical Measurements**

Baseline assessments included a personal interview, physical exam, electrocardiogram, and physician evaluation. Age, race/ethnicity, and smoking status were assessed by questionnaire. Prevalence of clinical conditions was determined using self-reported physician-diagnosed disease information. To assess medication use, participants were requested to bring all prescription and nonprescription medications taken in the preceding 2 weeks. A comorbidity index was calculated as the sum of yes (1) or no (0) self-report responses for 10 prevalent conditions: hypertension, heart attack, heart failure, stroke, cancer, diabetes, broken hip, arthritis, liver disease, and lung disease, as previously described (27). The Quality of Well-Being Scale–Self-Administered (QWB-SA) was used to assess health-related quality of life. This scale combines preference-weighted values for symptoms and functioning and ranges from 0 (death) to 1.0 (asymptomatic, optimum functioning; [27]). The SPPB comprising a timed usual pace 4-m walk repeated chair stands, and a balance test was used to assess lower extremity physical function (28). Each of these measures was scored from 0 to 4, with 4 indicating the highest level of performance and 0 the inability to complete the test. A summary score ranging from 0 (worst performers) to 12 (best performers) was calculated by adding the three individual scores together. Grip strength was measured using an adjustable, hydraulic dynamometer (Jamar Hand Dynamometer; Fred Sammons, Inc.). The best performance of two trials was selected for each hand, and the average of the left and right hand was used for analysis.

**Statistical Analysis**

Analyses were performed using SAS software version 9.2 (SAS Institute, Cary, NC). All inflammatory markers (except for IL-1sRII and IL-6sR) were log-transformed to achieve a normal distribution. Factor analysis was used to reduce the 12 inflammatory markers into smaller sets of factors that account for most of the variance of the inflammatory variables. The number of factors retained was based on the following rules: eigenvalues greater than or equal to 1 or factors above the break in the scree plot. A varimax rotation was used to obtain a set of independent and best interpretable factors. The factors were interpreted based on the loadings that relate the markers to the factors. Loadings greater than or equal to 0.40 were used to identify the variables comprising a factor. Factor scores were calculated for each participant. Given the relatively small sample size, data are presented in the overall group and/or stratified by gender. Spearman correlation coefficients were used to determine the association of BMI, waist circumference, and total fat mass with the individual inflammatory markers and each identified factor. For the primary analyses, linear regression models of the association between adiposity (independent variable) and inflammatory factor (dependent variable) were initially adjusted for age, gender (in nonstratified analyses), race/ethnicity, and clinical site (Model 1) and then further adjusted for smoking, anti-inflammatory medications, comorbidity index, QWB-SA score, and SPPB score (Model 2). To explore whether muscle impairment modified the association with inflammation, Model 2 was additionally adjusted for muscle impairment alone or muscle impairment plus the adiposity by muscle impairment interaction. Each adiposity variable of interest (BMI, waist circumference, and total fat mass) was included in the linear regression model with each muscle impairment variable of interest (total lean mass and grip strength). Regression coefficients were expressed per 1-SD increment as follows: BMI = 4.8 kg/m², waist circumference = 14.0 cm, total fat mass = 8.6 kg, total lean mass = 10.7 kg, and grip strength = 8.9 kg. A two-sided p value ≤ .05 was considered statistically significant for all analyses.

**Results**

**Participant Characteristics**

The overall study population was 76% white, 70% women, and had a mean ± SD age of 77 ± 4 years. Table 1 shows the demographic and clinical characteristics of the study population by gender. The prevalence of abdominal obesity, defined as a waist circumference greater than 102 cm in men and greater than 88 cm in women, was ~76% in both genders, while 41% of men and 35% of women had a BMI greater than or equal to 30 kg/m². Total body fat averaged 30% in men and 40% in women.

**Factor Analysis**

Factor analysis conducted in men and women combined revealed two eigenvalues greater than one, indicating the presence of two independent inflammatory factors, which was confirmed by the scree plot, where an elbow occurred at the second factor (data not shown). Three variables (IL-2sRα, sTNFRI, and sTNFRII) loaded highest on the first factor (factor 1) and three variables (CRP, IL-1ra, and IL-6) loaded highest on the second factor (factor 2), as shown in
**Table 1. Participant Characteristics by Gender**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men (n = 54)</th>
<th>Women (n = 125)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>76.3 ± 3.9</td>
<td>76.7 ± 4.1</td>
</tr>
<tr>
<td>White</td>
<td>41 (75.9)</td>
<td>95 (76.0)</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>38 (70.4)</td>
<td>104 (83.2)</td>
</tr>
<tr>
<td>Former</td>
<td>13 (24.1)</td>
<td>15 (12.0)</td>
</tr>
<tr>
<td>Current</td>
<td>3 (5.6)</td>
<td>6 (4.8)</td>
</tr>
<tr>
<td>Anti-inflammatory medications</td>
<td>38 (70.4)</td>
<td>80 (64.0)</td>
</tr>
<tr>
<td>Comorbidity index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(number of conditions)</td>
<td>1.84 ± 1.14</td>
<td>1.38 ± 0.99</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.8 ± 4.3</td>
<td>28.7 ± 5.0</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>108.6 ± 12.1</td>
<td>96.7 ± 13.3</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>27.2 ± 8.1</td>
<td>30.0 ± 8.7</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>59.1 ± 8.2</td>
<td>42.0 ± 6.9</td>
</tr>
<tr>
<td>Total body fat (%)</td>
<td>30.1 ± 5.6</td>
<td>40.0 ± 5.3</td>
</tr>
<tr>
<td>SPPB score (0–12)</td>
<td>7.80 ± 1.35</td>
<td>7.51 ± 1.38</td>
</tr>
<tr>
<td>Grip strength (kg)</td>
<td>33.6 ± 8.4</td>
<td>21.1 ± 6.1</td>
</tr>
<tr>
<td>QWB-SA score (0–1)</td>
<td>0.653 ± 0.119</td>
<td>0.638 ± 0.100</td>
</tr>
</tbody>
</table>

*Note: Table values are n (%) or mean ± SD. BMI = body mass index; QWB-SA = Quality of Well-Being Scale–Self-Administered; SPPB = Short Physical Performance Battery.*

Table 2. The two factors together explained 23% of the total variance in the data (factor 1: 15%; factor 2: 8%). To test the robustness of our approach, we performed secondary analyses reducing the set of inflammatory markers to only those that loaded onto one of the two factors (data not shown). In these analyses, the factor loadings were not only similar, but the same inflammatory markers clustered together to comprise two identified factors. In this case, the two factors together explained 37% of the variance.

**Correlations Between Adiposity and Inflammatory Variables**

As shown in Supplementary Table 1, there were no significant associations between factor 1 and BMI, waist circumference, or total fat mass in neither men nor women. In women only, factor 2 was positively associated with BMI (r = .27, p = .002), waist circumference (r = .28, p = .001), and total fat mass (r = .21, p = .02). Correlations between the individual inflammatory markers and adiposity variables were consistent with the factor results.

**Association Between Total and Abdominal Adiposity and Inflammatory Factors**

Linear regression analyses were used to determine the association between total and abdominal adiposity and the identified factors (Table 3). Overall, after adjusting for age, gender, race/ethnicity, and clinical site (Model 1), 1 SD increments in waist circumference (p = .0007), BMI (p = .001), and total fat mass (p = .003) were positively associated with factor 2. After further adjustment for smoking, anti-inflammatory medications, comorbidity index, QWB-SA score, and SPPB score (Model 2), the associations with factor 2 were slightly attenuated but remained significant. Factor 1 was not associated with any of the adiposity variables. Although we had limited power to detect associations in men, we did observe positive associations between adiposity and factor 2 in women.

**Relationship Among Adiposity, Muscle Impairment, and Inflammatory Factors**

In an exploratory analysis, we examined whether interactions between adiposity and muscle impairment are associated with the inflammatory factors. We first examined the effects of adding muscle impairment measures to the fully adjusted model (Model 2). Only waist circumference remained associated with factor 2 after adjusting for total lean mass (Supplementary Table 2). However, all three adiposity measures were associated with factor 2 after the addition of grip strength, although the associations were slightly attenuated (Supplementary Table 3). Next, we examined the effects of adding the interaction term to the model and found that the results were similar, as there were no significant interactions between adiposity and muscle impairment for either inflammatory factor (p > .05 for all, data not shown).

**Discussion**

Aging and obesity are associated with elevated levels of circulating inflammatory markers (3,4,11). Although a number of studies have investigated the association between inflammation and body composition, ours is the first to examine the grouping of inflammatory markers and how these groups relate to various measures of adiposity. We used a factor analysis approach to identify two factors that explained 23% of the total variance among 12 measured markers of inflammation in community-dwelling older adults at risk for disability. We found that BMI, waist
circumference, and total fat mass were positively associated with factor 2 (which included CRP, IL-1ra, and IL-6) but not factor 1 (which included IL-2sR, sTNFRI, and sTNFRII). These findings demonstrate that inflammation is independently associated with total and abdominal adiposity in this population of elderly men and women.

Factor analysis offers several advantages over other approaches to create summary variables as it accounts for the underlying correlational structure between individual markers, does not require a priori biological assumptions, and minimizes multiple testing issues. In our analysis, only 6 of the 12 markers loaded onto one of the two identified factors. However, secondary analyses revealed that the overall results were the same whether the analysis included all 12 inflammatory markers or just a reduced subset of markers. Univariate associations with the adiposity measures were not always stronger for the identified factors versus the individual markers. These findings suggest that calculating summary variables may not strengthen the association between inflammation and adiposity when compared with using a single marker—at least based on the set of 12 inflammatory markers we studied. Moreover, risk factors other than adiposity may be important contributors to a low-grade inflammatory state in older adults. Indeed, other variables associated with higher inflammation (ie, factor 1 and/or factor 2) in this population were older age, white race, and a higher number of prevalent conditions (data not shown).

We previously reported the use of principal component analysis to summarize eight inflammatory markers in the Health, Aging, and Body Composition (Health ABC) study (29). In this earlier analysis, two components were identified that explained 56% of the total variance: a TNF-α-related component that included TNF-α, sTNFRI, sTNFRII, IL-6sR, and IL-2sR and a CRP-related component that included CRP, IL-6, and PAI-1. We found similar results in the present study, as IL-2sR, sTNFRI, and sTNFRII clustered together (factor 1), while CRP and IL-6 clustered together (factor 2). In addition, although the factor loadings were lower (<0.40), TNF-α and IL-6sR did track better with factor 1 than factor 2. Consistent with our findings of a positive association between total fat mass and factor 2 (but not factor 1), the Health ABC study found that total fat mass was positively associated with the CRP-related component but not the TNF-α-related component (29). Taken together, these data indicate that at least two factors can be used to summarize inflammatory markers in older adults, which likely reflect biologically relevant pathways that link the specific markers together, for example, IL-6 is the principal regulator of CRP, and IL-2 stimulates the release of soluble TNF receptors (30,31).

Cesari and colleagues (9) reported that higher CRP and IL-6 levels were associated with higher BMI and total fat mass but lower appendicular lean mass in older adults from the TRAIN study. Interestingly, they found that inclusion of both obesity and sarcopenia in the same linear regression model resulted in significant associations with obesity, but not sarcopenia. In the InCHIANTI study, Schrager and colleagues (17) also observed higher CRP, IL-1ra, IL-6, and IL-6sR in individuals with global and/or central obesity and higher CRP and IL-6sR in individuals with low grip strength. In addition, they found that central obesity had a greater influence on inflammation than global obesity. Our data confirm the findings presented in these prior studies. We found that BMI, waist circumference, and total fat mass were independently associated with factor 2, while total lean mass and grip strength were not. Waist circumference was also a stronger correlate of inflammation than both BMI and total fat mass, and it was the only adiposity variable that remained associated with inflammation after adjusting for muscle mass or strength. These findings suggest that obesity is a stronger correlate of inflammation than muscle impairment and support the role of body fat distribution, particularly increased abdominal fat, in driving the association between obesity and a number of diseases and health conditions.
Recent evidence suggests that obese persons with low muscle mass or muscle weakness exhibit an important geriatric syndrome that is associated with negative health outcomes (15). With aging, increased adiposity promotes increased cytokine production, which enhances loss of muscle mass and strength, and thereby creates a vicious cycle leading to functional decline and physical disability. In the InCHIANTI study, the authors found a significant interaction between central obesity and grip strength for IL-6 and IL-6sR, such that individuals with waist circumference in the highest sex-specific tertile and grip strength in the lowest sex-specific tertile had higher blood pressure compared with the lowest data groups (17). In our study, there were no significant interactions between adiposity and muscle impairment, and in general, the main effect of adiposity was stronger. Future studies should clarify the clinical consequences of an imbalance between adiposity and muscle impairment and the associated effects on inflammatory pathways in older adults. In addition, a better understanding of the interrelationship of aging, inflammation, and physical inactivity could provide more insight into targeted lifestyle therapies that may slow or prevent the development of obesity and muscle impairment.

Although this study evaluated a large number of inflammatory markers, our results should be interpreted with caution given the small sample size, which may have limited our ability to detect significant associations, particularly in men. An additional limitation includes the study population, as LIFE-P participants were recruited to be older community dwelling, and at high risk for disability, which may not reflect the general population. It may also be difficult to extrapolate our findings given that our analysis was performed in the context of a randomized clinical trial. Moreover, this study was cross-sectional, and therefore, we cannot determine a causal relationship between adiposity and inflammation. A final consideration is the lack of direct measurements of abdominal fat, which may be relevant because visceral fat has been shown to exhibit heightened inflammatory activity compared with subcutaneous fat (32).

In conclusion, we used a factor analysis approach to identify an inflammatory factor related to CRP, IL-1ra, and IL-6 that had positive associations with total and abdominal adiposity in a nondisabled population of older adults. The two factors we identified are consistent with components identified previously in a similar population using a similar approach. Although it is likely that a defined set of markers can provide better characterization of the underlying inflammatory state, more research is needed to determine exactly which markers (reflecting specific pathways in the overall inflammatory cascade) are the most important. Future studies should not only focus on identifying factors from a more comprehensive set of inflammatory markers but should also explore associations with the identified factors in larger community-based populations. With the increasing prevalence of obesity and the growing proportion of the population surviving to very old ages, it will become imperative to identify clinically useful measures of the persistent low-grade inflammation common in older persons in order to better predict risk for aging-related morbidity and mortality.

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**Supplementary Material**

Supplementary material can be found at: http://biomedgerontology.oxfordjournals.org/

**Acknowledgments**

A list of the LIFE-P study investigators can be found in Appendix 1.

**References**

ADIPOSE AND INFLAMMATION IN OLDER ADULTS


APPENDIX 1. RESEARCH INVESTIGATORS FOR PILOT PHASE OF LIFE

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