Physical Inactivity and Short-term All-Cause Mortality in Adults With Chronic Disease

Brian C. Martinson, PhD; Patrick J. O’Connor, MD, MPH; Nicolaas P. Pronk, PhD

Objective: To ascertain the relationship of physical inactivity and short-term all-cause mortality in a prospective cohort of randomly selected managed care organization members aged 40 years and older who have multiple chronic diseases.

Methods: Clinical databases were used to identify all health plan members aged 40 years and older with 2 or more chronic health conditions (hypertension, coronary heart disease, diabetes mellitus, or dyslipemia) in 1994. A random sample of 2336 members was surveyed by mail and telephone interview regarding their health-related behaviors. Survey data were linked to mortality data from the 1995 to 1997 Minnesota Death Index. Cox proportional hazards regression was used to ascertain the association between physical inactivity and subsequent all-cause mortality, adjusting for potential confounders.

Results: Members who reported less than 30 minutes a week of physical activity at baseline had a subsequent mortality risk ratio of 2.82 (P<.001) vs those with 30 or more minutes of physical activity a week. Increased mortality risk persisted (mortality risk ratio, 2.15; P<.001) after adjustments for age, sex, current smoking, functional impairment, and comorbidity score.

Conclusions: In adults with chronic diseases, the physically inactive had higher observed mortality within a 42-month period. If physical inactivity reflects an independent mortality risk, efforts to maintain physical activity in such patients may yield significant clinical benefits within a short period. By contrast, if inactivity is primarily a proxy for other factors that elevate mortality risks, a simple physician inquiry regarding inactivity may help to identify patients at risk of death.

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The relationship between physical inactivity and adverse health outcomes has been well established. A graded, inverse relationship has been demonstrated between measures of total physical activity and all-cause mortality. Studies on changes in physical activity and fitness indicate that maintenance or improvement of physical activity or fitness levels reduces the risk of all-cause mortality. Hence, the adoption and maintenance of a physically active way of life appears to improve health and delay death.

Physical inactivity is a predictor of subsequent disability in midlife and older populations. Individuals with fewer health risks tend to live longer than those with more health risks, and have fewer years of disability, with delay in onset of disability and compression of disability into fewer years at the end of life. From a managed care perspective, it is of substantial interest to consider the short-term impact of physical inactivity on mortality and morbidity. Short-term impact may allow health benefits to accrue before the members disenroll from the health plan, so that health investments made by the managed care organization provide substantial return. Moreover, short-term impact may provide compelling arguments for investment in health improvement efforts by payers and health plans.

Managed care settings provide a unique environment to improve health in defined populations. If physical inactivity is directly related to short-term all-cause mortality in a midlife and an older population, independent of chronic disease morbidity, health plans and payers may want to invest resources in programs designed to promote physically active lifestyles. This study examines the relationship between physical inactivity and short-term all-cause mortality in a prospective cohort of adults aged 40 years and older diagnosed as having a chronic disease.
SUBJECTS AND METHODS

STUDY SUBJECTS

The study was conducted at HealthPartners, a Minnesota health plan with 750,000 members in owned or contracted clinics. All members aged 40 years and older enrolled as of December 15, 1994, were potential subjects for the study. These individuals were classified using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) and pharmaceutical data bases as having or not having each of 4 chronic conditions. A diagnosis of diabetes was assigned if the member had 2 or more ICD-9-CM codes 250.xx or a filled prescription for a diabetes-specific drug, such as insulin, a sulfonylurea, or a biguanide, in 1994. Heart disease was assigned if the member had 1 or more ICD-9-CM codes 412, 413.9, 429.2, or 428.0 in 1994. Hypertension was assigned if the member had 1 or more ICD-9-CM codes 401, 401.1, or 401.9 in 1994. Dyslipemia was assigned if the member had an ICD-9-CM code of 272.4 in 1994. A more detailed description of the identification of members with specific conditions and the sensitivity, specificity, and positive predictive value of this method has been previously published. From 7571 members aged 40 years and older who had 2 or more of the chronic conditions, a random sample of 2500 (33%) was selected. The study protocol was approved in advance by the HealthPartners Institutional Review Board.

In August 1995, study subjects were surveyed by mail, with postcard reminders sent 1 week after the initial survey mailing, and a second survey mailing sent to nonrespondents 3 weeks later. Nonrespondents received telephone follow-up. Of the original 2500 subjects, 164 were unable to complete the survey because of death (n=66) or disenrollment, mailing address problems, language problems, or other problems (n=98). These subjects were considered ineligible. Another 276 nonrespondents, and 159 proxy responses, were omitted from all analyses. Thus, a total of 1901 respondents (representing 81.4% of the total eligible sample [1901/2336]) are included in the present report. Characteristics of survey respondents and nonrespondents are listed in Table 1.

DATA DEFINITIONS

The 60-question survey instrument included items on demographics, health status, use of preventive services, modifiable health risks, and readiness to change modifiable health risks. The core of the survey was adapted from the Centers for Disease Control and Prevention’s Behavioral Risk Factor Surveillance System, which has reliability coefficients for behavioral risk factors above 0.70.

The primary outcome for this study was time to death during the follow-up period of August 15, 1993, through February 1, 1999. Mortal events and dates of death were gathered from the Minnesota Death Index for the years 1995 to 1997. This index provides surveillance of deaths occurring in state residents (within and outside of the state) as reported on death certificates. We supplemented these data with information on deaths in 1998 and the first month of 1999 identified in HealthPartners’ administrative records system. Because of incomplete recording of death information in this system, deaths occurring after 1997 may be undercounted, but this is not expected to bias our estimates of the association between physical activity and mortality.

Important independent variables needed for analyses included age, sex, and chronic disease status. Age and sex were obtained from health plan administrative databases. Age was calculated in years from date of birth to the date of the initial survey and centered on its mean. Chronic disease status for heart disease, diabetes mellitus, hypertension, and dyslipemia was determined based on 1994 data, as previously described.

Physical activity was assessed using 2 measures. First, the Godin Leisure Time Exercise Questionnaire measured

RESULTS

Of the 1901 subjects who responded to the 1995 survey (81.4% response rate), 1832 had complete data on all study variables, and are the basis of this report. There were 197 observed deaths within the 42-month follow-up period.

Table 2 lists the baseline characteristics of study subjects with and without mortal events during the follow-up period. There were significant differences in those with and without events. Those with no event were significantly younger, had significantly less chronic disease, had a higher body mass index, were less likely to be impaired in their activities, and were more physically active.

Table 3 summarizes the results of 4 Cox proportional hazards regression models predicting rates of all-cause mortality. The models differ only in which baseline measures of physical activity were included as covariates. Models 1 and 2 include measures based on the number of days in the past week that respondents reported getting at least 30 minutes of physical activity. Models 3 and 4 include measures of physical activity based on the Godin Leisure Time Exercise questions. All models include baseline smoking status and measures of important potential confounders, including age, sex, comorbidity (Charlson score ≥ 3), and functional impairment status. We assessed 2-way interaction terms between the physical activity measures and smoking, and between physical activity and impairment, and none were found to be significant. We report raw coefficient estimates from the models and mortality risk ratios (MRRs) associated with these coefficients. Coefficient estimates indicate the extent to which the baseline hazard rate (mortality rate in this case) is shifted up or down in association with a given individual characteristic. The MRR provides a more intuitive expression of the coefficient estimate, describing the mortality risk associated with a characteristic relative to the reference category.

The estimated effects of the confounding variables were nearly identical across these 4 models, so we limit our discussion of these variables to the estimates from model 1. Based on these results, we see that older age was associated with higher mortality,
how many times in a 7-day period respondents reported engaging in strenuous, moderate, or mild exercise for more than 15 minutes during their free time. Specific examples of each category of activity are included in the standard Godin items. The number of times reported for each of these categories was then multiplied by 9, 5, or 3 metabolic equivalents, respectively. The total weekly leisure activity score was calculated in arbitrary units by summing these products. For analysis, a total Godin score of 0 to 12 was coded as “low”; 13 to 25, “medium”; and 26 to 280, “high.” Since approximately 12% of the sample had incomplete information for computing the Godin score, our analyses also include an indicator variable for incomplete data on this measure.

Second, respondents reported how many days in the past week they “have gotten a total of 30 minutes or more” of physical activity. Respondents were not instructed in how to define physical activity, but the context for responding was set for them by having the Godin items appear in the survey immediately before the single-item measure of physical activity. For analysis, we categorize this measure as follows: no physical activity (0 days in the past week), low physical activity (1-3 days in the past week), or high physical activity (≥4 days in the past week). Our analyses also include an indicator of missing data on this measure.

A current smoker was defined as a respondent who reported ever having smoked at least 100 cigarettes and who indicated smoking now. Subjects were defined as functionally impaired using an indicator variable coded 1 if they responded “yes” or “unsure” to any of a set of 3 questions asking whether any impairments or health problems limited any of their activities in any way, or caused them to need help with personal care needs or routine household needs. A modified Charlson score was calculated using ICD-9-CM diagnostic codes.20 Diagnoses were identified over a 12-month period preceding the survey. Any members who had not received health care services in the 12 months before the survey were assigned a missing value for a score, and did not appear in subsequent analysis. Members with health services use, but with none of the 19 chronic conditions within the Charlson index, were assigned a score of 0. Since outpatient encounters may contain “rule-out” coding, for a member to receive a weight in one of the Charlson conditions, the member must have had 2 or more diagnoses within the condition. Primary and secondary ICD-9-CM codes were included. The distribution of the Charlson score is skewed, but previous work21 has demonstrated that scores of 3 or higher are strongly predictive of mortality. Therefore, we operationalized the Charlson score as an indicator variable (1 or 0) for a score of 3 or higher. Body mass index was calculated based on self-reported body weight and height as kilograms divided by the square of height in meters.

ANALYTIC MODEL

Cox proportional hazards regression was used as the primary analytic method.

We tested the assumption of proportionality of covariate effects for the primary independent variables of interest and found no substantial violations of this assumption. Analyses were restricted to individuals with complete responses on all analysis variables. A left truncation issue occurred in our data, due to the roughly 8-month duration between identification of the study cohort and first survey administration. This was addressed in standard fashion by removing all individuals from the risk set between the point of study origin (December 31, 1994) and the initial survey contact point (August 15, 1995). To assess undue influence of any individual observation on particular coefficient estimates, we obtained residuals of the approximate changes in estimates between the full sample and the sample minus the individual observation.22 These residuals were then plotted against identification number. We identified no observations that appeared to unduly influence any of the coefficient estimates.

with a 1-SD change in age (11.4 years) increasing the likelihood of death more than 2-fold (P <.001). Women were significantly less likely than men to have died during the follow-up period (P <.001). Having a Charlson comorbidity score of 3 or greater was associated with more than a 3-fold higher likelihood of death during follow-up (P <.001). Being functionally impaired in either routine care needs or usual activities was associated with a mortality rate that was approximately 70% higher than that of the reference category (P <.002). Current smokers were nearly twice as likely to die during follow-up as were nonsmokers in the reference category (P <.008).

In model 1, baseline physical activity was assessed in terms of an indicator variable distinguishing those who reported getting less than 30 minutes of physical activity in the past week from those who got 30 minutes or more of physical activity. The risk of mortality during follow-up was higher among those who were completely sedentary at baseline (P <.001), relative to those with at least 30 minutes of physical activity in the past week. By contrast, the unadjusted risk of mortality for those totally sedentary at baseline was somewhat higher (MRR, 2.82; P <.001).

Model 2 compares those who reported getting 30 minutes of physical activity on 1 to 3 days during the past week with those who reported getting physical activity on 4 or more days in the past week, with the totally sedentary individuals being the reference group. There appeared to be a protective effect of moderate and higher amounts of physical activity, with the risk of mortality being lower for the 1- to 3-day group (P <.001) and the 4-day or more group (P <.001), relative to those who were sedentary.

In models 3 and 4, we modeled physical activity in terms of the Godin scores, categorized into tertiles. Those with Godin scores ranging from 1 to 12 were assigned to the lowest tertile; 13 to 25, the middle tertile; and 26 to 280, the highest tertile. The results of these models were quite similar to the results for models 1 and 2. Model 3 contrasts those in the lowest tertile to those in the middle and highest tertiles, and indicates a roughly 2-fold higher mortality risk associated with being in the lowest tertile of the Godin score distribution (P <.001). Model 4 also indicates a protective effect of physical activity, with fol-
The extent to which the association between inactivity and mortality is modified by the inclusion of potential confounders in the model is not observable in Table 3. Of most interest are the modifying effects of including the Charlson measure and the impairment measure. In Table 4, we present the results of model 1 (from Table 3) in a form that demonstrates the effect modification attributable to these factors. Model 1.a adjusts for all factors except for Charlson score and impairment. Models 1.b and 1.c show some slight attenuation of this association after including the Charlson measure and the impairment measure, respectively.

We were concerned that lack of physical activity among those with chronic illnesses might be an indicator of physical limitations resulting from illnesses that independently increase their risk of mortality. We tested this possibility in additional models similar to those reported in Table 3, excluding from the sample those with Charlson scores of 3 or higher. Although $P$, increased somewhat because of reduced sample size, the physical activity findings remained consistent. A third set of analyses, including the subsample with high Charlson scores but excluding those reporting functional impairment as previously defined, also yielded similar results.

In the Commentary, we present the results of model 1 (from Table 3) in a form that demonstrates the effect modification attributable to these factors. Model 1.a adjusts for all factors except for Charlson score and impairment. Models 1.b and 1.c show some slight attenuation of this association after including the Charlson measure and the impairment measure, respectively.

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Study subjects who reported baseline activity of less than 30 minutes in the past week had a significantly increased risk of mortality over the subsequent 3 years (MRR, 2.82; $P$, <.001) vs those with 30 minutes or more of physical activity a week. Increased risk persisted (MRR, 2.15; $P$, <.001) after adjustments for age, sex, current smoking, functional impairment, and comorbidity scores. This result was consistently noted using various measures of physical activity.

Other studies have reported that low levels of physical activity are predictive of subsequent higher mortality rates. Some of these reports have also noted this association following a relatively short follow-up period. For example, in the Longitudinal Study on Aging, Rakowski and Mor reported 1098 deaths among a study sample of 6901 after 5 years of follow-up for men and women aged 70 years and older. Several measures of physical activity were inversely related to mortality. In one of the largest prospective studies conducted in the area of physical activity and mortality, Kushi et al, in a 7-year follow-up of 40417 postmenopausal women aged 55 to 69 years, demonstrated a graded, inverse relationship between physical activity and all-cause mortality. Their study provides evidence that even infrequent moderate-intensity activity, ie, once or twice a week, is associated with a significant reduction in death compared with a completely sedentary lifestyle.

While it is not clear why our study failed to detect a graded, inverse association, there are various possible explanations. Prior documentation of a graded effect of physical activity may be due, in part, to a correlation between the frequency of physical activity and its intensity.
We found little high-intensity activity in this sample of respondents with multiple chronic diseases. Thus, our measures would fail to capture any part of the graded effect that is due to effects of intensity, vs frequency, of physical activity.

Another possibility is that, unlike prior studies, ours was a study of people with multiple chronic illnesses. It is possible that the benefit “profile” of physical activity is simply different in healthy vs diseased populations. Our study documents a clear benefit at the low end of the physical activity profile. There may well be declining returns to increasing activity beyond some minimum level among those with chronic disease.

Finally, in one of the largest prior studies, the graded effect of physical activity on mortality was not uniform across the range of frequency of physical activity, but was more pronounced at lower frequencies, with much less grading in effect between those reporting moderate physical activity 2 to 4 times per week and those reporting physical activity more than 4 times per week. Thus, our choice of category cut points might obscure some effect of grading at lower frequencies of activity.

The Lipid Research Clinics Mortality Follow-up Study25 showed that in men with preexisting cardiovascular disease, the observed adjusted relative risks of dying after 8½ years was 2.9 for those who were unfit as quantified via treadmill exercise test. At approximately 3 years of follow-up, the relative risk of dying was 2.15 for those who reported completely sedentary lifestyles, similar to the estimates derived in our study.
have been reported as low in Minnesota and high in enrollment rates among older, sicker health plan members influenced by rates of disenrollment. Health plan disenrollment is a primary example of a positive return on investment for programs or interventions that promote physical activity. Thus, there are 2 competing interpretations of the associations found between inactivity and mortality. The first is that older adults with multiple chronic diseases may derive a survival benefit from at least minimal physical activity (at least 30 minutes a day, once a week). The primary clinical implication of this interpretation would be that there is need for wider recognition of the potential value of maintaining at least minimal levels of physical activity. In patients with multiple chronic conditions, there is a tendency for physicians to emphasize pharmacotherapy, a strategy that has proved worth. Alternative pathways to reduce mortality, such as encouraging physical activity, may not be emphasized.

In our analyses, we did not control for functional health status, chronic conditions, or from the point of view that activity benefits are greater for more recent physical activity than for more remote years of activity, and platelet adhesion. These short-term benefits of physical activity would be especially beneficial to patients similar to those enrolled in this study— those with established hypertension, lipid disorders, diabetes mellitus, or heart disease. Although physical activity levels at baseline may reflect longer-duration physical activity over decades, it is plausible on physiologic grounds. For example, regular physical activity has been shown to decrease insulin resistance, lower blood pressure, and reduce serum fibrinogen levels, plasminogen activator inhibitor 1 activity, and platelet adhesion. These short-term benefits of physical activity would be especially beneficial to patients similar to those enrolled in this study— those with established hypertension, lipid disorders, diabetes mellitus, or heart disease. Although physical activity levels at baseline may reflect longer-duration physical activity over decades, it is plausible from the biologic point of view that activity benefits are greater for more recent physical activity than for more remote years of physical activity. Thus, efforts to increase physical activity levels of patients who have long been sedentary may still have a pronounced beneficial effect. In the present study, we did not measure lifetime physical activity or change in physical activity level during the study period.

### Table 4. Probability of Mortality During the 42-Month Follow-up Using Model 1*

<table>
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<th>Variable</th>
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<td>MRR</td>
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<td>df</td>
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*Each type of model 1 is described in the “Results” section of the text. There were 1832 subjects in each type of the model. MRR indicates mortality risk ratio; PA, physical activity; and ellipses, data not applicable.

†P<.01.
‡Defined as 1 if the subjects responded “yes” or “unsure” to any of 3 items asking whether any impairments or health problems limited their activities in any way or caused them to need help with personal care needs or routine household needs.
§Defined as those having smoked at least 100 cigarettes and those who responded affirmatively to a question asking whether they smoke cigarettes now.

However, results presented herein extend our understanding of this topic in 2 ways. First, we controlled for functional health status, chronic conditions, and comorbidities in the analytic model. Most other reports controlled only for self-reported baseline disease or for limited measures of lipid levels, blood pressure, and glucose level. We were able to control in the analyses for 19 chronic conditions as diagnosed by physicians and scored as a sum of their weights. Second, the results indicate that in some groups of patients the relationship between physical inactivity and increased mortality operates over a short period—less than 3 years.

The fact that lower levels of physical activity were associated with significantly higher mortality within only a 42-month period is important from the perspective of health plans and from the point of view of public health policy. Health plans often estimate return on investment, and discount future benefits against present costs. Demonstrating a short-term relationship between physical activity and mortality indicates the possibility of a positive health plan return on investment for programs or interventions that promote physical activity. The potential of a positive return on investment is also influenced by rates of disenrollment. Health plan disenrollment rates among older, sicker health plan members have been reported as low in Minnesota and high in southern Florida. Furthermore, researchers have shown that a substantial fraction of health care costs is attributable to low levels of physical activity, obesity, and tobacco use. Some of these costs may be averted if health plan–enrolled populations maintain favorable profiles of modifiable health risks.

These findings are not without their limitations. Although the study was well designed, population based, and prospective, it is still observational. The data do not prove that increasing physical activity will reduce mortality—only that physical inactivity is associated with double the risk of mortality after adjustment for comorbidity, functional status, and other factors.

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An alternative interpretation of our primary findings is that inactivity among such patients is, to some extent, a proxy or marker for other factors that elevate mortality risk (eg, degree of functional impairment, severity of chronic diseases, or disease progression). While we have attempted to assess this issue several ways in our analyses, we cannot completely rule out the possibility that this explains some portion of the observed association. If this second interpretation is correct, the primary clinical implication would be that a simple inquiry regarding lack of activity in the past week may help clinicians to identify which of their patients with multiple chronic diseases are at increased risk of dying in the short-term.

While the data we present do not establish that increased levels of physical activity will reduce mortality in the short-term, other reports suggest that such a benefit is plausible on physiologic grounds. For example, regular physical activity has been shown to decrease insulin resistance, lower blood pressure, and reduce serum fibrinogen levels, plasminogen activator inhibitor 1 activity, and platelet adhesion. These short-term benefits of physical activity would be especially beneficial to patients similar to those enrolled in this study— those with established hypertension, lipid disorders, diabetes mellitus, or heart disease. Although physical activity levels at baseline may reflect longer-duration physical activity over decades, it is plausible from the biologic point of view that activity benefits are greater for more recent physical activity than for more remote years of physical activity. Thus, efforts to increase physical activity levels of patients who have long been sedentary may still have a pronounced beneficial effect. In the present study, we did not measure lifetime physical activity or change in physical activity level during the study pe-
short-term relationship of activity to mortality may actually derive from the cumulative effect of many previous years of activity or inactivity. However, physical activity in patients with chronic disease may exert a beneficial effect within a short time. Benefits of physical activity are likely mediated metabolically, and most proposed metabolic effects of physical activity are rapid in onset and relatively short.40

There is considerable interest in addressing the problem of physical inactivity through public health policy. The data presented herein suggest that, for some subjects—and possibly for many people—policy initiatives that encourage at least low levels of physical activity may have the potential to yield substantial clinical and public health benefits. Interventions that might be considered include such practices as walking a dog daily, walking in malls, or walking in the neighborhood. One logical group to target is those who are completely inactive—approximately 25% of adults in most surveys, including ours.

In a previous report,41 it was established that patient readiness to change to better health-related behaviors is higher in patients with chronic conditions than in those without such chronic conditions. Furthermore, lower health care charges are associated with higher levels of physical activity,27 and the net potential savings appear to be greatest in those with highest charges—namely, those who are oldest or sickest. Thus, the benefits of increased physical activity may be greatest in the oldest, sickest patients. Contrary to the fears of many elderly patients, and possibly their physicians, we observe no increased mortality associated with increasing levels of physical activity.

Despite the limitations of these data, we believe the results are interesting and important. The data establish that physical activity in patients with chronic conditions is associated with twice the rate of subsequent mortality over a short follow-up period than that of more active people, and suggest that clinical and population-based interventions to increase physical activity in such patients may have the potential to decrease mortality and costs of care.

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REFERENCES


Correction

Error in Symbol. In the article titled “Are We Inhibited? Renal Insufficiency Should Not Preclude the Use of ACE Inhibitors for Patients With Myocardial Infarction and Depressed Left Ventricular Function,” published in the September 25th issue of the ARCHIVES (2000;160:2645-2650) in the “Results” subsection of the “Abstract” on page 2645, lines 8 through 11 should have read as follows: “In stratified models, the receipt of an ACE inhibitor was associated with a 37% (16%-52%) lower mortality for patients who had poor renal function (serum creatinine level, >265 µmol/L [>3 mg/dL] and a 16% (8%-23%) lower mortality for patients who had better renal function.” The journal regrets the error.
ally a beer-drinking country. In the 1990s it is much more common for Danes to drink wine, and consequently intellectual, social, and personality differences between wine drinkers and no wine drinkers may be far less pronounced than in the 1970 population study by Grønbæk et al.6 Nevertheless, 20 years later, we found 2 sets of characteristics significantly associated with beer and wine drinking in a Danish population. In support of our conclusions, a 1999 study of the introduction of coffee into the traditionally tea-drinking society of Scotland shows similar relationships to social status and health risk factors.14

Social status gradients in health are one of the most consistent findings of public health epidemiology.15,16 The present study demonstrates strong relationships among beverage choice and intelligence and socioeconomic status (these results were corroborated by a parallel analysis of a 20- to 26-year-old sample from the same cohort). A substantial part of the variation in intelligence (and personality) is independent of social status, and consequently, residual confounding is likely when a single index of social status is used as a covariate in studies of the beneficial health effects of wine.

Our results suggest that wine drinking is associated with optimal social, intellectual, and personality functioning, while beer drinking is associated with suboptimal characteristics. According to Bouchard,17 virtually any dimension of behavior scaled from the less valued to the more valued correlates positively with IQ. The dramatic IQ differences and differences in personality measures between wine drinkers and beer drinkers strongly suggest that wine and beer drinking in Denmark is associated with many known and unknown factors that may affect health, morbidity, and mortality.

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Correction

In the Original Investigation by Martinson et al titled “Physical Inactivity and Short-term All-Cause Mortality in Adults With Chronic Disease,” published in the May 14 issue of the ARCHIVES (2001;161:1173-1180), an incorrect title was given for the article listed in reference 27 on page 1179. The correct title is “Relationship between modifiable health risks and short-term health care charges.”