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C-Reactive Protein Levels in African Americans: A Diet and Lifestyle Randomized Community Trial

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Abstract

Background—Chronic Inflammation is linked to poor lifestyle behaviors and a variety of chronic diseases that are prevalent among African Americans, especially in the southeastern U.S.

Purpose—The goal of the study was to test the effect of a community-based diet, physical activity, and stress reduction intervention conducted in 2009–2012 on reducing serum C-reactive protein (CRP) in overweight and obese African-American adults.

Methods—An RCT intervention was designed jointly by members of African-American churches and academic researchers. In late 2012, regression (i.e., mixed) models were fit that included both intention-to-treat and post hoc analyses conducted to identify important predictors of intervention success. Outcomes were assessed at 3 months and 1 year.

Results—At baseline, the 159 individuals who were recruited in 13 churches and had evaluable outcome data were, on average, obese (BMI=33.1 [± 7.1]) and had a mean CRP level of 3.7 (± 3.9) mg/L. Reductions were observed in waist-to-hip ratio at 3 months (2%, $p=0.03$) and 1 year (5%, $p<0.01$). In female participants attending 60% of intervention classes, there was a significant decrease in CRP at 3 months of 0.8 mg/L ($p=0.05$), but no change after 1 year. No differences were noted in BMI or interleukin-6.

Conclusions—In overweight/obese, but otherwise “healthy,” African-American church members with very high baseline CRP levels, this intervention produced significant reductions in

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CRP at 3 and 12 months, and in waist-to-hip ratio, which is an important anthropometric predictor of overall risk of inflammation and downstream health effects.

Trial registration—This study is registered at www.clinicaltrials.gov NCT01760902.

Introduction

Chronic inflammation figures prominently in a host of chronic health conditions that plague post-industrial societies and that tend to concentrate in minority populations in affluent countries such as African Americans in the U.S.¹ These range from diabetes,² to cardiovascular disease (CVD) and stroke,³ to cancers of many anatomic sites.^{4,5} The most well described modulators of chronic inflammation include diet, physical activity, cardiorespiratory fitness, and related factors such as obesity.^{6–9}

The acute-phase protein C-reactive protein (CRP) is produced in response to stimulation by interleukins (IL), such as IL-6.¹⁰ Although used as a marker of inflammation in such conditions as rheumatoid arthritis for many decades, the more recent development of a CRP assay has permitted the detection of inflammation at the vascular level.¹¹ CRP and inflammatory cytokines, such as IL-6, are found at higher levels among obese individuals and are positively correlated with body weight.¹² Ridker et al.¹³ found that each component of the metabolic syndrome (obesity; hypertriglyceridemia; low levels of high-density lipoprotein [HDL]-cholesterol; hypertension; abnormal glucose metabolism) is significantly associated with higher CRP levels.

African Americans in South Carolina have some of the largest health disparities in the nation.^{4,14} The chronic diseases that present the greatest public health challenge are now known to be related to inflammation,^{15–18} and African Americans tend to have greater inherent sensitivity to inflammation modulators.^{3,19,20} The African-American faith community is an ideal setting in which to base interventions aimed at reducing chronic inflammation. Partnerships with African-American churches, a cornerstone of African-American culture and heritage, involves local leaders who provide trusted information, advice, and access to a high-risk population.²¹ South Carolina ranks third in the U.S. in church attendance and has some of the highest rates of attendance among African Americans as well.²² The African-American faith community is a prime location for health-related interventions and is highly motivated to engage in concerted efforts to address health disparities.²³ This RCT of diet, physical activity, and stress reduction, which draws on the strengths of the African-American faith community in its design, was implemented to test the effect of a diet, physical activity, and stress reduction intervention on CRP levels in African Americans at high risk of chronic inflammation.

Methods

Study Overview

This multiple risk factor intervention, conducted in 2009–2012, was designed to improve diet, increase physical activity, and reduce stress. Its goal was to reduce inflammation in a population at high risk of diabetes, CVD, and many types of cancer. The study was designed using principles of community-based participatory research (CBPR) to engage community partners from the African-American faith community. The study design included a 12-month intervention arm as well as a delayed-intervention arm that served as the control group. The delayed-intervention churches were asked to establish a monthly meeting sequence with the participants focusing on health issues; not to include nutrition and physical activity. The clinical trial team provided support in the navigation of resources and services that were related to health disparities.

During recruitment, each church committed to at least 24 months of participation to accommodate both the 12-month intervention and the year delay if they were randomized into the control group. Churches, which had a 50% probability of being randomized to immediate intervention, were the units of randomization, and individual church members were the unit of measurement for all health-related outcomes. All procedures and consent documents were approved by the IRB of the University of South Carolina.

Churches were recruited in the Midlands of South Carolina (within 40 miles of the University of South Carolina—Columbia campus). A variety of recruitment methods were used, including word of mouth; media (TV and radio); and community liaisons with connections to area churches. These churches were invited to an information session that provided the specifics of the research. Each prospective church was oriented during an educational forum prior to signing a Memorandum of Agreement (MOA). Following the forum, participants were enrolled, screened, and asked to identify a partner to support them in their efforts to participate in the project.

Each church pastor selected three lay health leaders, who constituted the Church Education Team (CET) that facilitated the study. Their duties included promotion of the study within the church, recruitment of individuals, reminders of clinical visits, and leading the intervention. Eligible individuals within each church were aged ≥30 years and had no reported cancer diagnosis or unstable comorbidities that might limit participation in the intervention. Lay leaders and participants received small incentives throughout the study. The number of subjects included in the analyses from each church ranged from five to 22, with a mean of ~12 subjects per church.

Churches were randomized by blocks according to social class and educational level as reported by the CETs' identification of the entire church population. These two factors were combined so that poor, working, or lower-middle (vs middle and upper-middle) class participants (i.e., with an educational level of high school/GED or less, vs at least some college) were classified as low SES. Others were categorized as high SES. The randomization assignment was generated using the RANUNI function in SAS. This assignment was conveyed to the churches. All enrolled participants were assigned to intervention or control according to their church's assignment.

Intervention Design and Implementation

Design of the Healthy Eating and Active Living in the Spirit (HEALS) intervention was based on insights and experience from other successful studies conducted by the current authors over the past 20 years.^{6,24} This included an intensive 12-week healthy diet and physical activity program combined with stress reduction. Materials were modified to meet individuals' and churches' needs and goals within a structure that included: (1) cooking classes and recipes; (2) tips for increasing physical activity level as part of one's daily routine; (3) suggestions for stress reduction; and (4) assistance tracking basic measurements such as weight and blood pressure.

The second phase of the intervention included monthly boosters for an additional 9 months to reinforce and expand on topics introduced in the first, 12-week, phase. Self-awareness and goal setting were central features of all modules. Social-ecologic models^{25,26} provided the framework for the intervention approach. The PEN-3 (individual influence [perceptions, enablers, and nurturers]; cultural influence (positive, exotic, and negative); and health education [person, extended family, neighborhood]) cultural identity model was used to guide the formative stages of culturally tailoring study protocols.²⁷ Strategies from Social Cognitive Theory²⁸ and the Transtheoretical Model²⁹ were used in conjunction with PEN-3 to guide intervention messages delivered to individual members of the congregation and

church leaders. Details of the intervention, including the class syllabus, are available on request.

Data Collection

Data were obtained via questionnaire, anthropometric measurement, objective physical activity and energy expenditure monitoring, and laboratory analyses of blood samples collected during clinic visits. Clinics were held in the respective churches, although make-up clinics were often held in the offices of the Cancer Prevention and Control Program in Columbia. Eligible participants were scheduled for in-clinic appointments at the beginning of their involvement, at the end of the 12-week intervention (or time-concordant in the control group), and at the end of 1 year. At the first visit, questionnaires were obtained, anthropometric measurements were taken, and a fasting blood sample was drawn. The same protocol was followed for the post-intervention and final in-clinic appointments.

Questionnaire-derived data—Prior to the first clinic, participants were mailed questionnaires designed to assess demographics⁶; dietary intake (using a version of the National Cancer Institute food-frequency questionnaire modified for use in South Carolina)³⁰; physical activity³¹; depression³²; social support³³; and social desirability and approval, which previously have been found to either modify or confound effects of the intervention or bias dietary or other self-reports.^{34,35} Participants brought completed questionnaires to the clinic where they were reviewed for completeness and accuracy.

Anthropometric data—All blood pressure and anthropometric measurements, including height, hip and waist circumferences, total body weight,³⁶ and fat mass obtained via bioelectrical impedance assessment (BIA),³⁷ were taken during the clinic visits by trained study staff. Height was measured to the closest cm by use of a stadiometer. Using a GulickTM measuring tape, hip and waist circumferences were obtained by measuring the widest part of the hips and immediately above the iliac crest, respectively. Weight and fat mass were measured on a Tanita TBF 300A[®] electronic scale precise to 0.1 kg and 0.1% fat, respectively. BMI was calculated by standard formula.

Objectively measured physical activity data—Participants were provided Sensewear[®] armband monitors (www.bodymedia.com). The monitors provide valid assessments of total energy expenditure, intensity of physical activity, and bouts of physical activity.^{38,39} Using software provided by Sensewear[®] participants' age, date of birth, height and weight, current smoking habits, and dominant hand were used to calibrate each armband monitor. Participants were requested to wear the monitors for 7 days during periods of wakefulness, but not while sleeping. Only subjects with 4 days of monitoring based on armband monitor usage were allowed to enter the study. Amount of time spent sleeping was obtained from the Pittsburgh Sleep Quality Index (PSQI).⁴⁰ In order for a day to “count” in achieving a minimum of 4 days of usable data, at least 20 of the 24 hours in any day needed to be accounted for by wearing the armband plus time reported spent sleeping (e.g., 14 hours of armband usage + 8 hours of sleep would be acceptable). Among all participants, the mean number of days of usable data was 5.2 ± 1.0 days, with a minimum of 4 days (i.e., needed to qualify for entry into the study).

Laboratory-derived data—Blood was collected in ethylenediamine tetraacetic acid (EDTA) Vacutainers[®], centrifuged at 3000 rpm for 20 minutes, and plasma specimens were aliquoted and stored at -80°C until they were analyzed for CRP and IL-6. Plasma cytokine levels were measured using an enzyme-linked immunosorbent assay kit (Quantikine kits DCRP00 [for CRP] and HS600B [for IL-6]) according to manufacturer's instructions. All

samples were run in duplicate (CRP: CV=3.9%, sensitivity=0.022 ng/ml; IL-6: CV=3.7%, sensitivity=0.110 g/ml).

Statistical Power and Data Analysis

Power was based on an RCT focused on the effects on coronary heart disease risk factors, including CRP. Based on a standard, this would provide slightly >80% power to detect an estimated reduction of 0.4 mg/L in CRP with 132 subjects/group; and 0.5mg/L in CRP with 85 subjects/group.⁴¹ Based on results from the first two, of three, intervention waves that indicated a larger than expected reduction in CRP in subjects who attended 60% of all class sessions, an interim analytic data set was created that was used for statistical analysis.

Summary statistics were used to describe the study population at baseline separately for both intervention and control groups. Comparisons of baseline characteristics by intervention status were made using χ^2 tests for categoric variables and two-sample t-tests for continuous variables. In addition, all categoric variables were examined to ensure that each stratum contained at least 10% of the total population. Variables were recoded if necessary. Linear mixed models (Proc MIXED in SAS) were used to compute least-square Ms and 95% CIs for each dependent variable comparing those in the intervention (i.e., treatment) group to controls after adjustment for age and smoking status (i.e., current, former, or never) at baseline, and the baseline value for the outcome measure.

For example, when analyzing 12-week follow-up CRP values as the outcome, the baseline CRP values were included in the list of covariates. Church was fit as a random factor using the *RANDOM* statement because randomization occurred at the church level and not the individual level. The primary outcome variable for this analysis was CRP. IL-6 was also analyzed as a dependent variable. Secondary anthropometric outcomes included BMI, waist-to-hip ratio (WHR), and body fat percent.

The main analyses were performed for the 12-week and 1-year follow-ups. For the 12-week follow-up analyses, two CRP values were removed from the respective analyses due to elevated studentized residuals (i.e., -6.0 and 8.4) and Cook's D values of 1.99 and 0.22 which are higher than the suggested cut-point (i.e., 4/sample size = 0.025). For the 1-year follow-up analyses, one CRP value with a studentized residual of 5.5 and a Cook's D of 0.23 was removed. For the 12-week analyses, IL-6 values were log-transformed to obtain normally distributed model residuals and least-square Ms were back-transformed for presentation. All analyses were performed on all subjects and then stratified by gender. All statistical tests were based on hypotheses determined a priori, and therefore no adjustment was made for multiple comparisons.

The first round of analyses was based on intention to treat (ITT), including data on subjects on for whom there were evaluable endpoint measures ($n=159$). Following this, a variety of post hoc analyses were conducted designed to examine the effect of the intervention "dose." This utilized a three-level intervention status variable which included controls; those in the intervention group who attended <60% of intervention classes (the optimal cutpoint in terms of intervention response, which also coincides with minimal cutpoints for compliance and overall response rates^{42,43}); and those in the intervention group who attended 60% of intervention classes. All analyses described above were repeated for the three-level intervention status exposure variable. After additional adjustment for BMI, the interpretation of the results did not change for CRP or IL-6.

Because there were 19 pairs of same-household participants (e.g., husband-wife or mother-daughter combinations) three different post hoc ancillary analyses were performed to assess the possible effect of non-independence. First, the intraclass correlation for baseline CRP

(using the covariance parameter estimates from Proc MIXED) was computed. Second, one member of each of the 19 pairs of same-household participants was removed at random and the models then rerun. Third, data from all same-household participants were retained in the analyses, but an additional RANDOM statement was included using an unstructured covariance matrix, in PROC MIXED, to account for potential differences in between-subject variation associated with household. Finally, to account for individuals lost to follow-up, PROC MI in SAS was used to impute missing data as an alternative ITT analysis.

Results

A total of 437 potential participants were approached in the 14 churches included in Waves 1 and 2 (i.e., from March 2010 to July 2012); Wave 3 has yet to be completed. Of these, 61 were excluded (57 incomplete/failed screening assessment, four refusals). Of the remaining 376 subjects, 31 were lost to follow-up due to one church dropping out; 91 did not attend the baseline clinic; and 95 subjects were lost to follow-up prior to final endpoint determination. Of the remaining 254 healthy participants recruited from 13 churches enrolled in Waves 1 and 2, a total of 159 attended both baseline and the 3-month follow-up clinics and therefore had evaluable endpoint data for analyses. A total of 80 (Wave 1: women=53, men=10; Wave 2: women=14, men=3) were in the intervention arm, and 79 (Wave 1: women=44, men=15; Wave 2: women= 16, men=4) were controls.

Baseline characteristics of the study population are presented by intervention arm (Table 1). Those randomized to the control group tended to be older than those in the intervention group (mean age 57.5 ± 9.6 vs 54.2 ± 10.8 years). Although participants were generally well educated (39% of intervention group and 44% of controls had completed college), intervention group subjects were more likely than the control group to have less than a high school education (31% vs 14%). Overall, the majority of participants were women (80%), who were much less likely to be married compared to men (56% vs 91%). Individuals were, on average, obese (mean BMI of intervention group= 33.6 ± 7.6 , control group= 32.6 ± 6.3). CRP values were initially high at baseline (mean CRP of intervention group 3.8 ± 3.9 mg/l, control group= 3.5 ± 3.9).

Results stratified by intervention status are shown for both 12 weeks and 1 year (Table 2). At both 12 weeks and 1 year, both women and men in the intervention group had significant reductions in WHR. The influence of the intervention on many other parameters was not as consistent (e.g., women had a marginal increase in BMI and there was no effect in men). Results from analyses using imputed data were consistent with these findings. Despite their small sample size, at 1 year men in the intervention group had a significant 36% decrease in CRP levels. Although there were intervention effects observed for the main outcome variable, CRP, in none of these analyses was a significant relationship observed for IL-6.

As shown in Table 3, women who attended 60% classes (about 54% of the intervention group) evinced 18% lower CRP values at 12 weeks, whereas the difference in WHR was confined mainly to women who attended < 60% of classes (Table 3). Of note, compared to those attending <60% of classes, those attending 60% classes were more likely to be women (93% vs 73% men, $p=0.03$); widowed (21% vs 0%, $p=0.01$); and to have either never used or currently use alcohol (28% vs 11% and 44% vs 28%, $p=0.01$). Whereas <60% attendees were more likely to be former alcohol users compared to 60% attendees (61% vs 28%, $p=0.01$), no differences in CRP at baseline were observed between those attending <60% of classes vs 60% (5.5 ± 6.2 vs 5.2 ± 5.2 , $p=0.89$). At 1 year, there was a suggestion of lower CRP in women attending 60% classes, and significantly lower WHRs were seen among women regardless of class attendance. As for the intervention-control analyses shown in Table 2, there was no effect of intervention dose on IL-6.

A post hoc logistic regression analysis indicated that those who did not attend Clinic 2 (i.e., after 12 weeks (and therefore for whom there were no outcome data) were more likely than those who did to have fair or poor health (OR=3.32, 95% CI=1.35, 8.16).

Results of the first of the three types of analyses designed to account for the 19 pairs of same-household participants revealed no change estimates beyond reduced power due to decreasing sample size by 12%. Results of the PROC MIXED model with the unstructured covariance matrix with the additional *RANDOM* statement also revealed no change, overall or by gender. Finally, the intraclass correlation of baseline CRP levels was zero, indicating that data from subjects within household were independent. Taken together, these additional analyses indicate that the influence of some of the participants being from the same household was negligible and therefore all available data could be used in analyses.

Discussion

Significantly lower levels of CRP, the main inflammatory marker measured in this study, was observed in women who attended 60% of all HEALS classes. In a much smaller group of men (i.e., ~20% of the total sample), CRP levels were lower in intervention group members after both 12 weeks and 1 year. Of interest, the principle cytokine associated with CRP, IL-6, did not evince a similar intervention effect.

Subjects were, on average, obese and had very high levels of CRP. The intervention was presented as an opportunity to change lifestyle, not as a weight loss program, and there was no apparent difference in weight between the intervention and control groups. Individuals in the intervention group had consistently lower WHR than did subjects in the control group at both 12 weeks and 1 year. There is a literature on populations that are “metabolically obese” (i.e., who have attributes associated with obesity evident at relatively lower total body weight).^{16,44,45} This phenomenon may be related to the relatively larger impact of intra-abdominal adiposity,⁴⁶ which may be difficult to discern using BMI.

Distinguishing the effect of increased metabolic activity due to central obesity using BMI as the indicator would be especially difficult when individuals are involved in physical activity that adds lean body mass while simultaneously reducing intra-abdominal adipose stores. African Americans may be particularly susceptible to loss of intra-abdominal fat; hence, the observed correlated effects of CRP and WHR decreasing in the intervention group.

As noted previously, it was not possible to obtain complete data on all participants enrolled at baseline. Those who withdrew from the study did not differ from participants for any characteristic presented in Table 1, except for perceived health. Given that those who dropped or withdrew from the study were more likely to have poorer than average health, it is a matter of conjecture what influence the intervention would have had if these subjects had been retained. It is likely that these drop-outs had higher levels of chronic inflammation,^{17,18,47} a possibility partially corroborated by findings that drop-outs had nonsignificantly higher mean baseline CRP values compared to participants (4.8 ± 5.0 vs 4.3 ± 5.9 , $p=0.40$). These less-healthy individuals may be more resistant to healthy changes in lifestyle (i.e., had lower motivation to succeed); however, there is evidence that those who are less healthy actually may be more susceptible to diet interventions.⁴⁸

Some discussion of the expression of principles of CBPR in the design of the intervention is warranted. Community members perceived that diet was an issue profoundly affecting health and expressed interest in increasing consumption of plant foods, which are a rich source of bioactive phytochemicals; the biologic properties of which made them prime candidates for past interventions.^{49,50} Such components of diet are generally known to have

a much wider safety margin with chronic use than do anti-inflammatory drugs⁵¹ and are therefore excellent targets for dietary intervention aimed at reducing inflammatory markers.

Many food items that are used traditionally in the South Carolina diet have anti-oxidative, anti-inflammatory, anti-mutagenic, and chemopreventive properties. These include peas, beans, and lentils⁵²; cruciferous vegetables⁵¹; allium vegetables, including onion and garlic⁵³; and tea.⁵⁰ Community members expressed the need to “ground” the intervention partly in what is familiar, and therefore consumption of these foods was emphasized in the diet-related components of the intervention.

In order to maintain good community relations, delayed intervention churches were provided support in the navigation to resources and services related to health disparities, as long as they were not related to nutrition and physical activity. This support had the added benefit of acting as an “attention control.” All of the churches, in both groups, had some form of health ministry, which enabled them to assimilate and distribute information to congregants.

Community members also saw a need to increase physical activity, and evidence was shared on its role in mechanisms further upstream in inflammation-modulated processes including carcinogenesis,⁵⁴ atherosclerosis and stroke,^{55,56} and diabetes and metabolic syndrome.^{57,58} A major barrier to any lifestyle intervention is the additional stress that it places on people and institutions already under economic and social stress. The stress reduction was designed to be consistent with the religious traditions of the participants and integrated to enhance well-established neuro-endocrine pathways^{59, 60} and lifestyle behaviors, especially diet and physical activity,^{6,61} known to affect health. The fact that the intervention was delivered by lay church members speaks to the feasibility of the approach and its potential for greater sustainability and wider dissemination.

Although these members of the African-American faith community are clearly suffering from very high levels of inflammation, they do not necessarily typify all such individuals or even all African Americans. Participants in this study are similar to those in past reports of faith-based research conducted in African-American communities with respect to gender, age, and BMI.^{62–64} However, it is less clear how these populations compare to each other and to the larger African-American community on other predictors of inflammation. Although conducted in the South, where 55% of all African Americans live, and 45% of these in rural areas, subjects were recruited within driving radius of a university campus, which may have resulted in a population that is less generalizable to those recruited from even more rural settings. The relatively low participation by men precluded the apparent difference in baseline CRP values between the genders from reaching significance (i.e., 3.4 ± 5.3 in men vs 4.7 ± 5.7 in women).

The intervention, although demanding, was of lower intensity than other, clinic-based work.^{6,65} Also, the mean class attendance was only 60% of classes. So, it is hard to judge how much more successful this intervention might have been had attendance been higher and the intervention more intense. Although it was created using principles of CBPR, participants were wary of data collection, especially an extensive battery of questionnaires and blood collection. As these considerations concern implementation as a research project, it is important to understand how the HEALS intervention might be implemented as a “best practice.” Future work is needed to refine and disseminate this intervention and to test its applicability in other settings.

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Table 1Population and Lifestyle Characteristics at Baseline by Intervention Status, M $\pm$ SD or *n* (%)

Characteristic	Intervention Status		
	Intervention ^a (n=80)	Control (n=79)	<i>p</i> -value*
Age (years)^b	54.2 $\pm$ 10.8	57.5 $\pm$ 9.6	0.04
Gender^c			
Female	67 (84)	60 (80)	0.22
Male	13 (16)	19 (24)	
Marital Status^c			
Married or living with partner	48 (60)	52 (66)	0.80
Widowed	9 (11)	6 (8)	
Divorced or separated	14 (18)	14 (18)	
Single, never married	9 (11)	7 (9)	
Education Status^c			
High school or less	25 (31)	11 (14)	0.03
Some college	26 (33)	24 (30)	
Complete college	17 (21)	25 (32)	
Postgraduate	12 (15)	19 (24)	
Employment Status^c			
Full-time	44 (55)	38 (48)	0.19
Part-time	5 (6)	8 (10)	
Retired	20 (25)	28 (35)	
Not employed	11 (14)	5 (6)	
Perceived Health^c			
Excellent or very good	33 (41)	30 (38)	0.90
Good	38 (48)	39 (49)	
Fair or poor	9 (11)	10 (13)	
Smoking Status^c			
Current or Former	17 (21)	15 (19)	0.72
Never	63 (78)	64 (81)	
Alcohol Use^c			
Current	16 (20)	27 (34)	0.08
Former	34 (43)	23 (29)	
Never	29 (37)	29 (37)	
Physical Activity Level^d			
Sedentary or Infrequent	21 (27)	16 (21)	0.35
< Recommended activity	22 (28)	17 (22)	
Recommended activity	36 (46)	44 (57)	
BMI^b	33.6 $\pm$ 7.6	32.6 $\pm$ 6.3	0.40
Waist-to-Hip Ratio^b	0.86 $\pm$ 0.09	0.85 $\pm$ 0.07	0.26

Characteristic	Intervention Status		
	Intervention ^a (n=80)	Control (n=79)	p-value [*]
Body Fat Percentage ^b	41.4 ± 9.5	39.8 ± 9.0	0.27
CRP (mg/l) ^b	3.8 ± 3.9	3.5 ± 3.9	0.63
IL-6 (pg/ml) ^c	2.7 ± 2.0	1.9 ± 1.7	0.32

^a Column percentages may not equal 100 due to rounding.

^b p-value based on *t*-test

^c p-value based on Chi-square tests or Fisher's exact test for comparisons with cell counts of 5

^d Based on recommendation of 30 minutes of moderate to intense physical activity 5 days a week

^e Wilcoxon rank sums test used for this comparison

^{*} p-value based on test of difference in means or, for IL-6, Wilcoxon rank sums test

CRP, C-reactive protein; IL-6, interleukin-6

Table 2

Inflammatory and Anthropometric Outcomes, Overall and Stratified by gender^a

Variable/Treatment	All Subjects (N=159)			Women (n=127)			Men (n=32)		
	LSM (95% CI)	p-value*		LSM (95% CI)	p-value*		LSM (95% CI)	p-value*	
12-WEEK FOLLOW-UP									
CRP (mg/l)									
Intervention	4.0 (3.3, 4.7)	0.16		4.1 (3.4, 4.9)	0.32		2.3 (1.0, 3.6)	0.07	
Control	4.5 (3.8, 5.2)			4.5 (3.7, 5.3)			3.6 (2.6, 4.7)		
IL-6 (pg/ml) ^b									
Intervention	2.1 (1.9, 2.4)	0.57		2.3 (2.0, 2.7)	0.55		1.6 (1.3, 2.0)	0.82	
Control	2.0 (1.8, 2.3)	--		2.2 (1.8, 2.7)			1.6 (1.2, 2.1)		
BMI									
Intervention	33.1 (33.0, 34.3)	0.08		33.0 (32.4, 33.7)	0.13		32.0 (31.0, 33.1)	0.66	
Control	33.6 (32.4, 33.8)	--		33.6 (33.0, 34.3)			32.2 (31.5, 33.0)		
Fat Percentage									
Intervention	41.6 (39.8, 43.4)	0.91		43.9 (42.0, 45.9)	0.58		34.5 (29.4, 39.7)	0.32	
Control	41.7 (39.8, 43.6)	--		44.5 (42.4, 46.5)			31.7 (27.9, 35.4)		
Waist-to-Hip Ratio									
Intervention	0.84 (0.82, 0.86)	0.02		0.81 (0.79, 0.84)	0.23		0.90 (0.88, 0.95)	0.04	
Control	0.86 (0.84, 0.88)	--		0.83 (0.80, 0.85)			0.93 (0.91, 0.95)		
1-YEAR FOLLOW-UP									
CRP (mg/l)									
Intervention	4.1 (3.4, 4.8)	0.16		4.4 (3.8, 5.6)	0.54		3.0 (1.8, 4.2)	0.02	
Control	4.7 (4.0, 5.4)			4.7 (3.8, 5.6)			4.7 (3.5, 5.9)		
IL-6 (pg/ml) ^b									
Intervention	2.7 (2.3, 3.2)	0.57		2.9 (2.4, 3.5)	0.55		2.0 (1.3, 2.6)	0.53	
Control	2.9 (2.4, 3.3)			3.1 (2.5, 3.7)			2.2 (1.7, 2.6)		
BMI									
Intervention	32.6 (31.9, 33.2)	0.08		32.6 (31.7, 33.6)	0.12		30.8 (29.7, 31.8)	0.45	

Variable/Treatment	All Subjects (N=159)			Women (n=127)			Men (n=32)		
	LSM (95% CI)	p-value *		LSM (95% CI)	p-value *		LSM (95% CI)	p-value *	
Control	33.2 (32.5, 33.8)			33.3 (32.3, 34.3)			31.3 (30.4, 32.2)		
Fat Percentage									
Intervention	40.4 (39.3, 41.5)	0.14		43.1 (41.8, 44.3)	0.22		30.0 (27.4, 32.6)	0.51	
Control	41.1 (40.1, 42.2)			43.8 (42.5, 45.0)			30.8 (28.7, 33.0)		
Waist-to-Hip Ratio									
Intervention	0.84 (0.82, 0.86)	<0.01		0.81 (0.78, 0.84)	<0.01		0.92 (0.89, 0.95)	0.05	
Control	0.88 (0.86, 0.90)			0.85 (0.82, 0.88)			0.96 (0.93, 0.98)		

^aLSM (and 95% CIs) from repeated measures regression are adjusted for baseline outcome value, age, and smoking status.

^bFrom analysis using log values which were back-transformed for presentation

* p-value represents change between intervention and control groups.

CRP, C-reactive protein; IL-6, interleukin-6; LSM, least-squares mean

Table 3
Inflammatory and Anthropometric Outcomes Overall and Stratified by gender for three-Level Intervention Status

Variable/Treatment	All Subjects (N=159)		Women (n=127)		Men (n=32)	
	LSM (95% CI)	p-value*	LSM (95% CI)	p-value*	LSM (95% CI)	p-value*
12-WEEK FOLLOW-UP						
CRP (mg/l)						
60% Classes ^b	3.7 (2.9, 4.6)	0.05	3.7 (2.8, 4.5)	0.05	2.6 (0.2, 5.3)	0.44
<60% Classes ^b	4.4 (3.5, 5.3)	0.76	4.8 (3.9, 5.7)	0.54	2.2 (0.7, 3.7)	0.08
Control	4.5 (3.8, 5.2)	--	4.5 (3.7, 5.3)	--	3.6 (2.5, 4.8)	--
IL-6 (pg/ml)^c						
60% Classes	2.3 (1.8, 2.3)	0.33	2.4 (2.0, 2.9)	0.30	1.2 (0.7, 2.1)	0.28
<60% Classes	2.0 (1.7, 2.4)	0.94	2.1 (1.7, 2.6)	0.87	1.7 (1.3, 2.3)	0.83
Control	2.0 (1.9, 2.7)	--	2.1 (1.8, 2.5)	--	1.6 (1.3, 2.0)	--
BMI						
60% Classes	32.9 (32.0, 33.7)	0.04	32.8 (32.0, 33.5)	0.06	32.6 (31.2, 34.1)	0.36
<60% Classes	33.4 (32.5, 34.2)	0.46	33.4 (32.5, 34.3)	0.67	31.2 (30.4, 31.9)	0.11
Control	33.7 (33.0, 34.3)	--	33.6 (33.0, 34.3)	--	31.9 (31.3, 32.5)	--
Fat Percentage						
60% Classes	41.6 (39.4, 43.8)	0.99	43.7 (41.4, 45.9)	0.47	37.2 (29.8, 44.5)	0.09
<60% Classes	41.8 (39.5, 44.0)	0.86	44.3 (41.8, 46.7)	0.88	31.1 (26.4, 35.8)	0.78
Control	41.6 (39.8, 43.4)	--	44.4 (42.4, 46.4)	--	30.4 (27.1, 33.7)	--
Waist-to-Hip Ratio						
60% Classes	0.83 (0.83, 0.88)	0.78	0.84 (0.81, 0.86)	0.52	0.89 (0.86, 0.93)	0.05
<60% Classes	0.81 (0.79, 0.84)	<0.01	0.78 (0.75, 0.81)	<0.01	0.92 (0.90, 0.94)	0.17
Control	0.86 (0.84, 0.88)	--	0.83 (0.80, 0.85)	--	0.93 (0.92, 0.95)	--
1-YEAR FOLLOW-UP						
CRP (mg/l)						
60% Classes	3.8 (2.9, 4.7)	0.07	3.9 (3.0, 4.9)	0.18	3.8 (1.3, 6.3)	0.39
<60% Classes	4.5 (3.6, 5.4)	0.65	5.1 (3.9, 6.2)	0.60	2.7 (1.2, 4.2)	0.03

Variable/Treatment	All Subjects (N=159)			Women (n=127)			Men (n=32)		
	LSM (95% CI)	p-value*	LSM (95% CI)	p-value*	LSM (95% CI)	p-value*			
Control	4.7 (4.0, 5.5)	--	4.7 (3.8, 5.6)	--	4.8 (3.6, 6.1)	--			
IL-6 (pg/ml)^d									
60% Classes	2.8 (2.2, 3.3)	0.77	2.9 (2.3, 3.5)	0.61	2.2 (1.1, 3.2)	0.92			
<60% Classes	2.7 (2.1, 3.3)	0.51	2.9 (2.2, 3.6)	0.63	1.8 (1.0, 2.6)	0.38			
Control	2.9 (2.4, 3.3)	--	3.1 (2.5, 3.7)	--	2.1 (1.7, 2.6)	--			
BMI									
60% Classes	32.4 (31.7, 33.1)	0.05	32.4 (31.4, 33.5)	0.07	31.4 (29.3, 33.4)	0.95			
<60% Classes	32.9 (32.1, 33.7)	0.46	33.0 (31.8, 34.2)	0.64	30.5 (29.1, 31.8)	0.31			
Control	33.2 (32.6, 33.8)	--	33.3 (32.3, 34.3)	--	31.3 (30.4, 32.2)	--			
Fat Percentage									
60% Classes	40.4 (39.2, 41.7)	0.25	43.0 (41.6, 44.3)	0.64	30.9 (26.9, 34.9)	0.82			
<60% Classes	40.3 (39.0, 41.7)	0.20	43.3 (41.8, 44.9)	0.72	28.6 (25.8, 31.3)	0.23			
Control	41.1 (40.1, 42.2)	--	43.8 (42.5, 45.1)	--	30.5 (28.5, 23.4)	--			
Waist-to-Hip Ratio									
60% Classes	0.85 (0.83, 0.87)	0.01	0.82 (0.79, 0.85)	0.06	0.92 (0.86, 0.98)	0.22			
<60% Classes	0.83 (0.81, 0.85)	<0.01	0.79 (0.75, 0.82)	<0.01	0.92 (0.88, 0.96)	0.10			
Control	0.88 (0.86, 0.90)	--	0.85 (0.82, 0.87)	--	0.96 (0.93, 0.98)	--			

Note: n for 60% Classes, <60% Classes, and Controls were 37, 43, 79 for all subjects; 27, 40, 60 for women; 19, 10, 3 for men.

^aLSM (and 95% CIs) from repeated measures regression are adjusted for baseline outcome value, age, and smoking status.

^b60% Classes indicates those who attended 60% of intervention classes and <60% Classes indicates those who attended <60% of intervention classes.

^cFrom analysis using log values which were back-transformed for presentation

*p-value represents change between intervention and control groups.

CRP, C-reactive protein; IL-6, interleukin-6; LSM, least-squares mean