# Physical Activity, Exercise, and Inflammatory Markers in Older Adults: Findings from The Health, Aging and Body Composition Study

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**OBJECTIVES:** To examine the association between physical activity and inflammatory markers, with consideration for body fatness and antioxidant use.

**DESIGN:** Cross-sectional study, using baseline data from the Health, Aging and Body Composition Study.

**SETTING:** Metropolitan areas surrounding Pittsburgh, Pennsylvania, and Memphis, Tennessee.

**PARTICIPANTS:** Black and white, well-functioning men and women (N = 3,075), aged 70 to 79.

**MEASUREMENTS:** Interviewer-administered questionnaires of previous-week household, walking, exercise, and occupational/volunteer physical activities. Analysis of covariance was used to examine the association between activity level and serum C-reactive protein (CRP), interleukin-6 (IL-6), and plasma tumor necrosis factor alpha (TNFα) with covariate adjustment. Antioxidant supplement use (multivitamin, vitamins E or C, beta carotene) was evaluated as an effect modifier of the association.

**RESULTS:** Higher levels of exercise were associated with lower levels of CRP ($P<.01$), IL-6 ($P<.001$), and TNFα ($P=.02$) (e.g., CRP = 1.95 mg/L for no exercise and 1.72 for >180 min/wk). Adjustment for body fatness attenuated the associations somewhat. Use of antioxidant supplements modified the CRP ($P_{interaction}=.01$) and IL-6 ($P_{interaction}=.08$) associations such that concentrations were low in those taking supplements (e.g., CRP = 1.79–1.84 across exercise levels) and higher in nonsupplement users who did no exercise (2.03) than in those who did the most (1.72). Among nonexercisers, higher levels of other physical activity were related to lower levels of CRP ($P<.01$) and IL-6 ($P=.02$) but not TNFα ($P=.36$), even after accounting for body fat.

**CONCLUSION:** Inflammatory markers are lower in older adults with higher levels of exercise and nonexercise activity and in antioxidant supplement users regardless of exercise level. **J Am Geriatr Soc 52:1098–1104, 2004.**

**Key words: interleukin-6, C-reactive protein, tumor necrosis factor alpha, physical activity, inflammation, exercise, aging**

Certain serum cytokines and acute-phase proteins serve as markers of systemic inflammation, including tumor necrosis factor alpha (TNFα), C-reactive protein (CRP), and interleukin-6 (IL-6).[1] Inflammation is related to a variety of chronic diseases and conditions including rheumatoid arthritis,[2] hypertension,[3] cardiovascular disease,[4–6] peripheral vascular disease,[7] diabetes mellitus,[8,9] osteoporosis,[10] and cancer.[11] Chronic inflammation is also associated with increased age[12] and obesity.[8,13,14] Given the growing aged population in the United States and the strong link between inflammation and disability and total mortality in the elderly,[15–18] identifying lifestyle factors that might lower inflammation levels seems warranted.

Numerous studies suggest that acute bouts of intense or prolonged exercise can stimulate an inflammatory response,[19] but less well studied is the effect of regular physical activity or exercise on levels of inflammatory

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markers.[19] Four studies have found that regular exercise training of various duration and intensity decreases resting levels of TNFα and CRP levels in younger individuals.[14,20–22] More recently, a number of cross-sectional studies found higher levels of reported physical activity or measured fitness to be associated with lower plasma concentrations of IL-6 and CRP in various age groups, from young adults to the elderly.[17,23–28]

It is becoming increasingly clear that physical activity and fitness may be related to lower levels of the inflammatory markers, but many studies have examined only leisure-time activity and have not considered whether different types of activity with different intensity levels may have differential relationships with inflammatory marker levels. Published studies have primarily examined CRP.[20–22,25–27]

One mechanism through which physical activity may reduce systemic inflammation is through its effect on body weight and fatness.[29] Obesity is associated with inflammation, and adipose tissue is a known source of proinflammatory cytokines.[30] In studies that have adjusted for body mass index (BMI) or waist-to-hip ratio, the association between physical activity and inflammatory markers remains, but none of the studies have examined the potential effect modification of body fatness.[17,23,24,26,27] Moreover, lifestyle factors such as antioxidant use may modify the association between physical activity and inflammatory marker levels, but this has not been examined. Identifying modifiers of inflammation is potentially important in developing appropriate strategies and treatments to minimize adverse chronic health outcomes in the elderly. This cross-sectional analysis is designed to examine the association between physical activity levels and markers of inflammation in the elderly and to further explore any interaction with body fat/obesity and antioxidant vitamin use.

## METHODS

### Study Population

Participants were from the Health, Aging and Body Composition Study, a cohort of 3,075 black and white men and women. The population, which is 48.5% male and 58.3% white, was recruited in 1997–98 from the metropolitan areas surrounding the two field sites, the University of Pittsburgh and the University of Tennessee at Memphis. Eligibility requirements included being aged 70 to 79 at baseline and reporting no difficulty walking one-quarter of a mile, climbing 10 steps without resting, or performing activities of daily living, no use of special equipment for ambulation, no history of cancer treatment in the previous 3 years, and no plan to move from the area for 3 years. Of the initial 3,075, 30 had no valid data for any of the inflammatory markers, another 65 were missing valid data on body fatness, and 16 more were missing data on physical activity.

### Physical Activity

Physical activity was measured at baseline through an interviewer-administered questionnaire. Participants were asked whether they had performed any activities from the following categories in the previous 12 months (with appropriate examples given in each category): gardening or yard work, housework, stair climbing, walking for exercise, other walking, aerobics/calisthenics, weight training, high-intensity exercises, moderate-intensity exercises, or work/volunteer/caregiving activities. If they had participated in a given activity, the frequency and duration spent in the activity in the previous 7 days was ascertained, as was the intensity level or pace for the exercise and walking questions. Approximate metabolic equivalent unit (MET) values were assigned to each activity category to derive a caloric expenditure estimate in kcal/kg/h.[31] An exercise variable was created from the sum of the aerobics/calisthenics, weight training, high-intensity exercises, and walking for exercise (reported as brisk). Total physical activity of all types of those who did not report these types of exercise was examined.

### Markers of Inflammation

The inflammatory markers were measured in serum (CRP and IL-6) or plasma (TNFα) that had been drawn during the clinic visit after an overnight fast within 2 weeks, on average, of the baseline questionnaire and frozen at −70°C until assayed. IL-6 and TNFα were measured in duplicate using enzyme-linked immunosorbent assays (ELISA) (R&D Systems, Minneapolis, MN). The detectable limits for IL-6 (using HS600 Quantikine kit) and TNFα (using HSTA50 kit) were 0.10 pg/mL and 0.18 pg/mL, respectively. Serum levels of CRP were measured in duplicate using ELISA based on purified protein and polyclonal anti-CRP antibodies (Calbiochem, San Diego, CA), as previously described.[32] The CRP assay was standardized according to the World Health Organization First International Reference Standard with a sensitivity of 0.08 mg/L. Twenty-six participants had IL-6 above the limits of detection of the assay, and their values were set to the limit of 12 pg/mL. Laboratory coefficients of variation for each of the assays were CRP, 5.14%; IL-6, 13.09% to 18.08%; and TNFα, 14.75% to 14.87%. The final number of participants with valid data available for analysis of IL-6, CRP, and TNFα were 2,861, 2,956, and 2,798, respectively.

## COVARIATES

The baseline questionnaire assessed smoking and drinking habits, with smoking coded as never, current, or former, and current drinking as none, one to seven drinks/wk, or more than seven drinks/wk. Participants were asked to bring all medications and supplements used in the previous 2 weeks to their clinic visit. Current antiinflammatory use (including Celecoxib, oral salicylates such as aspirin, nonsteroidal antiinflammatories such as ibuprofen, and oral corticosteroids) was determined from the medication inventory. Use of antioxidant supplements was defined as any current use of one or more of the following: multivitamin, vitamin E or C, or beta-carotene. Disease prevalence was assessed through self-reported measures of physician-diagnosed conditions at baseline as well as medication use. For this analysis, presence of the following conditions with a potential relationship to inflammation was considered in the models: hypertension, arthritis, cardiovascular disease, cerebrovascular disease, peripheral vascular disease, diabetes mellitus, osteoporosis, and respiratory disease. To account for the

presence of acute inflammation, it was noted whether participants had had an upper respiratory tract infection within 2 weeks of their clinic visit. BMI (kg/m$^2$) was calculated from measured height and weight and used linearly. Total body fat was measured using fan beam dual-energy x-ray absorptiometry (Hologic QDR4500A, software version 8.21, Waltham, MA). Visceral abdominal fat was measured using computerized tomography (9800 Advantage, General Electric, Milwaukee, WI, in the Pittsburgh site; Somatron Plus, Siemens, Iselin, NJ, and PQ2000S, Picker, Cleveland, OH, in Memphis) with the data from all sites analyzed at the University of Colorado Health Sciences Center according to a standardized protocol.[33] Fat measures were also used linearly in the models.

### Statistical Analysis

All analyses were performed using SAS software (SAS Institute, Inc., Cary, NC). The inflammatory marker measurements were not normally distributed, so all values were log-transformed before statistical analysis. Analysis of covariance was used to compare inflammatory marker levels across physical activity categories. Least squares means were used to compare levels of activity. The geometric mean values of the markers are presented in the tables rather than the log values, for ease of interpretation. Three levels of exercise activity were examined based on the distribution of exercise level in the participants: none, 1 to 179 min/wk, and 180 min/wk or more. For the analysis of other physical activity in nonexercisers, activity was divided into approximate quartiles based on the kcal/kg/wk expended. For exercise and other physical activity, tests for linear trend were performed with contrast statements using orthogonal polynomial coefficients based on the median values of activity in each quartile. Statistical interactions between race and sex and physical activity for IL-6, CRP, and TNFα were tested using cross-product terms within the models. Three different statistical models are presented for the primary analyses: one with adjustment

**Table 1. Characteristics of Participants by Level of Exercise**

| Characteristic | Exercise (min/wk) | | |
|---|---|---|---|
| | 0 | >0 to <180 | ≥180 |
| Sample size | 1,836 | 657 | 471 |
| Age, mean ± SD | 73.6 ± 2.9 | 73.6 ± 2.9 | 73.6 ± 2.9 |
| Male (%) | 45.7 | 52.2 | 54.6 |
| White (%) | 51.1 | 67.0 | 76.4 |
| Body mass index, kg/m$^2$, mean ± SD | 27.8 ± 5.0 | 27.2 ± 4.4 | 26.1 ± 4.0 |
| Total fat, cm$^2$, mean ± SD | 26.6 ± 8.9 | 25.5 ± 8.0 | 23.7 ± 7.7 |
| Visceral fat, cm$^2$, mean ± SD | 146 ± 69 | 140 ± 65 | 133 ± 64 |
| Inflammatory markers, median (range)* | | | |
| IL-6, pg/mL | 1.9 (0.2–16.0) | 1.8 (0.3–13.2) | 1.5 (0.4–14.1) |
| TNFα, pg/mL | 3.2 (0.6–29.5) | 3.2 (1.0–15.1) | 3.0 (0.8–16.2) |
| CRP, mg/L | 1.8 (0.2–63.9) | 1.6 (0.2–30.8) | 1.3 (0.2–85.2) |
| Smoking, % | | | |
| Never | 42.8 | 45.0 | 46.5 |
| Former | 43.9 | 48.9 | 48.8 |
| Current | 13.3 | 6.1 | 4.7 |
| Current drinking, % | | | |
| None | 53.4 | 45.2 | 44.2 |
| <1–7/wk | 40.0 | 46.1 | 47.4 |
| >1/day | 6.7 | 8.7 | 8.5 |
| Antiinflammatory use, % | 52.1 | 54.0 | 53.3 |
| Antioxidant vitamin use, %† | 41.2 | 52.5 | 58.0 |
| Diseases, % | | | |
| Hypertension | 67.8 | 63.3 | 49.7 |
| Arthritis | 16.0 | 19.5 | 15.9 |
| Cardiovascular | 22.8 | 22.2 | 21.9 |
| Cerebrovascular | 8.7 | 7.3 | 6.4 |
| Peripheral vascular | 5.8 | 3.4 | 3.8 |
| Respiratory | 25.7 | 23.1 | 17.2 |
| Diabetes mellitus | 25.9 | 21.8 | 15.9 |
| Osteoporosis | 14.4 | 16.7 | 13.4 |
| Upper respiratory tract infection | 6.6 | 6.4 | 4.5 |

* Some participants were missing data for the inflammatory markers: interleukin-6 (IL-6), n = 103; tumor necrosis factor-alpha (TNFα), n = 166; C-reactive protein (CRP), n = 8.

† Any use of multivitamins, vitamin C, vitamin E or derivatives, or beta-carotene or derivatives.

for age, sex, and race; a second, with further adjustment for smoking, alcohol use, antiinflammatory drug use, antioxidant vitamin use, prevalent disease, and recent upper respiratory tract infection; and a third, with further adjustment for total body fat and visceral fat, because these may mediate a reduction in inflammation with increasing physical activity. To more fully examine the potential for disease prevalence to confound the physical activity and inflammatory marker relationship, the main associations were reexamined after removing persons with various diseases from the analysis. Whether BMI and antioxidant use were effect modifiers of the relationship between the inflammatory markers and exercise was examined by including cross-product terms in the models and through stratified analysis. A *P*-value $<.05$ was considered to be significant for all analyses.

## RESULTS

The median values (interquartile ranges) for the whole population for IL-6, TNFα, and CRP were 1.83 (1.27–2.81 pg/mL), 3.16 (2.45–4.11 pg/mL), and 1.67 (0.99–3.11 mg/L), respectively. Table 1 shows that participant characteristics varied by exercise group. Women, blacks, and current smokers were less likely to report regular exercise, whereas drinking activity increased with level of exercise. BMI and levels of IL-6, TNFα, and CRP were lower in those who exercised, as were prevalence of hypertension, cerebrovascular disease, peripheral vascular disease, respiratory disease, and diabetes mellitus.

The association between exercise level and the inflammatory markers is presented in Table 2. There is a significant trend toward a lower IL-6 concentration with increasing amount of exercise ($P<.001$) that is not appreciably attenuated with adjustment for body fat ($P=.006$). TNFα concentration also decreases with increasing level of exercise ($P=.019$), and in this case, further adjustment for body fat attenuates the association ($P=.071$). Similarly, CRP decreases across increasing levels of exercise ($P=.006$), and the linear trend ($P=.096$) and all pairwise comparisons between exercisers and nonexercisers become statistically insignificant after adjustment for body fatness.

Because more than half of the cohort did not report participating in any exercise, the association between nonexercise physical activity and the inflammatory markers was examined separately in these participants (Table 3). IL-6 ($P=.014$) and CRP ($P=.001$) were lower in those reporting more total physical activity, and in these analyses, adjustment for body fatness did not appreciably alter the estimates. Although statistical tests suggest a significant linear trend across quartiles for IL-6 and CRP, the association with IL-6 appears to indicate more of a threshold effect, with participation in any but the lowest level of activity being associated with lower IL-6. TNFα was not linearly related to total activity, although TNFα was somewhat lower in the second and third quartiles than in the first. The amount and type of activity performed are indicated in Figure 1. Across quartiles of activity, there are substantial increases in household and work-related activities done outside the home. To address the concern that those who did the least amount of activity were inactive due to illness, the association between CRP and total activity was reexamined after removing participants with specific inflammation-related diseases. The association with CRP was still significant after removal of those with vascular disease ($P=.022$), diabetes mellitus ($P=.010$), and respiratory disease ($P=.025$).

**Table 2. Inflammatory Marker Concentrations by Level of Exercise**

| | Exercise (min/wk) | | | |
|---|---|---|---|---|
| Inflammatory Marker | 0 | >0 to <180 | ≥180 | *P*-trend |
| Interleukin-6 (pg/mL) | | | | |
| Participants, n | 1,772 | 635 | 454 | |
| Model 1‡ | 2.05 | 1.88* | 1.68* | <.001 |
| Model 2§ | 2.01 | 1.92 | 1.75* | <.001 |
| Model 3‖ | 1.99 | 1.93 | 1.80* | .006 |
| Tumor necrosis factor-α (pg/mL) | | | | |
| Participants, n | 1,733 | 616 | 449 | |
| Model 1‡ | 3.25 | 3.13 | 3.00* | .002 |
| Model 2§ | 3.22 | 3.16 | 3.03* | .019 |
| Model 3‖ | 3.22 | 3.16 | 3.06† | .071 |
| C-reactive protein (mg/L) | | | | |
| Participants, n | 1,829 | 656 | 471 | |
| Model 1‡ | 1.97 | 1.84 | 1.63* | <.001 |
| Model 2§ | 1.95 | 1.86 | 1.72* | .006 |
| Model 3‖ | 1.92 | 1.88 | 1.79 | .096 |

*Note:* Presented as geometric mean values with statistical analysis performed on log-transformed data due to non-normal distribution.
Versus no exercise group: * $P<.01$; † $P<.05$.
‡ Adjusted for age, sex, and race.
§ Additionally adjusted for smoking, alcohol, use of antiinflammatory drugs, use of antioxidant vitamins, hypertension, arthritis, cardiovascular disease, cerebrovascular disease, peripheral vascular disease, osteoporosis, diabetes mellitus, respiratory disease, and acute upper respiratory tract infection.
‖ Additionally adjusted for total body fat, and visceral fat.

The relationship between exercise and inflammatory marker was further examined by level of BMI and by antioxidant vitamin use. In a model including the BMI and exercise interaction term, and adjusted for all covariates noted in the tables except body fat (Model 2), BMI was significantly related to CRP ($P<.001$), IL-6 ($P<.001$), and TNFα ($P=.010$). There was no statistically significant interaction between BMI and exercise for CRP ($P=.872$), IL-6 ($P=.325$), or TNFα ($P=.330$). For antioxidant supplement use, fully adjusted exercise models (Model 3) with the supplement-by-exercise interaction terms in the models revealed that antioxidant use was significantly associated with IL-6 ($P=.013$) but not CRP ($P=.106$) or TNFα ($P=.850$). There was a significant interaction for CRP with the use of antioxidant vitamins ($P=.007$), such that CRP levels decreased with exercise in those who did not take antioxidant vitamins ($P=.018$) (Figure 2), but for the antioxidant users, CRP levels were similar across exercise groups ($P=.901$), such that taking an antioxidant supplement was associated with CRP levels similar to those seen in those who exercised 180 min/wk or more and did not take supplements. The interaction was similar in the IL-6 analysis, although not significant ($P=.076$), and there was no interaction with TNFα ($P=.576$).

**Table 3. Inflammatory Marker Concentration by Quartile of Nonexercise Physical Activity of Those Who Do No Exercise**

| Inflammatory Marker | Non-Exercise Physical Activity (kcal/kg/wk) | | | | |
|---|---|---|---|---|---|
| | <33 | 33–57 | 58–97 | >97 | *P*-trend |
| Interleukin-6 (pg/mL) | | | | | |
| Participants, n | 449 | 428 | 448 | 447 | |
| Model 1‡ | 2.27 | 1.99* | 2.03† | 1.93* | .002 |
| Model 2§ | 2.25 | 1.97* | 2.03† | 1.97* | .014 |
| Model 3‖ | 2.25 | 1.99* | 2.03† | 1.97* | .019 |
| Tumor necrosis factor-α (pg/mL) | | | | | |
| Participants, n | 439 | 424 | 434 | 436 | |
| Model 1‡ | 3.35 | 3.19 | 3.13† | 3.19 | .149 |
| Model 2§ | 3.35 | 3.16† | 3.13† | 3.22 | .299 |
| Model 3‖ | 3.35 | 3.16 | 3.13† | 3.22 | .362 |
| C-reactive protein (mg/L) | | | | | |
| Participants, n | 470 | 440 | 459 | 460 | |
| Model 1‡ | 2.32 | 1.97* | 2.01† | 1.82* | <.001 |
| Model 2§ | 2.27 | 1.97† | 1.99† | 1.86* | .001 |
| Model 3‖ | 2.25 | 1.99† | 1.99† | 1.86* | .002 |

Presented as geometric mean values with statistical analysis performed on log-transformed data due to non-normal distribution.
Versus no exercise group: * $P<.01$; † $P<.05$.
‡ Adjusted for age, sex, and race.
§ Additionally adjusted for smoking, alcohol, use of antiinflammatory drugs, use of antioxidant vitamins, hypertension, arthritis, cardiovascular disease, cerebrovascular disease, peripheral vascular disease, osteoporosis, diabetes mellitus, respiratory disease, and acute upper respiratory tract infection.
‖ Additionally adjusted for total body fat, visceral fat.

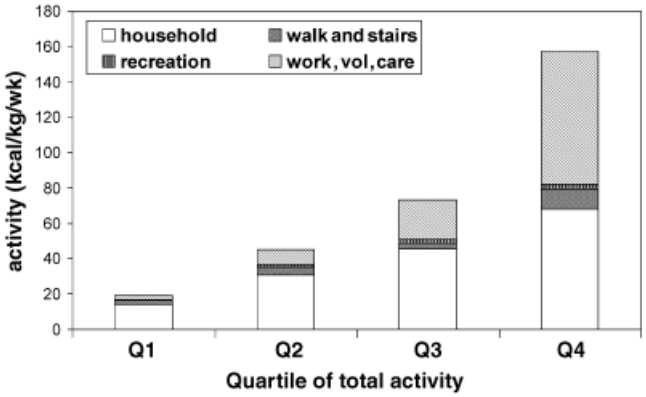


**Figure 1.** Types of activity reported by quartile of nonexercise physical activity by those who reported no exercise. Types included household; walking and stair climbing; recreation; and occupational, volunteer, or caregiving activities.

## DISCUSSION

These results are consistent with other epidemiological studies in finding an inverse association between physical activity and markers of inflammation such as CRP and IL-6. Additionally, lower TNFα levels were found in participants reporting higher exercise levels, consistent with the effects of exercise in intervention studies. No published study has examined different categories of physical activity in association with markers of inflammation. The current findings point to a significant association between exercise and nonexercise physical activities such as household and occupational or volunteer work and lower levels of inflammatory markers. This information is important, particularly in the elderly, who infrequently perform exercise activities[34] and who may be more receptive to incorporating other forms of activity into their daily routine.

More modest associations were found between activity and TNFα. Exercise intervention studies have reported significant lowering of TNFα levels and soluble TNF receptor type I with exercise training of different durations and intensities in overweight and obese participants.[14,22] These studies were small, and in one case, the baseline TNFα values were much higher than the average levels in this study.[22] Perhaps TNFα is not as sensitive to changes in physical activity as the other markers. TNFα, along with IL-1, is important in initiating the inflammatory response, whereas IL-6 is released later in the cascade.[35] IL-6 is also the primary stimulus for hepatic synthesis of CRP.[36] The relative consistency of the association between activity, IL-6, and CRP compared with TNFα seen here may thus be related to these relationships. One study found differential relationships between certain cardiovascular risk factors, including physical activity, and the inflammatory markers IL-6 and CRP, suggesting that the two markers do not necessarily track together and that CRP may also be upregulated through IL-6-independent pathways.[28] Nevertheless, the current data are consistent with those from the one other study examining both markers, which found a significant relationship between physical activity and both IL-6 and CRP.[17]

All three of the inflammatory markers examined have been related to measures of body fatness.[13,14,37] These relationships with measures of BMI are not surprising, given that IL-6 and TNFα are produced within adipose tissue,[30] although only IL-6 is thought to be released into the circulatory system from this source.[38] Because IL-6 is the main stimulus for liver production and secretion of CRP,[36] higher circulating IL-6 could result in higher CRP and hence an association between CRP and obesity. Because of these relationships, it was felt to be important to carefully examine body fatness as a potential confounder and as an effect modifier of the association between physical activity and the inflammatory markers. Adjustment for total and visceral fat did not significantly change any of the associations seen with total physical activity, but it did attenuate the associations seen for TNFα and CRP with exercise. These results imply that lowering body fat with regular exercise may be partially responsible for the lower levels of the markers, but it is not the sole mechanism that could explain this association. When the sample was stratified by BMI to examine the CRP and exercise association, no significant effect modification was found by level of BMI. Again, this implies that the exercise and inflammatory marker association is consistent across body composition, suggesting that lower body fatness is not the sole mechanism explaining this association.

An interesting interaction between IL-6 and CRP levels and the use of antioxidant vitamin supplements, in which those who took supplements tended to have lower IL-6 and

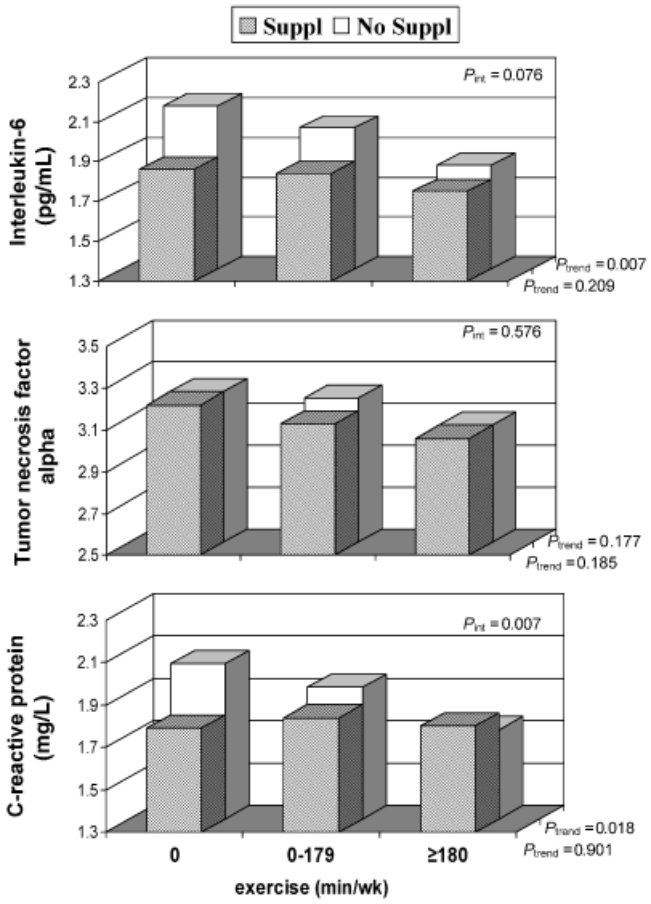


**Figure 2.** Inflammatory marker concentration by level of exercise and supplementation with antioxidant vitamins. Presented as geometric means due to nonnormal distribution of the data. Taking antioxidant vitamins defined as reported use of any multivitamin, vitamin C, vitamin E or its derivatives, or beta-carotene or its derivatives. Log values adjusted for age, sex, race, smoking, alcohol, use of antiinflammatory drugs, hypertension, arthritis, cardiovascular disease, osteoporosis, cerebrovascular disease, peripheral vascular disease, diabetes mellitus, respiratory disease, acute upper respiratory tract infection, total body fat, and visceral fat. Number of subjects with and without supplement use in the analyses were, respectively: 1,329 and 1,532 in interleukin-6 analysis, 1,302 and 1,496 in tumor necrosis factor alpha analysis, and 1,371 and 1,585 in C-reactive protein analysis.

CRP levels regardless of exercise level, was found. Although interesting, it must be considered that supplement use could be serving as a marker of an overall healthy lifestyle or of other healthy lifestyle choices, and thus the interaction noted could be the result of bias. This observation may be worthy of further exploration in future studies.

Limitations of this study include the cross-sectional design, which does not allow for the inference of a cause-and-effect association. Additionally, although a number of potential confounding factors were statistically controlled for in the models, it is possible that residual confounding may have contributed to the associations found. However, exercise intervention studies have demonstrated that, even with protocols of varying length and intensity, TNFα and CRP can be reduced from baseline levels with exercise.[14,20,21] Even though participants may have been inactive because of prevalent inflammation-related diseases, statistical control for prevalent conditions and removal of participants with specific inflammation-related diseases suggests otherwise. However, given their advanced age, a large percentage of the cohort may also have had prevalent subclinical conditions such as cardiovascular disease or airway obstruction that were not controlled for, and which might have biased the results. Not many of the elderly participants reported participation in moderate or vigorous exercises, limiting the ability to look for dose-response associations with the inflammatory markers, but this is consistent with participation in leisure-time activity reported by elderly U.S. adults.[34] Strengths of this study include the large sample of male and female and black and white participants and the examination of exercise and nonexercise physical activity. Last, a number of potential confounders and modifiers of the associations between physical activity and markers of inflammation were considered.

The data suggest that exercise and nonexercise physical activity is associated with lower levels of systemic inflammation as indicated by the plasma markers IL-6, CRP, and TNFα. Differences in body fatness by activity level may explain part but not all of the associations noted. It was also found that the use of antioxidant vitamins modified the exercise and inflammatory marker associations such that a dose-response physical activity association was found for IL-6 and CRP in persons not taking antioxidant vitamins but not in those who used the supplements. This work adds to the growing number of studies suggesting that physical activity, a modifiable lifestyle factor, is associated with reduced levels of inflammatory markers. As a result, physical activity should be strongly encouraged in older adults, for it not only improves physical function and the maintenance of independence, but may also attenuate chronic low-level systemic inflammation.

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