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Sex dimorphisms in inflammatory markers and adiposity in African American youth

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Abstract

Objective: There are demonstrated sex differences in the association between adiposity and inflammation in adults. Our aim was to determine sex differences in inflammatory markers and in the association between adiposity and inflammation in a sample of African American adolescents.

Methods: Adiposity variables including BMI, waist circumference, weight, total fat, trunk fat, and inflammatory markers including IL-6, leptin, MCP1, CRP, adiponectin were examined in 166 (53% female) African American adolescents, ages 14-19 years. Total fat and trunk fat were measured using Dual-Energy X-Ray Absorptiometry (DXA).

Results: Results revealed males had higher weight ($p = .01$); females had higher BMI, trunk fat, and total fat (p's $< .01$). With inflammation, males had higher MCP1 ($p = .024$); females had higher leptin ($p < .001$), adiponectin ($p = .006$), and IL-6 ($p = .026$). Partial correlations in males indicated associations of adiposity variables with leptin, adiponectin (all p's $< .01$), and CRP ($p < .05$); in females, leptin, CRP, and IL-6 were associated with adiposity variables (all p's $< .05$). Multiple regression analyses revealed female adiposity variables predicted CRP, (R^2=.254), IL-6 (R^2=.167), and MCP1 (R^2= .220). Adiposity variables in males predicted lower adiponectin (R^2=.245). For both, leptin was predicted by adiposity (males R^2=.420 and females R^2=.410).

Conclusions: Data indicate clear sex dimorphisms in the associations between inflammatory markers and adiposity in African American adolescents, suggesting that preventive measures and treatments for adolescent obesity may need to be sex-specific.

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INTRODUCTION

Obesity is an inflammatory condition, characterized by abnormal cytokine production [1-3]. Adipose tissue releases cytokines, which are related to increased cardiovascular disease risk and insulin resistance [4]. Adipose tissue acts as an endocrine organ, secreting hormones and cytokines with either proinflammatory or anti-inflammatory activity. These in turn regulate endothelial function or glucose metabolism, potentially leading to diabetes and/or heart disease [2, 4]. It is suggested that endothelial function may be the mechanism responsible for the link between cardiovascular disease and type 2 diabetes [5, 6]. In fact, many cite diabetes as a further risk factor for cardiovascular disease [7].

Increased adiposity is associated with inflammatory response in adults. Cytokines such as interleukin-6 (IL-6) and adiponectin correlate with adiposity measures (body mass index (BMI) and fat mass) [4, 8]. This relationship has also been established in youth, with markers like C-reactive protein (CRP) and leptin correlating with adiposity [4, 9]. These markers not only demonstrate alteration in obese youth, but potentially lead to the relationship between childhood obesity and diabetes, as well as adult cardiovascular disease (regardless of adult weight status) [9]. Further, many of these markers, like CRP and MCP-1, have been linked to both diabetes and cardiovascular disease [10-12].

There are demonstrated sex dimorphisms in the relationships between adiposity and inflammation in adults [4, 13]. Fat distribution and the metabolism of adipose tissue involves sex hormones, and differs by sex [14, 15]. For example, plasma adiponection levels are found to be higher in women [2, 14], as are CRP levels [14, 16, 17]. However, levels of IL-6 are found to be higher in men in some studies [18], higher in women in others [17]. Fewer investigations have examined sex differences in youth, though existing results are similar to adult findings. Further, these studies have found correlations between inflammation markers and sex hormones such as testosterone and dehydroepiandrosterone sulfate (DHEA-S) [19, 20]. It is posited that certain markers, such as adiponectin and leptin, track with physical and pubertal development. For example, boys experience significant declines in levels of adiponectin beginning in puberty, and continuing through adolescence and into adulthood [19-22]. However, few studies have examined sex differences in the relationship of inflammatory markers with body composition in adolescents, and none in a solely African American sample. In fact, Thorand et al. (2007) call for further investigation into the interaction between sex and inflammation, in order to determine which markers are best used prognostically [18].

To our knowledge, this is the first study to explore sex differences in inflammatory markers, as well as in the association between adiposity and inflammatory markers, in African American adolescents. Previous studies report associations between markers of inflammation and body composition. However, few have included the specific cytokines studied here (particularly IL-6, monocyte chemotactic protein 1 - MCP1), and even fewer utilized dual energy X-ray absorptiometry (DXA) to measure body composition [8]. Furthermore, of the studies using DXA and multiple inflammatory markers, sex dimorphisms are not investigated in African Americans. The purpose of the current study was to examine the sex-specific association of adiposity variables with five inflammation markers: IL-6, leptin, CRP, adiponectin, and MCP1. The adiposity variables included BMI, waist circumference, and DXA measures of total fat mass and trunk fat mass. We hypothesized there would be sex-specific differences in associations between markers of adiposity and inflammation.

METHODS

Subject recruitment and protocol

The protocol was approved by the Human Assurance Committee of the Medical College of Georgia. A sample of 166 healthy normotensive African American adolescents including 78 males and 88 females were recruited from local public high schools in the Augusta-Richmond County area via school announcements, flyers, handouts, or word of mouth. Written informed parental consent and subject assent were obtained before testing. Data were collected between June 2006 and July 2008. Race (African American) was identified by self-report of each subject, and by parent if the subject was less than 18 years of age. Height, weight, and blood pressure (BP) were obtained. Height and weight were measured with clothing on, but without shoes. BP was measured with the Dinamap monitor (Dinamap Compact Monitor, Tampa, FL). Normotension was based on BP screening, namely <95th percentile for age, sex, and height, or <140/90 mm/Hg for subjects aged 18 years or older [23]. Blood samples were non-fasting. Lipids, including total cholesterol, triglycerides, high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL), and the total cholesterol/HDL ratio, were measured with the Cholestech LDX analyzer (Cholestech, Hayward, CA). Other exclusion criteria included any chronic illness, any medication use (including oral contraceptives), or a positive pregnancy test. In addition, tobacco and alcohol use were exclusionary criteria. Females were not tested while on their menses, but were tested on the week following completion of their menstrual flow to ensure that all females were tested in the same phase of their menstrual cycle.

Adiposity Measures

Body composition was obtained via waist circumference measurement and BMI (kg/m^2, from height and weight measurement). Waist circumference was measured with thin clothing on, at the midpoint between the lowest rib and the iliac crest. To measure body fat, DXA (QDR 4500W, Hologic Inc., Bedford, MA) segmented the body into fat, bone, and fat free soft tissue. Percent body fat adjusted for body mass. Pubertal stage was not assessed in this population.

Inflammatory Markers/ Blood cytokines Analysis

Plasma samples were stored in −80°C. IL-6, high-sensitivity (hs)-CRP, MCP1, leptin, and adiponectin were measured in duplicate by ELISA kits (R&D Systems, Inc. Minneapolis, MN). All kits were conducted according to the manufacturer's instructions. The detection limits were 0.016 pg/mLfor IL-6, 0.005 ng/mL for hs-CRP, 5.0 pg/mL for MCP1, 7.8 pg/mL for leptin, and 0.079 ng/mL for adiponectin. The intra- and interassay coefficients of variation were less than 7.8% and 9.6% for IL-6, 8.3% and 7.0% for hs-CRP, 7.8% and 6.7% for MCP1, 3.3% and 5.4% for leptin, and 4.7% and 6.9% for adiponectin. Undetectable values were assigned a value of zero for the purposes of analysis.

Statistical analyses

The general characteristics of the subjects are presented as mean ± SD. T-tests were conducted with these general characteristics to examine the potential differences between sexes. Any variables not normally distributed were log-transformed — these included trunk fat, total fat, leptin, MCP1, adiponectin, IL6, and CRP. Simple bivariate correlations were first conducted, then partial correlations to adjust for potential confounders, such as age. A value of $p < 0.05$ was deemed statistically significant. Then linear multiple regression analyses were conducted to further examine the sex-specific relationships between adiposity and inflammatory markers. The statistical analyses were performed with SPSS 16.0 (SPSS, Inc., Chicago, IL).

RESULTS

Clinical Characteristics

A comparison of adiposity variables and inflammation markers by sex is shown in Table 1. Adiposity variables included BMI, BMI percentile, BMI z-score, weight, waist circumference, total fat mass, and trunk fat mass (as measured by DXA). Inflammation variables included CRP, leptin, IL-6, MCP1, and adiponectin. It is noteworthy that of the adiposity and inflammation variables, the only ones that were not significantly different between the sexes were waist circumference, CRP, BMI percentile and BMI z-score (though the latter two trended towards significance) with all being higher in females. In addition, DBP, Triglycerides, total cholesterol, LDL, and TC/HDL ratio were not significantly different between the sexes. However, males had significantly higher weight, SBP, VLDL, and significantly higher levels of MCP1. Females had significantly higher BMI, trunk fat mass, total fat mass, HDL, and significantly higher levels of leptin, adiponectin, and IL-6.

Associations between Adiposity Markers and Inflammation Markers

When the sample was examined by sex, certain differences were noted. Table 2 shows the partial correlations for each inflammation marker and adiposity variable, separated by sex, and controlling for age. In males, leptin was associated with the adiposity variables including: BMI, waist circumference, weight, total fat mass, and trunk fat mass. Neither IL-6 nor MCP1 in males were significantly associated with the adiposity variables, while adiponectin was negatively associated with BMI, waist circumference, weight, total fat mass, and trunk fat mass. CRP in males was associated with total fat mass and trunk fat mass. In females, too, leptin was associated with BMI, waist circumference, weight, total fat mass, and trunk fat mass. However, CRP in females was associated with all of the adiposity variables, including BMI, waist circumference, weight, total fat mass, and trunk fat mass, whereas IL-6 was associated with total fat mass, and showed a trend with weight ($r = .182$, $p = .088$). Adiponectin in females was associated only with waist circumference, and showed a trend with total fat ($r = -.178$, $p = .097$). MCP1 in females was not associated with any of the adiposity variables, though it demonstrated a trend with trunk fat ($r = -.211$, $p = .080$).

In order to further explore sex-specific differences, a standard multiple regression was performed between each inflammation marker as the dependent variable and the adiposity variables, including BMI, weight, waist circumference, trunk fat mass, and total fat mass as independent variables (Table 3). Sex and age were also included as independent variables in the overall models used with the total sample. All models demonstrated a significant relationship between adiposity and inflammation. Further, sex accounted for a significant proportion of variance in every model except CRP. In addition, ten multiple regressions were conducted (five with each sex separately), on IL-6, adiponectin, MCP1, CRP, and leptin, in order to best determine sex-specific differences regarding which adiposity measures contributed the most variance to an individual inflammatory marker. In males, the models for leptin and adiponectin were significant; however, the models for CRP, leptin, and MCP1 were not significant. In females, the models for IL-6, CRP, leptin, and MCP1 were significant, while the model for adiponectin was not significant. Further, the specific adiposity variables contributing to variance differed by sex, with trunk fat contributing significantly to leptin levels in girls ($\beta = .963$, $p = .002$), waist circumference contributing significantly to MCP1 levels in girls, and weight contributing significantly to adiponectin levels in boys ($\beta = -.650$, $p = .035$). No other individual variables were significant contributors. When looking at specific variables within each of these models age was not associated with any inflammation marker, for either sex.

DISCUSSION

There are several novel findings in this study. First, our sample of all African American adolescents is unique in the literature, given the genetic and physiological differences inherent with this race [13, 24, 25]. Second, while our sample primarily consists of subjects in late adolescence, and so are past puberty, they have also not yet experienced the levels of risk that adults have (sedentary lifestyles, poor dietary choices, smoking, medications, clinical inflammation, etc), resulting in a sample that is less influenced by these lifestyle choices. Third, levels of inflammatory markers (both pro- and anti-) differed significantly among adolescent African American females and their male counterparts. Specifically, levels of leptin, IL-6, and adiponectin were higher in this community sample of females. However, African American males had significantly higher levels of mcp1 – a previously unexamined marker in healthy African American adolescents. MCP1 was examined in one study of German adolescents, and our findings replicate the sex difference found in that study, males having higher levels than females [26]. However, the levels of MCP1 in German adolescents are much higher than the levels found in our sample, indicative of another possible factor unique to African Americans. Levels of CRP were not significantly different between sexes, though adult literature cites females as having higher levels of CRP than males [14]. Sex dimorphisms regarding adiposity variables differed, with males being significantly higher in weight, but females having significantly higher BMI, trunk fat, and total fat, consistent with previous literature [27].

Fourth, in addition to the sex dimorphisms with regards to levels of adiposity and inflammation markers, there were also sex-specific differences in the relationships between adiposity and inflammation. All adiposity variables were associated with levels of CRP in females, but not in males, as only total fat mass and trunk fat mass demonstrated a relationship with CRP. Further, these adiposity variables were inversely associated with adiponectin in males; however, only waist circumference in females was inversely related to adiponectin. Total fat mass in females was associated with levels of IL-6, but this relationship was not significant in males. Finally, all adiposity variables were related to leptin levels in both males and females. This finding extends the literature with regards to inflammation and diabetes risk and cardiovascular risk in adolescents, specifically with regards to sex differences [4, 8]. Given current health disparities with regards to African Americans, females in particular, evaluation of inflammation markers might be especially useful in identifying early cardiometabolic risk [25].

All adiposity variables contributed to CRP and IL-6 levels in females, but not males, and to adiponectin levels in males but not females in healthy African American adolescents. Sex-specific differences were also seen with regards to the impact of single adiposity variables. For example, trunk fat contributed most to leptin levels in females, but adiponectin levels had the highest contribution by weight in males. With regards to MCP1 in females, waist circumference contributed most, while no adiposity variables significantly contributed to MCP1 in males. There were no single significant contributors to CRP or IL-6 levels in either males or females.

The reasons for sex-specific differences in the levels of inflammation markers, and the relationship between adiposity and inflammation remain unclear. Two possible mechanisms are discussed. First, there are differences in body composition between males and females, specifically with regards to abdominal obesity [2, 8]. Female adolescents in this sample had significantly higher trunk fat than did males, consistent with previous research [8, 28, 29]. There are known cardiometabolic associations with abdominal obesity, over and above the risks associated with total body obesity [2]. Since trunk fat includes both visceral and subcutaneous adiposity, it will be necessary for more precise measurements to be

implemented, in order to delineate whether visceral and subcutaneous adiposity differ in this population. Previous research has indicated that in African Americans, subcutaneous adiposity is more related to metabolic risk than visceral adiposity, and that even when visceral and subcutaneous adiposity are lower in this group, inflammatory marker levels are still higher (compared to Caucasians). This indicates a further health disparity that needs to be examined more thoroughly [13].

Second, it seems likely that endogenous sex hormones play a considerable role, given that weight gain typically occurs during puberty and continues through adolescence, and expansion of adipose tissue leads to increased production of adipocytokines [3]. While weight gain does occur during these years, it is also the case that there are strong differences in both weight gain and distribution, with females tending to gain more and have more abdominal adiposity (trunk fat) [30]. Furthermore, previous studies have indicated a sex-specific difference in levels of inflammation markers (adiponectin) before and after puberty in healthy children, with the additional implication that sex hormones may play a role beyond simple weight gain [31]. In adults, previous research has found differences in diabetes and cardiovascular risk factors, related to sex and hormone levels. One study found that high levels of testosterone predicted increased risk of Type 2 diabetes in women, but not men, and another reported that high concentrations of sex hormone-binding-globulin is associated with decreased risk of diabetes, particularly in postmenopausal women [2, 32].

Strengths, Limitations, and Future Directions

Our study demonstrated several strengths, including the sample itself — a normotensive African American adolescent community sample. We had multiple measures of body composition, and a wider variety of inflammatory markers than have previously been measured in healthy adolescents. Specifically, we used DXA (in addition to waist circumference and BMI) to obtain more precise body composition measurements, allowing for robust associations between adiposity and inflammation. DXA has been infrequently used in previous studies with healthy adolescent populations, due to cost involved. Finally, few studies have reported examining the number and type of inflammation markers we measured, again allowing for a more specific examination of inflammation in healthy adolescents.

While we did find clear associations between adiposity variables and level of inflammation markers, results need to be replicated in an independent sample. Our findings are limited to a cross-sectional sample of African American adolescents residing the southeastern United States. We did not examine the influence of genetics, pubertal status, physical activity, diet, tobacco or alcohol use, oral contraceptive use, or socioeconomic status on the relationship between adiposity and inflammation — all of which likely play a pivotal role. In addition, we did not specifically measure visceral and subcutaneous adipose tissue. Future studies will need to focus on the interplay between diet and physical activity, and how those potentially mediate the relationship between adiposity and inflammation. Other investigations will also need to take hormonal factors into account, by measuring hormone levels and examining the mediating relationship of hormones with adiposity and inflammation.

In conclusion, our results show that there are sex-specific differences in levels of inflammation, as early as adolescence, and that adiposity is strongly, but differentially, related to these inflammatory markers. In African American adolescents, females specifically have higher levels of leptin, adiponectin, and IL-6, while males have higher levels of MCP1. Further, adiposity in females was strongly related to CRP, and to a lesser extent, IL-6 and adiponectin. Adiposity in males was strongly related to adiponectin. Leptin was related to adiposity in both males and females. Reduction in adipose tissue continues to

be the most promising solution to reducing subclinical inflammation, and seems particularly important in adolescent females.

For future investigations, it will be important to examine a variety of inflammatory markers, in order to represent the diverse status of inflammation. In addition, our study suggests that when examining inflammation markers, one should analyze data from males and females separately, rather than adjusting for sex, in order to obtain a more complete picture of the results. Finally, given the sex-specific differences in the association between adiposity and inflammation, preventive measures and treatments for obesity may need to differ by sex, in order to identify early cardiometabolic risk and engage individuals in lifestyle changes.

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Table 1

Descriptive Characteristics of African American Study Participants, n = 166

	Male	Female	*p*
Age (years)	17.04 (1.2)	16.96 (1.3)	.603
Wt (kg)	75.33 (16.4)	70.01 (18.1)	.012
BMI (kg/m^2)	24.35 (4.7)	26.16 (6.6)	.009
BMI percentile	67.88 (24.21)	74.93 (24.92)	.060
BMI z-score	.643 (.89)	.907 (.923)	.057
WC (cm)	80.32 (11.43)	78.46 (11.34)	.282
SBP (mm/Hg)	116.71 (10.2)	107.75 (9.6)	<.001
DBP (mm/Hg)	60.64 (6.4)	60.35 (5.8)	.749
Triglycerides (mg/dL)	89.15 (42.6)	86.21 (42.6)	.671
Total Cholesterol (mg/dL)	136.74 (41.2)	127.01 (47.0)	.178
HDL (mg/dL)	46.34 (15.0)	52.63 (19.3)	.029
LDL (mg/dL)	83.67 (30.1)	76.25 (36.3)	.196
VLDL (mg/dL)	16.32 (9.2)	12.83 (7.6)	.022
TC/HDL	14.97 (34.3)	19.80 (33.8)	.390
Trunkfat(g) Log Trunkfat	5281.7 (4037.7) 3.63 (.27)	10504.33 (7255.8) 3.93 (.29)	<.001 <.001
Totalfat (g) Log Totalfat	12812.55 (8630.3) 4.03 (.24)	22831.88 (11773.2) 4.30 (.22)	<.001 <.001
Leptin (ng/mL) Log leptin	4.80 (6.6) .36 (.55)	22.24 (19.3) 1.23 (.32)	<.001 <.001
MCP1 (pg/mL) Log MCP1	131.76 (28.3) 2.11 (.09)	116.87 (44.6) 2.04 (.14)	.024 .001
Adipo (ug/mL) Log adiponectin	5.41 (3.2) .66 (.26)	6.99 (4.3) .76 (.30)	.006 .021
IL6 (pg/mL) Log IL6	1.29 (1.3) −.03 (.33)	1.80 (1.7) .11 (.35)	.026 .006
CRP (ug/mL) Log CRP	1.01 (1.8) −.432 (.58)	1.27 (1.8) −.260 (.60)	.363 .100

Note: CRP was undetectable in 31 subjects, leaving a total of 159 subjects for analyses.

Table 2

Partial correlations of body composition variables with markers of inflammation in African American adolescent males and females, controlling for age.

	CRP	Leptin	IL6	MCP1	Adiponectin
Males					
BMI	.179	.589 ***	.130	.112	−.324 **
WC	.173	.587 ***	.093	.071	−.321 **
Wtkg	.220	.502 ***	.168	.135	−.382 ***
TotalFat	.252 *	.588 ***	.211	.148	−.262 **
TrunkFat	.251 *	.440 **	.181	.139	−.356 **
Females					
BMI	.442 ***	.496 ***	.164	−.041	−.176
WC	.447 ***	.335 **	.155	.025	−.214 *
Wtkg	.441 ***	.428 ***	.182	.029	−.141
TotalFat	.486 ***	.475 ***	.227 *	−.159	−.178
TrunkFat	.459 ***	.498 ***	.157	−.211	−.144

* $p < .05$

** $p < .01$

*** $p < .001$

Table 3

Regression results of body composition variables with markers of inflammation in African American adolescent males and females. Sex, age, BMI, WC, Wtkg, TotalFat, and TrunkFat were included in each model; all variables except sex were included in the sex-specific models.

	Total Sample		Contribution of Sex in each model	
	R^2	p	R^2	p
CRP	.173	(F (7,141) = 4.21, p < .001) ***	.016	(F (1,147) = 2.44, p = .121)
Leptin	.491	(F (7,115) = 15.86, p < .001) ***	.262	(F (1,121) = 43.05, p < .001) ***
IL6	.091	(F (7,160) = 2.30, p = .03) *	.026	(F (1,166) = 4.51, p = .035) *
MCP1	.157	(F (7,119) = 3.18, p = .004) **	.037	(F (1,125) = 4.82, p = .030) *
Adiponectin	.142	(F (7,150) = 3.71 p < .001) ***	.036	(F (1,156) = 5.80, p = .017) *
	Males		Females	
	R^2	p	R^2	p
CRP	.086	(F (6, 64) = 1.00, p = .43)	.254	(F (6,71) = 4.04, p = .002) **
Leptin	.420	(F (6,49) = 5.90, p < .001) **	.410	(F (6,66) = 30.09, p < .001) ***
IL6	.067	(F (6,71) = .846, p = .539)	.167	(F (6,83) = 2.81, p = .015) *
MCP1	.048	(F (6,53) = .446, p = .845)	.220	(F (6,64) = 3.00, p = .012) *
Adiponectin	.245	(F (6,74) = 4.00, p < .002) **	.087	(F (6,82) = 1.30, p = .266)

* p < .05

** p < .01

*** p < .001