Relationship Between Physical Activity and Inflammation Among Apparently Healthy Middle-aged and Older US Adults

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**Background:** Physical activity has been associated with a reduced risk of coronary heart disease, but the mechanism underlying this association is unclear. Because coronary heart disease is increasingly seen as an inflammatory process, it might be reasonable to hypothesize that physical activity reduces risk of coronary heart disease by reducing or preventing inflammation.

**Methods:** The study examined the relationship between physical activity and elevated inflammation as indicated by a high C-reactive protein level, white blood cell count, or fibrinogen level. Study subjects were 3638 apparently healthy US men and women 40 years and older who participated in the Third National Health and Nutrition Examination Survey.

**Results:** More frequent physical activity was independently associated with a lower odds of having an elevated C-reactive protein level. Compared with those engaging in physical activity 0 to 3 times per month, the odds of having an elevated C-reactive protein level was reduced among those engaging in physical activity 4 to 21 times per month (odds ratio, 0.77; 95% confidence interval, 0.58-1.02) and 22 or more times per month (odds ratio, 0.63; 95% confidence interval, 0.43-0.93) (P for trend, .02). Similar associations were seen for white blood cell count and fibrinogen levels.

**Conclusions:** More frequent physical activity is independently associated with a lower odds of having elevated inflammation levels among apparently healthy US adults 40 years and older, independent of several confounding factors. The results suggest that the association between physical activity and reduced coronary heart disease risk may be mediated by anti-inflammatory effects of regular physical activity.

Arch Intern Med. 2002;162:1286-1292

A **NUMBER OF STUDIES** have shown that physical activity is associated with a lower risk of coronary heart disease (CHD). The mechanisms underlying this association are not entirely clear. Recent reports have indicated that markers of inflammation are predictive of increased CHD incidence and mortality, and the development of CHD is increasingly being viewed as an inflammatory process. As such, it might be reasonable to hypothesize that if physical activity lowers CHD risk, it may do so in part by preventing or reducing inflammation.

A few previous reports using general population samples have indicated that higher levels of physical activity are cross-sectionally associated with lower levels of inflammation markers such as C-reactive protein (CRP), white blood cell (WBC) count, and fibrinogen. These reports, however, have been limited to some degree in certain respects. First, although several studies examined the association of physical activity with fibrinogen, they did not examine the relationship between physical activity and other markers of systemic inflammation, such as CRP, that are more established markers of CHD risk. Second, some of the studies failed to control for healthy lifestyle practices, particularly diet, which could potentially confound the association between physical activity and inflammation. Third, several studies included persons with prevalent diseases (such as CHD, stroke, asthma, emphysema, or rheumatoid arthritis) that could confound this association. Some studies have attempted to statistically adjust for the presence of certain diseases such as CHD, but one might expect that despite such adjustment, there may be residual confounding according to disease severity. A better strategy is to exclude persons with diseases that could affect both physical activity and inflammation levels.

Given the limitations of the studies noted above, the relationship between physical activity and inflammation in the
SUBJECTS AND METHODS

STUDY DESIGN AND PARTICIPANTS

The present study was based on data from the Third National Health and Nutrition Examination Survey (NHANES III), a cross-sectional study conducted from 1988 to 1994 by the National Center for Health Statistics of the US Centers for Disease Control and Prevention, Atlanta, Ga. Using a complex, stratified, cluster-sampling procedure, investigators assembled a group of 33994 participants who were representative of the noninstitutionalized civilian US population. Data on each study participant were collected in 2 stages. The first stage involved an initial in-home interview in which participants were questioned about demographic factors, health status, health behaviors, and a variety of other variables. The second stage, which was conducted within 4 weeks after the initial in-home interview, involved the administration of additional questionnaires as well as a medical examination by a board-eligible physician. Further details about the design of NHANES III have been published elsewhere.23

Of the 33994 persons who participated in NHANES III, we excluded 22346 persons who were younger than 40 years. Excluding persons younger than 40 years was necessary because fibrinogen, one of the markers of inflammation included in our analysis, was measured only in subjects 40 years or older. We additionally excluded 6332 persons with a history of diseases that could affect inflammation levels as well as one’s ability to engage in physical activity. These diseases included rheumatoid arthritis, asthma, bronchitis, emphysema, cancer, stroke, diabetes, heart failure, and CHD. The history of each of the aforementioned diseases was based on self-report, except for history of CHD, which was based on self-reported CHD history (myocardial infarction or angina) or an electrocardiogram reading during the medical examination that was indicative of a previous myocardial infarction. We further excluded 1478 persons with missing data on physical activity, markers of inflammation, or other covariates used in our analyses. After all exclusions, the present study arrived at a sample of 3638 apparently healthy adults 40 years or older.

RESULTS

Of the 3638 persons included in our analyses, the mean age was 52.9 years. Slightly less than half (49.4%) of the participants were female. Whites made up 87.8% of the sample, while 8.1% of the participants were black and 4.1% were members of other racial or ethnic groups. Approximately 34.8%, 31.5%, and 33.7% of the participants reported engaging in physical activity 0 to 3 times, 4 to 21 times, and 22 or more times in the preceding month, respectively. The means±SEs of CRP, WBC count, and fibrinogen—in a representative sample of apparently healthy US adults after control for diet and other potential confounding factors.

STUDY MEASURES

Physical Activity

During the household interview, participants were asked about the frequency with which they participated in the following 9 activities during the preceding month: walking a mile without stopping, jogging or running, swimming, regular dancing, aerobic exercise or aerobic dancing, riding a regular bicycle or exercise bicycle, calisthenics, garden or yard work, and weight lifting. They were also asked to list the frequency with which they participated in up to 4 physical activities not included in the list noted above. From this information, the total number of times that a participant engaged in any physical activity in the month before the household interview was determined. Only frequency of activity was recorded; no information on duration of each activity was collected.

Markers of Inflammation

During the medical examination, phlebotomists obtained blood samples from participants by venipuncture. These samples were stored in vials in refrigerated (4°C to 8°C) or frozen (−20°C) conditions, and then sent to analytical laboratories for testing. We chose to focus on 3 markers of inflammation that were obtained from these samples: serum CRP level, WBC count, and plasma fibrinogen level. These markers were chosen because each has been associated with CHD risk.9,10,14 The CRP level was measured by means of latex-enhanced nephelometry. This method of measuring CRP was unable to detect CRP levels less than 0.22 mg/dL. Thus, persons with CRP levels below this limit were simply grouped together as having CRP levels that were in the “undetectable” range of less than 0.22 mg/dL. White blood cell count was measured using a quantitative automated hematology analyzer (Coulter Counter Model S-Plus JR; Coulter Electronics, Hialeah, Fla). Fibrinogen levels were determined by a quantitative assay that compared the clotting time of a blood sample with the clotting time of a standardized fibrinogen preparation. Quality control procedures relating to the measurement of CRP, WBC, and fibrinogen in NHANES III have been described previously.24

Continued on next page
Other Study Measures

To see whether physical activity was associated with inflammatory markers independent of other factors that may predict inflammation, we included a number of other factors in our analyses as control variables. Demographic factors included age, sex, 3 categories of race (white, black, and other), and 3 categories of educational attainment (<12 years, 12 years, and >12 years). Total and high-density lipoprotein cholesterol levels, as well as serum glucose levels, were also included in analyses. Anthropometric measurements allowed us to include 2 measures of obesity—body mass index (weight in kilograms divided by the square of height in meters) and waist-to-hip ratio—in our analyses. Smoking status was accounted for on the basis of self-reported smoking of cigarettes, cigars, or pipes, and participants were categorized as current smokers, past smokers, or never smokers. Frequency of alcohol consumption during the preceding month was categorized as 0 times, 1 to 30 times, and more than 30 times. Current use of antihypertensive medications, based on self-report, was also included as a variable in our analyses. Finally, we also controlled for dietary factors that we thought might be indicative of an overall healthy diet. These dietary factors included total fat intake as well as total intake of vitamins C and E as determined from a 24-hour food recall completed during the household interview.

STATISTICAL ANALYSIS

The main goal of the analysis was to determine whether the frequency of physical activity was associated with markers of inflammation after controlling for other factors. We accomplished this goal by running multivariable adjusted logistic regression models that used physical activity as the predictor variable and dichotomous indicators of elevated levels of CRP, WBC, or fibrinogen as the outcome variables. The other study measures were included as control variables in these models. In the logistic regression models, the frequency of physical activity in the previous month was divided into 3 levels: low (0-3 times), medium (4-21 times), and high (22 or more times). The highest 2 activity levels were entered into the models as dummy variables, and the lowest activity level was treated as the referent level. These 3 levels of activity were chosen because they represented approximate tertiles of the weighted distribution of the frequency of physical activity in our sample. Elevated levels of CRP, WBC, and fibrinogen were defined as greater than or equal to 0.70 mg/dL, greater than or equal to 9550/µL, and greater than or equal to 373 mg/dL, respectively (top 10% of the weighted distribution of each marker). We decided to analyze these inflammation markers as dichotomous outcomes in a logistic regression model because the distributions of the markers (especially CRP) were quite skewed, which would have made analyzing these markers as continuous variables in a linear regression model potentially inappropriate. The top 10% cut-off points ensured that the odds ratios (ORs) generated by the logistic regression models would be roughly interpretable as relative risks. Other study measures were entered into the models as continuous independent variables (age, systolic and diastolic blood pressure, body mass index and waist-to-hip ratio, glucose, total fat intake, and total intake of vitamins C and E) or categorical variables according to the categories described above (sex, race, smoking, alcohol consumption, and antihypertensive medication use). To test whether a linear trend existed between increasing levels of physical activity and the odds of having an elevated inflammation level, we ran models that contained an ordinal physical activity variable. This ordinal variable was created by assigning values of 1, 2, and 3 to persons reporting an activity frequency of 0 to 3, 4 to 21, and 22 or more times per month, respectively.

Because NHANES III was a complex, stratified cluster sample, standard statistical techniques that assume a simple random sample are inappropriate. Consequently, all of our analyses were conducted in SUDAAN (Research Triangle Institute, Research Triangle Park, NC), a statistical program that accounts for the complex design of the NHANES III sample. In particular, SUDAAN allowed us to incorporate sampling weights that corrected for unequal probabilities of selection and differential nonresponse rates, thereby ensuring that the results of our analyses would be generalizable to the population from which the NHANES III sample was drawn (ie, the noninstitutionalized civilian population of the United States). All of the means, percentages, and ORs presented in this study are weighted. In addition, the weighting, stratification, and clustering inherent in the NHANES III sample can affect SEs, and SUDAAN uses techniques to adjust the SEs accordingly. This ensured that the SEs and P values reported in our study were valid given the complex nature of the sample.

have elevated CRP levels. Among those engaging in low, medium, and high physical activity levels, the percentages of persons with elevated CRP levels were 15.1%, 9.7%, and 6.5%, respectively (P<.001 according to χ² test). Similarly, the percentage of persons with elevated WBC or fibrinogen levels decreased with increasing levels of physical activity (χ², P<.001 and .01, respectively). In addition to showing crude associations with CRP, WBC, and fibrinogen levels, however, physical activity showed crude associations with many of the other study variables (Table 1). For example, higher levels of physical activity were less likely to be seen among women and among those taking antihypertensive medication, whereas they were more likely to be seen among those with higher education levels and those with frequent alcohol consumption. In addition, increasing levels of physical activity tended to be associated with lower mean values of age, systolic and diastolic blood pressures, total cholesterol level, body mass index, and waist-to-hip ratio. In contrast, higher physical activity levels were associated with higher mean values of high-density lipoprotein cholesterol and vitamin C intake. Physical activity did not show a clear relationship with race, although there did seem to be evidence that the middle category of physical activity was less common among black and members of other racial or ethnic groups. With regard to smoking status, there was some indication that higher levels of activity were more likely to be seen among former smokers and less likely to be seen among current smokers.

To see whether the unadjusted associations between physical activity and markers of elevated inflam-
flammation would withstand adjustment for other study variables, we ran multivariable-adjusted logistic regression models (Table 2). Model 1 showed that, as physical activity levels increased, the odds of having an elevated CRP level significantly decreased, independent of other factors. For those engaging in physical activity 4 to 21 times and 22 or more times in the previous month, the ORs of having an elevated CRP level were 0.77 (95% confidence interval [CI], 0.58-1.02) and 0.63 (95% CI, 0.43-0.93), respectively (P for trend, .02), compared with those engaging in physical activity 0 to 3 times.

Similarly, model 2 showed a significant trend between increasing physical activity levels and lower odds of having an elevated WBC count after adjustment for other factors. For those with medium and high levels of physical activity in the preceding month, the ORs of having an elevated WBC count were 0.81 (95% CI, 0.55-1.19), and 0.59 (95% CI, 0.41-0.84), respectively (P for trend, .006), compared with those with a low level of physical activity.

Model 3 showed an association between increasing physical activity and a decreasing odds of having an elevated fibrinogen level. However, the association in model 3 did not achieve statistical significance. For those engaging in physical activity 4 to 21 times and 22 or more times in the previous month, the ORs of having an elevated fibrinogen level were 0.77 (95% CI, 0.52-1.16), and 0.77 (95% CI, 0.56-1.06), respectively (P for trend, .10), compared with those with the lowest physical activity level.

We then conducted 2 sets of secondary analyses. First, because of recent interest in possible sex differences in chronic disease risk factors and outcomes, we attempted to see whether there was an interaction between sex and physical activity on inflammation outcomes. In logistic regression models, however, we found that terms representing the interaction between sex and physical activity were not significant predictors of any of the 3 inflammatory outcomes (data not shown). Second, because our categorization of physical activity into tertiles led to fairly broad categories, we sought to examine how a more refined categorization of physical activity would be related to the inflammation outcomes. Thus, we divided physical activity into quartiles (0-1 time, 2-9 times, 10-29 times, and ≥30 times in the previous month). The results from using quartiles in adjusted logistic regression models were fairly similar to those that were obtained when tertiles were used, although the linearity of the association was not as clear. For example, we found that increasing quartiles of activity tended to be associated with a reduced odds of having an elevated CRP level. For those in the second through fourth quartiles, the ORs of having an elevated CRP level compared with those in the first quartile were 0.77, 0.81, and 0.55, respectively (P for trend, .001). For the WBC outcome, the results for the second through fourth quartiles were 0.66, 0.55, and 0.51, respectively (P for trend, .005), compared with the lowest quartile of physical activity. For the fibrinogen outcome, the ORs for the second through fourth quartiles were 0.81, 0.82, and 0.77, respectively (P for trend, .17), compared with the first quartile.

The present study investigated the cross-sectional relationship between physical activity and markers of systemic inflammation among a representative sample of apparently healthy middle-aged and older US adults. The findings of the study indicated that a higher frequency of physical activity was associated with a significantly lower odds of having elevated CRP and WBC levels after adjustment for a number of potential confounding factors. The study also found that more frequent physical activity was associated with a lower odds of having an elevated fibrinogen level after controlling for confounders, although this association was not statistically significant. Overall, these results suggest that more frequent physical activity may be associated with lower levels of systemic inflammation among healthy US adults who are 40 years or older.

Previous cross-sectional studies had reported that higher activity levels were associated with lower levels of inflammation markers such as CRP, WBC, and fibrinogen. As noted earlier however, these previous studies were limited in a number of respects. First, some studies examined the association of physical activity with fibrinogen level but failed to look at the relationship between physical activity and other markers of systemic inflammation, such as CRP level, that are more estab-
lished predictors of CHD. Second, other studies failed to control for important potential confounders such as diet. Third, some of the previous studies were based on cross-sectional analyses of samples that included persons with existing diseases that could affect both inflammation and physical activity such as CHD, emphysema, and rheumatoid arthritis. This third limitation is highly problematic because it could cause one to find an inverse association between activity and inflammation, simply because the diseases with high inflammation levels are precluding persons from being active. The present study overcomes the limitations of these previous stud-

Table 1. Unadjusted Association of Other Study Variables With Physical Activity Level*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Physical Activity Level (No. of Times Physical Activity Was Engaged in During Previous Month)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low (0-3) (n = 1548)</td>
</tr>
<tr>
<td>Age, y</td>
<td>54.5 ± 0.46</td>
</tr>
<tr>
<td>Sex, % F</td>
<td>52.5</td>
</tr>
<tr>
<td>Race, %</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>85.4</td>
</tr>
<tr>
<td>Black</td>
<td>9.9</td>
</tr>
<tr>
<td>Other</td>
<td>4.7</td>
</tr>
<tr>
<td>Education, %</td>
<td></td>
</tr>
<tr>
<td>&lt;12 y</td>
<td>32.8</td>
</tr>
<tr>
<td>12 y</td>
<td>37.8</td>
</tr>
<tr>
<td>&gt;12 y</td>
<td>29.4</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>129.0 ± 0.65</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>77.3 ± 0.32</td>
</tr>
<tr>
<td>Antihypertensive medication use, %</td>
<td>14.9</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL†</td>
<td>50.1 ± 0.57</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL†</td>
<td>213.4 ± 1.45</td>
</tr>
<tr>
<td>Glucose, mg/dL‡</td>
<td>95.7 ± 0.34</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.5 ± 0.36</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.94 ± 0.01</td>
</tr>
<tr>
<td>Total vitamin C intake, mg</td>
<td>95.6 ± 4.13</td>
</tr>
<tr>
<td>Total vitamin E intake, α-tocopherol equivalents</td>
<td>9.2 ± 0.45</td>
</tr>
<tr>
<td>Total fat intake, g</td>
<td>79.4 ± 2.01</td>
</tr>
<tr>
<td>Smoking status, %</td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>41.4</td>
</tr>
<tr>
<td>Former</td>
<td>28.7</td>
</tr>
<tr>
<td>Current</td>
<td>29.9</td>
</tr>
</tbody>
</table>

*Values are mean ± SD unless otherwise stated. P values comparing the distribution of categorical variables according to activity levels are based on χ² tests; P values comparing distribution of continuous variables according to activity levels are based on analysis of variance. Numbers of persons in each activity level are unweighted; percentages and means are weighted. BP indicates blood pressure; HDL, high-density lipoprotein.

†To convert to millimoles per liter, multiply by 0.02586.
‡To convert to millimoles per liter, multiply by 0.05551.

Table 2. Multivariable-Adjusted Logistic Regression Models Assessing the Association Between Physical Activity and Markers of Inflammation*

<table>
<thead>
<tr>
<th>Physical Activity Level (No. of Times Physical Activity Was Engaged in During Previous Month)</th>
<th>Model 1: Odds Ratio (95% CI) for Elevated CRP</th>
<th>Model 2: Odds Ratio (95% CI) for Elevated WBC</th>
<th>Model 3: Odds Ratio (95% CI) for Elevated Fibrinogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (0-3) (n = 1548)</td>
<td>1.00 (Referent)</td>
<td>1.00 (Referent)</td>
<td>1.00 (Referent)</td>
</tr>
<tr>
<td>Medium (4-21) (n = 991)</td>
<td>0.77 (0.58-1.02)</td>
<td>0.81 (0.55-1.19)</td>
<td>0.77 (0.52-1.16)</td>
</tr>
<tr>
<td>High (≥22) (n = 1099)</td>
<td>0.63 (0.45-0.93)</td>
<td>0.59 (0.41-0.84)</td>
<td>0.77 (0.56-1.08)</td>
</tr>
</tbody>
</table>

*CI indicates confidence interval; CRP, C-reactive protein; and WBC, white blood cell count. Elevated CRP level was defined as 0.70 mg/dL or more. Elevated WBC count was defined as 9550/µL or more. Elevated fibrinogen level was defined as 373 mg/dL or more.

†Adjusted for age, sex, race, education, systolic and diastolic blood pressure, antihypertensive medication use, high-density lipoprotein and low-density lipoprotein cholesterol levels, glucose level, body mass index and waist-hip ratio, vitamin C and E intake, total fat intake, alcohol consumption, and smoking.


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ies to a certain extent, and therefore provides stronger evidence that a higher frequency of physical activity may help reduce levels of systemic inflammation in the general population.

Although the findings of the present study indicate that physical activity may help reduce inflammation, one must consider that this study had a number of limitations that may have affected its findings. First, our measurement of physical activity was fairly crude in that it was based on self-report and assessed only frequency of activity without considering duration of activity. In addition, although quality control measures were used to help ensure that the measurements of the inflammation markers were as accurate as possible, it is likely that there was some mismeasurement of these markers. Thus, both physical activity and inflammation were probably misclassified to a certain extent in this study. However, it is likely that such misclassification was random and would have biased our results toward the null. As such, misclassification of this type would not explain away our results, but instead would suggest that we may have underestimated the inverse association between physical activity and inflammation.

Second, our study was based on observational data. Therefore, our findings may have been due to residual confounding from some of our control variables. In addition, there may have been some unknown confounders that we did not control for, and these unknown confounders may have affected our results.

Third, as was the case with previous large-scale epidemiologic studies investigating the association between physical activity and markers of inflammation, our study was based on cross-sectional data. Consequently, the temporal ordering of the association we observed between physical activity and reduced levels of inflammation is unclear. The association could indicate that activity reduces inflammation or protects against the onset of inflammation, but it could also indicate that diseases associated with elevated inflammation levels prevent one from being active. As already noted, we minimized the possibility of the latter scenario by excluding persons with diseases that could be related to inflammation and the ability to be active. However, it is certainly possible that persons with subclinical diseases were included in our study. Thus, we cannot rule out that our results are due to the fact that diseases with high inflammation levels precluded persons from being active in our study. However, 2 studies based on very small samples have shown that physical activity can prospectively reduce CRP levels.20-22 Because of small sample sizes, the generalizability of these 2 studies is limited. Nevertheless, they suggest that the findings of the present study may reasonably be interpreted to indicate that physical activity leads to the prevention or reduction of systemic inflammation.

Assuming physical activity does indeed help prevent or reduce inflammation, what is the mechanism by which it would accomplish this effect? Strenuous physical activity can lead to muscle damage and thereby increase inflammation.23 In contrast, however, there are plausible mechanisms by which physical activity could also reduce inflammation. For example, obesity is a factor that is strongly related to higher levels of inflammation,24 and it has been suggested that physical activity may reduce inflammation by reducing obesity levels.16 However, in the present study, we observed that physical activity was associated with lower levels of inflammation even after adjustment for measures of general obesity (body mass index) and central obesity (waist-to-hip ratio). Thus, it seems unlikely that the association between activity and inflammation is mediated entirely by reductions in obesity. Other mechanisms linking exercise to lower inflammation levels may involve antioxidant effects of exercise. Although exercise increases oxidative metabolism and thereby induces oxidative stress, there is also evidence from studies involving animals and humans that adapting to long-term exercise or physical training can significantly elevate antioxidant defenses.30-34 The ability of long-term exercise to induce an antioxidant effect may explain why such exercise has been reported to reduce the susceptibility of low-density lipoprotein to oxidation35 and prevent age-related impairment in nitric oxide availability.36 Preventing low-density lipoprotein oxidation and impairments in nitric oxide availability would, in turn, help prevent endothelial injury or dysfunction and the inflammation that could result from such dysfunction. Indeed, some investigators have reported that exercise does improve coronary endothelial dysfunction in persons with existing CHD.37 Still other evidence indicates that physical training decreases the expression of adhesion molecules on leukocytes,38 a phenomenon that would presumably inhibit the inflammatory process. The effects of physical training on antioxidant defenses, oxidation of low-density lipoprotein, availability of nitric oxide, and adhesion molecule expression are far from clear, however, and explaining the association we found in terms of such effects is necessarily speculative.

In summary, the present study has demonstrated that increasing levels of physical activity are associated with lower levels of CRP and other markers of inflammation, in a representative sample of apparently healthy middle-aged and older US adults. Since elevated levels of CRP and other markers of inflammation have been shown to be important predictors of increased CHD risk, the current study implies, although it does not prove, that physical activity may lower CHD risk by reducing inflammation. Studies that examine physical activity as a prospective predictor of inflammation in general population samples are needed to definitively establish whether physical activity truly prevents or reduces inflammation, and whether this reduction accounts for the association between increased physical activity and lower CHD risk. In addition, if physical activity is proved to reduce inflammation, further research would need to clarify the duration and intensity of activity that is required to best achieve reductions in inflammation.

Accepted for publication October 15, 2001.

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