Influence of Individual and Combined Health Behaviors on Total and Cause-Specific Mortality in Men and Women

The United Kingdom Health and Lifestyle Survey

Elisabeth Kvaavik, PhD; G. David Batty, PhD; Giske Ursin, MD, PhD; Rachel Huxley, DPhil; Catharine R. Gale, PhD

Background: Physical activity, diet, smoking, and alcohol consumption have been shown to be related to mortality. We examined prospectively the individual and combined influence of these risk factors on total and cause-specific mortality.

Methods: The prospective cohort study included 4886 individuals at least 18 years old from a United Kingdom–wide population in 1984 to 1985. A health behavior score was calculated, allocating 1 point for each poor behavior: smoking; fruits and vegetables consumed less than 3 times daily; less than 2 hours physical activity per week; and weekly consumption of more than 14 units of alcohol (in women) and more than 21 units (in men) (range of points, 0-4). We examined the relationship between health behaviors and mortality using Cox models and compared it with the mortality risk associated with aging.

Results: During a mean follow-up period of 20 years, 1080 participants died, 431 from cardiovascular diseases, 318 from cancer, and 331 from other causes. Adjusted hazard ratios and 95% confidence intervals (CIs) for total mortality associated with 1, 2, 3, and 4 poor health behaviors compared with those with none were 1.85 (95% CI, 1.28-2.68), 2.23 (95% CI, 1.55-3.20), 2.76 (95% CI, 1.91-3.99), and 3.49 (95% CI, 2.31-5.26), respectively (P value for trend, <.001). The effect of combined health behaviors was strongest for other deaths and weakest for cancer mortality. Those with 4 compared with those with no poor health behaviors had an all-cause mortality risk equivalent to being 12 years older.

Conclusion: The combined effect of poor health behaviors on mortality was substantial, indicating that modest, but sustained, improvements to diet and lifestyle could have significant public health benefits.

Arch Intern Med. 2010;170(8):711-718

Several studies have shown that specific health behaviors, including cigarette smoking,1-4 physical inactivity,5-9 higher alcohol intake,10,11 and, to a lesser extent, diets low in fruits and vegetables,12-15 are associated with an increased risk of cardiovascular disease (CVD), cancer, and premature mortality. It has been stated that these modifiable behaviors are especially important in the prevention of chronic diseases.16 Typically in these studies, mutual statistical control is made for other behaviors to identify "independent" effects. However, these poor lifestyle choices frequently coexist. To fully understand the public health impact of these behaviors, it is necessary to examine both their individual and combined impact on health outcomes.

We are aware of only 3 studies that have examined the combined effect of diet, physical activity, smoking, and alcohol intake on mortality17-19; 1 study investigated the combined effect of these factors on coronary heart disease among men only.20,21 2 additional studies investigated the combined impact of health behaviors on risk of stroke,21,22 1 investigated the combined effect of lifestyle factors on coronary heart disease in women,23 1 studied the association between 4 lifestyle factors and the risk of developing major chronic diseases,24 and 1 described the associations between 4 lifestyle factors and overweight and new onset of diabetes mellitus in elderly individuals.25 In all of these study populations, poor health behaviors were associated with increased mortality or morbidity. Of the lifestyle-mortality studies, 1 study sampled only socioeconomically advantaged female health professionals aged 34 to 59 years at baseline,10 another study was restricted to 45- to 79-year-old residents of Norfolk, England,17 while the participants of the third study were all elderly, thus limiting the generalizability of these studies.18
Herein, we examine both the individual and collective influence of smoking, diet, alcohol intake, and physical activity on 20 years’ risk of total and cause-specific mortality in men and women from a United Kingdom–wide population-based study with participants who were at least 18 years old.

**METHODS**

**THE HEALTH AND LIFESTYLE SURVEY**

The target population for the Health and Lifestyle Survey (HALS) was the entire adult population of England, Wales, and Scotland who were 18 years or older in 1984 to 1985. Details of the study have been described previously.26 A total of 12 672 addresses were randomly selected from electoral registers. In each household, 1 person aged at least 18 years was selected. For different reasons (vacant address, holiday home, business premises, demolished, untraced), 418 of the selected addresses were excluded from the study, resulting in 12 254 included addresses. A high proportion of those interviewed (n=9003) consented to a follow-up visit from a research nurse (n=7414), at which time other measurements were taken.

**DATA COLLECTION**

The interviewer administered a questionnaire that asked about demographic details, smoking, alcohol consumption, leisure time exercise activities in the past fortnight, and frequency of consumption of fruit and vegetables. The questionnaire also inquired about history of chronic diseases and current or most recent occupation. Data on occupation were used to derive occupational social class using the schema of the United Kingdom Registrar General (6 categories, with a higher score indicating greater deprivation). Height, weight, and blood pressure were measured during the subsequent visit by a research nurse. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

**ASSESSMENT OF HEALTH BEHAVIORS**

Smoking behavior was divided into 3 categories: current, former, and never smoking. Poor smoking behavior was defined as being a current smoker. Alcohol consumption was assessed by amounts of beer, cider, lager, shandy (beer and lemonade combined), sherry, vermouth, wines, spirits, liqueurs, and other types of alcoholic drinks consumed the previous week, and calculated as number of units per week (1 U=8 g of alcohol). Poor drinking behavior was defined as consuming more than 21 U per week for men and more than 14 U per week for women. Consuming less was classified as the favorable drinking behavior.9,27 Because we did not find a completely J- or U-shaped association between alcohol and mortality in preliminary analyses of this data set, the abstainers were categorized in the low-risk group.

Physical activity was assessed by number of leisure time exercise activities, such as keeping fit, sports, jogging, swimming, cycling, or dancing, engaged in during the past fortnight. The respondent was asked to report the number of times he or she had performed each specific activity during the past fortnight, and the time spent doing it. Poor physical activity behavior was defined as spending little or no time on exercise activities (<120 minutes during 1 week).

Intake of fruits and vegetables was assessed by a food frequency questionnaire reporting frequency of consumption of fresh fruit, salads, raw vegetables, root vegetables, peas and beans, green vegetables, and other cooked vegetables. There were 6 response categories: never, less than once a week, once or twice a week, most days (3-6 days), once a day, and more than once a day. The scores were recoded to represent intakes in terms of times per day. All 8 types of fruits and vegetables were summed, using the respective daily frequency of consumption, to create a daily intake frequency of total fruit and vegetable consumption. Poor dietary behavior was defined as having had fruits and/or vegetables less than 3 times daily on a yearly basis.

A health behavior score was calculated based on the 4 poor health behaviors: cigarette smoking, high alcohol intake, physical inactivity, and a low fruit and vegetable intake. Participants scored 1 point for the presence of each of the poor health behaviors. The poor health behavior score thus ranged from 0 (no poor health behaviors) to 4 (ie, 4 poor health behaviors).

In total, 98% of participants were subsequently registered on the United Kingdom National Health Service (NHS) Central Registry, and deaths from all causes, CVD, cancer and other causes were ascertained from death certificates and coded according to the International Classification of Diseases, Ninth Revision (ICD-9). Disease classifications included in the analyses were ICD-9 codes 390-434 and 436-448 for CVD and ICD-9 codes 140-209 for cancers.

**STATISTICAL ANALYSES**

From each of the 12 254 households included in the study, 1 person at least 18 years old was selected, yielding interviews with 9003 individuals (73.5%). Nonresponse was due to refusals (n=2341), failure to establish contact (n=646), or other reasons (n=264).26 The study population included in the current data analysis (n=4909) was compared with that of the 1981 census, and the populations were generally very similar for key variables such as sex (52% were women in the study sample vs 49% in the census), age (61% vs 51% were aged 20-50 years), and ethnicity (98% vs 96% were white Europeans).

We used χ² tests and t tests to examine differences in characteristics between men and women. Having ascertained that the proportional hazards assumption had not been violated,28 we used Cox proportional hazards regression with calendar time (number of days) as the time variable to examine the relationship between individual and combined health behaviors and risk of death from all causes, from CVD, cancer, and other (non-CVD) deaths.

Follow-up started at the date of the survey in 1985 and continued until date of death or May 30, 2005. Models for each health behavior were adjusted for age, sex, and other potentially confounding variables selected a priori: occupational social class, BMI, blood pressure, and the other 3 health behaviors. Analyses using number of poor health behaviors as the exposure variable were adjusted for sex, age, occupational social class, BMI, and blood pressure. We tested for effect modification (on a multiplicative scale) by including an interaction term between the exposure variable and the potential effect modifier. All P values for effect modification by sex were statistically nonsignificant (P > .05 for all comparisons). Given that there was no evidence of significant effect modification by sex, we pooled the data for men and women in the main analyses and adjusted for sex. (Sex-specific data are given in eTables 1 and 2, http://www.archinternmed.com.) Survival curves were made using regression survival function plots. To express the difference in survival of those with 4 poor health behaviors compared with those with none that is equivalent to mortality associated with each yearly increase in age, we divided the β coefficient for mortality in those with a score of 4 compared with no poor health behaviors with the β coefficient for mortality associated with each yearly increase in age. Population attributable risks (PAR) (by percentage) for total, CVD, and cancer mortality by the 4 exposure groups 1, 2, 3, and
4 poor health behaviors were calculated using the method of Mason and Tu\(^2\): \( \text{PAR} = \frac{\left[ \text{Ep} \times (\text{HR}_{i-1}) \right]}{\left[ \text{Ep} \times (\text{HR}_{-1}) \right] + 1} \times 100; i=1, 2, 3, 4 \), and 4 poor health behaviors, \( \text{Ep} \), exposure prevalence.

Combined PAR for all exposure categories was calculated using the formula \( \left[ \text{Ep} \times (\text{HR}_{i-1}) \right] + \text{Ep} \times (\text{HR}_{-1}) + \text{Ep} \times (\text{HR}_{-2}) + \text{Ep} \times (\text{HR}_{-3}) + \text{Ep} \times (\text{HR}_{-4}) + \text{Ep} \times (\text{HR}_{-5}) + \text{Ep} \times (\text{HR}_{-6}) + \text{Ep} \times (\text{HR}_{-7}) \times 100; i=1, j=2, k=3, \) and \( l=4 \) poor health behaviors, \( \text{Ep} \), exposure prevalence, as shown by Hanley.\(^3\)

Complete data on health behaviors were available for 4886 individuals (99.5%) with a combined total of 89,056 person-years of exposure. Table 1 shows the distribution of age and health behaviors among the study members. All behaviors except for physical activity differed between men and women, with men being more likely than women to have unfavorable levels. This sex differential was particularly marked for alcohol consumption.

Table 2 presents the hazard ratios (HRs) (95% confidence intervals [CIs]) for mortality associated with each poor health behavior. In age- and sex-adjusted analyses, each of the 4 health behaviors was associated with an increased risk of total and cause-specific mortality, although statistical significance at conventional levels was not always apparent. There was some variation in the strength of the health behaviors–death relationships across the mortality end points. Smoking was more strongly related to cancer and other deaths, while physical inactivity was more powerfully associated with CVD mortality than the other outcomes. Higher levels of alcohol intake had similar relationships across all mortality outcomes. Results for men and women separately are presented in Table 1. Controlling for occupational social class, BMI, blood pressure, and prior illness led to some attenuation of these effects. The greatest attenuations were seen for physical activity and for fruit and vegetable intake associated with CVD and other deaths and all-cause mortality.

The HRs (95% CIs) for all-cause and cause-specific mortality in relation to poor health behaviors are shown in Table 1. In comparison with people with no poor health behaviors, the risk of mortality for each outcome rose as the number of poor health behaviors increased (\( P \) value for trend, <.001). There was some variation in the strength of these gradients, although all tests for trend were statistically significant. Thus, in the fully adjusted model, the

---

**Table 1. Baseline and Follow-up Mortality Characteristics of 4886 Men and Women 18 Years or Older in 1984-1985**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men (n=2509)</th>
<th>Women (n=2377)</th>
<th>Combined (n=4886)</th>
<th>No. of Deaths/No. of Participants</th>
<th>HR (95% CI) for Total Mortality(^a)</th>
<th>Poor Habit, %(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>44.5 (16.8)</td>
<td>42.9 (15.7)</td>
<td>43.7 (16.3)</td>
<td>1080/4886</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>23.6</td>
<td>42.0</td>
<td>32.6</td>
<td>214/1591</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>Former smoker</td>
<td>26.9</td>
<td>20.9</td>
<td>24.0</td>
<td>369/1171</td>
<td>1.41 (1.18-1.68)</td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>49.5</td>
<td>37.1</td>
<td>43.5</td>
<td>497/2124</td>
<td>1.83 (1.54-2.17)</td>
<td>43.5</td>
</tr>
<tr>
<td>( P ) value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol intake, U/wk, %(^d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>11.4</td>
<td>20.2</td>
<td>15.7</td>
<td>168/767</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>1-2</td>
<td>8.7</td>
<td>20.0</td>
<td>14.2</td>
<td>174/695</td>
<td>1.10 (0.89-1.36)</td>
<td></td>
</tr>
<tr>
<td>3-14</td>
<td>38.1</td>
<td>50.5</td>
<td>41.1</td>
<td>469/2156</td>
<td>0.99 (0.83-1.19)</td>
<td></td>
</tr>
<tr>
<td>15-21</td>
<td>12.0</td>
<td>5.6</td>
<td>8.8</td>
<td>96/432</td>
<td>1.11 (0.86-1.44)</td>
<td></td>
</tr>
<tr>
<td>( \geq 22 )</td>
<td>29.8</td>
<td>3.7</td>
<td>17.1</td>
<td>173/836</td>
<td>1.33 (1.06-1.66)</td>
<td></td>
</tr>
<tr>
<td>( P ) value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity, min/wk, %(^e)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \leq 15 )</td>
<td>56.4</td>
<td>53.3</td>
<td>55.4</td>
<td>852/2706</td>
<td>1.54 (1.09-2.18)</td>
<td></td>
</tr>
<tr>
<td>15-119</td>
<td>8.2</td>
<td>9.9</td>
<td>9.0</td>
<td>85/441</td>
<td>1.47 (0.96-2.25)</td>
<td></td>
</tr>
<tr>
<td>120-179</td>
<td>9.6</td>
<td>12.5</td>
<td>11.0</td>
<td>55/359</td>
<td>1.01 (0.66-1.56)</td>
<td></td>
</tr>
<tr>
<td>180-359</td>
<td>7.6</td>
<td>9.1</td>
<td>8.4</td>
<td>32/408</td>
<td>0.90 (0.55-1.45)</td>
<td></td>
</tr>
<tr>
<td>360-540</td>
<td>9.5</td>
<td>8.2</td>
<td>8.9</td>
<td>49/433</td>
<td>1.06 (0.68-1.64)</td>
<td></td>
</tr>
<tr>
<td>( &gt; 540 )</td>
<td>8.6</td>
<td>6.0</td>
<td>7.3</td>
<td>34/359</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>( P ) value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fruit and vegetable intake, times/d, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( &lt; 1 )</td>
<td>11.4</td>
<td>5.7</td>
<td>8.6</td>
<td>106/421</td>
<td>1.31 (0.78-2.21)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>30.2</td>
<td>22.0</td>
<td>26.2</td>
<td>287/1280</td>
<td>1.10 (0.67-1.80)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>31.2</td>
<td>32.2</td>
<td>31.7</td>
<td>358/1550</td>
<td>1.12 (0.69-1.82)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>21.3</td>
<td>29.7</td>
<td>25.4</td>
<td>255/1240</td>
<td>0.92 (0.56-1.50)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>4.5</td>
<td>8.1</td>
<td>6.2</td>
<td>58/304</td>
<td>0.84 (0.49-1.45)</td>
<td></td>
</tr>
<tr>
<td>( \geq 5 )</td>
<td>1.4</td>
<td>2.4</td>
<td>1.9</td>
<td>17/91</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>( P ) value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; HR, hazard ratio.

\(^a\) Cox regression adjusted for social class, sex, age, body mass index, and blood pressure.

\(^b\) Defined by the cutoff point for use in the analyses for mortality by combined behaviors.

\(^c\) \( P \) value for trend for each behavior in relation to total mortality.

\(^d\) One unit=8 g; poor drinking behavior was defined as consuming more than 21 U/wk for men and more than 14 U/wk for women.

\(^e\) The physical activity score is the sum of score for frequency and score for amount per week, where 1 symbolizes very little activity (\( \leq 1 \times 30 \) min/wk) and 6 symbolizes very high activity (\( > 5 \) times/wk and/or \( > 180 \) min/wk) with 2 to 5 symbolizing the intermediary levels.
HR (95% CI) for 4 poor health behaviors compared with none for all-cause mortality was 3.49 (95% CI, 2.23-5.26), whereas the corresponding effect estimates for CVD, cancer, and other deaths were 3.14 (95% CI, 1.57-6.29), 3.35 (95% CI, 1.67-6.70), and 4.29 (95% CI, 2.01-9.15), respectively. Sex-specific results for all-cause mortality were 3.48 (95% CI, 2.31-5.26), 3.35 (95% CI, 1.67-6.70), and 4.29 (95% CI, 2.01-9.15), respectively. Sex-specific results for all-cause mortality were similar to those for men and women combined (Table 2). The increase in mortality risk from no to 4 poor health behaviors was equivalent to an increase in chronological age of about 12 years: in the fully adjusted regression model, the sex coefficient was 1.255 for 4 compared with 85% for those with 4 poor health behaviors.

We examined the issue of reverse causality—that illness at study induction might have influenced health behaviors and also mortality risk—using 2 methods. First, we excluded participants who had a history of cancer; heart conditions; diabetes mellitus; and respiratory, rheumatic, or gastrointestinal tract diseases at baseline. Second, we excluded deaths that occurred in the first 4 years of follow-up (Table 4). Both these exclusions had little influence on the effect estimates for all-cause, CVD, and cancer mortality by combined health behaviors (Table 5). The Figure shows the combined survival curves over 20 years by the number of poor health behaviors (multiply adjusted). The adjusted cumulative survival was 96% for participants with no poor health behaviors compared with 85% for those with 4 poor health behaviors. The increase in mortality risk from no to 4 poor health behaviors was equivalent to an increase in chronological age of about 12 years: in the fully adjusted regression model, the β coefficient was 1.255 for 4 compared with no poor health behaviors and 0.102 for each subsequence yearly increase in chronological age.

We calculated the PAR for all-cause, CVD, and cancer mortality. The PARs for CVD and cancer mortality by combined health behaviors were 3.14 (95% CI, 1.57-6.29), 3.35 (95% CI, 1.67-6.70), and 4.29 (95% CI, 2.01-9.15), respectively. The PAR for all-cause mortality was 3.49 (95% CI, 2.23-5.26).
In this study, we examined the combined influence of smoking, having a low intake of fruit and vegetables, a high intake of alcohol, and a low level of physical activity on mortality in adults from a United Kingdom–wide population over 20 years of follow-up. The combined effect of these 4 poor health behaviors was associated with significantly higher mortality from all causes, CVD, and cancer and from all other causes, although the magnitude of the effect differed according to outcome. For example, individuals who exhibited all 4 poor health behaviors had about 3 times the risk of CVD and cancer mortality and 4 times the risk of dying from other deaths compared with those exhibiting none of the behaviors.

Table 4. Individual Health Behaviors in Relation to All-Cause, CVD, and Cancer Mortality Risk in 4241 Men and Women and in 3981 Men and Women for Mortality From Other Causes, 18 Years or Older at Baseline in 1984-1985

<table>
<thead>
<tr>
<th>Health Behavior</th>
<th>All-Cause a</th>
<th>CVD a</th>
<th>Cancer a</th>
<th>Other b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=700)</td>
<td>(n=266)</td>
<td>(n=226)</td>
<td>(n=178)</td>
</tr>
<tr>
<td>Current smoking vs never and former smoking</td>
<td>1.72 (1.48-2.00)</td>
<td>1.57 (1.34-1.85)</td>
<td>1.41 (1.10-1.81)</td>
<td>1.28 (0.98-1.66)</td>
</tr>
<tr>
<td>Alcohol intake, &gt;14/21 U/wk vs ≤14/21 U/wk b</td>
<td>1.34 (1.11-1.63)</td>
<td>1.22 (1.00-1.48)</td>
<td>1.42 (1.05-1.93)</td>
<td>1.24 (0.90-1.71)</td>
</tr>
<tr>
<td>Leisure time PA, &lt;2 h/wk vs ≥2 h/d c</td>
<td>1.36 (1.12-1.64)</td>
<td>1.24 (1.02-1.50)</td>
<td>1.27 (0.94-1.73)</td>
<td>1.14 (0.84-1.55)</td>
</tr>
<tr>
<td>Fruit intake, &lt;3 times/d vs ≥3 times/d</td>
<td>1.37 (1.17-1.61)</td>
<td>1.18 (1.00-1.39)</td>
<td>1.46 (1.12-1.90)</td>
<td>1.28 (0.97-1.68)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; PA, physical activity.

a One unit=8 g; poor drinking behavior was defined as consuming more than 21 U/wk for men and more than 14 U/wk for women.

b Also exclusion of prior reports of arthritis, rheumatism, stomach ulcer, and other gastrointestinal tract diseases.

c Analyses are adjusted for age, sex, occupational social class, body mass index, blood pressure, and the other 3 health behaviors.

d One unit=8 g; poor drinking behavior was defined as consuming more than 21 U/wk for men and more than 14 U/wk for women.

Table 5. Collective Health Behaviors in Relation to 20 Years’ All-Cause, CVD, and Cancer Mortality Risk in 4241 Men and Women and in 3981 Men and Women for Mortality From Other Causes, 18 Years or Older at Baseline in 1984-1985

<table>
<thead>
<tr>
<th>No. of Poor Health Behaviors (No.)</th>
<th>All-Cause a</th>
<th>CVD a</th>
<th>Cancer a</th>
<th>Other b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=700)</td>
<td>(n=266)</td>
<td>(n=226)</td>
<td>(n=178)</td>
</tr>
<tr>
<td>0 (361)</td>
<td>26 (7.2)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
</tr>
<tr>
<td>1 (1110)</td>
<td>153 (13.8)</td>
<td>1.87 (1.23-2.64)</td>
<td>1.91 (0.97-3.73)</td>
<td>1.87 (0.92-3.81)</td>
</tr>
<tr>
<td>2 (1546)</td>
<td>264 (17.1)</td>
<td>2.20 (1.47-3.30)</td>
<td>2.20 (1.14-4.22)</td>
<td>2.01 (1.01-4.03)</td>
</tr>
<tr>
<td>3 (957)</td>
<td>192 (20.1)</td>
<td>2.97 (1.96-4.50)</td>
<td>2.70 (1.38-5.29)</td>
<td>2.98 (1.47-6.05)</td>
</tr>
<tr>
<td>4 (267)</td>
<td>65 (24.3)</td>
<td>3.69 (2.31-5.89)</td>
<td>3.09 (1.45-6.63)</td>
<td>3.70 (1.67-8.22)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio.

a Analyses are adjusted for age, sex, occupational social class, body mass index, and blood pressure. P<.001 for all comparisons.

b Also exclusion of prior reports of arthritis, rheumatism, stomach ulcer, and other gastrointestinal tract diseases.

Comment

In this study, we examined the combined influence of smoking, having a low intake of fruit and vegetables, a high intake of alcohol, and a low level of physical activity on mortality in adults from a United Kingdom–wide population over 20 years of follow-up. The combined effect of these 4 poor health behaviors was associated with significantly higher mortality from all causes, CVD, and cancer and from all other causes, although the magnitude of the effect differed according to outcome. For example, individuals who exhibited all 4 poor health behaviors had about 3 times the risk of CVD and cancer mortality and 4 times the risk of dying from other deaths compared with those exhibiting none of the behaviors.

Exclusion of those individuals who may have had some preexisting health condition did not alter these findings. It is possible, however, that excluding only 4 years of follow-up was insufficient to capture diseases with a long latency period (eg, some cancers), and, hence, the possibility of reverse causality remains.

Comparison With Other Studies

Lifestyle factors such as smoking, dietary habits, physical activity, and alcohol consumption have been shown to be independently related to morbidity and mortality in numerous studies, but few studies have investigated the combined impact of these factors. Khaw et al examined the combined impact of not smoking, not being physically inactive, having a moderate alcohol intake, and having a high fruit and vegetable intake on mortality among men and women aged 45 to 79 years followed up...
for about 11 years. The study concluded that there was a strong trend of decreasing mortality risk associated with an increasing number of positive health behaviors, with those who had 4 positive health behaviors having about one-quarter the mortality risk of those who had none. Furthermore, the difference between the highest and lowest health behavior score in that study was equivalent to approximately 14 years in chronological age, similar to the 12-year difference we observed in the current study. Our findings are also consistent with those from a large multicenter European study¹⁸ that examined the combined impact of a Mediterranean diet, being physically active, moderate alcohol use, and not smoking on all-cause and cause-specific mortality among men and women aged 70 to 90 years in 11 European countries, followed up for 10 years. The study concluded that adherence to a Mediterranean diet and healthful lifestyle was associated with more than a 50% lower rate of all-cause and cause-specific mortality.

**METHODOLOGICAL CONSIDERATIONS**

There are several methodological differences between the current study and previous ones that may explain the variation in risk estimates associated with certain risk behaviors. For example, in some studies, only those individuals who had never smoked were defined as “not smoking.”¹⁷,¹⁹ whereas in the current study we classified both former and never smokers as “not smoking.” This is likely to have underestimated the impact of smoking given that former smokers have an increased mortality risk compared with never smokers (although markedly reduced compared with current smokers). For diet, HRs for mortality in some other studies¹⁷,¹⁹ seem to be higher than those reported in the current study, and again this may be attributed to differences in the definitions used to categorize a favorable diet. It is also possible that we have underestimated the importance of diet on subsequent mortality risk by examining only fruit and vegetable intake. Data on other dietary constituents, such as unsaturated fat, fish, and whole grains, that may also have an impact on health outcomes, were not collected. By comparison, the associations between alcohol intake and mortality found here are similar to those found previously.¹⁷,¹⁹ Food frequency questionnaires similar to the one used in our study have been shown to be valid and capable of identifying both low and high consumers of fruit and vegetables²²,²³ and of alcohol.²⁴,²⁵ However, we cannot preclude the possibility that there may have been some misclassification of intakes.

The cutoff point for defining a physically active person differed considerably between our study and previous investigations of the combined impact of health behaviors on mortality. In the current study, the physical activity score was solely based on leisure time exercise, whereas in other studies all types of activity (ie, leisure time; walking; and work-related,¹⁷ household, and gardening activities) were included in the definition of an active life.¹⁸ The cutoff point for defining a physically active person in the current study was low compared with other studies, and below the recommended time spent on physical activities.³⁶ This may have resulted in the higher HRs that we observe for physical activity.

Our method of calculating risk factor scores used in this study has some strengths and limitations. On the one hand, an advantage is that accumulated risk can be quickly and easily calculated by a health profession or patient rather than requiring the use of a more complicated risk algorithm and associated computer software. On the other hand, in not attaching a weight to each poor health behavior, the higher HRs that we observe for physical activity.
risk factor, certain behaviors (eg, smoking) are more powerful risk predictors than others (eg, diet), and we have presented a somewhat oversimplified approach.

**STRENGTHS AND LIMITATIONS**

An advantage of this study is its generalizability, combined with its low loss to follow-up over 20 years, indicating that selection is unlikely to have been a major source of bias. The use of electoral registers to recruit individuals to this study is unlikely to have resulted in any major selection bias because almost all adult citizens of the United Kingdom are registered, although inevitably some individuals, most typically homeless people, will be missed. Additional support for the generalizability of the study findings comes from a study investigating whether nonparticipation in the HALS resurvey in 1992 distorted the exposure-disease relationship. The study concluded that the associations between baseline (1985) risk factors and later CVD mortality were not biased by resurvey (1992) nonresponse. Classification according to lifestyle groups was performed on the basis of measurements of behaviors made at baseline, which may have resulted in misclassification of participants because individuals may have altered their behavior during follow-up. Few studies have investigated tracking of health behaviors over a long follow-up period, but evidence suggests that the degree of stability varies according to the behavior. The stability of physical activity from childhood or adolescence into adulthood is low or moderate, but whether stability increases at older ages is unclear. Dietary factors, such as fruit and vegetable consumption, is moderate to high. Smoking behavior tracks from adolescence into adulthood, whereas tracking of alcohol consumption seems to be lower in adolescence into adulthood, but higher with increasing age at baseline. However, stability of alcohol consumption also depends on type of drinking pattern and type of alcohol. The likelihood of changes in health behaviors among some of our participants during the follow-up time is considerable, depending on age at baseline and type of behavior; however, the hypothesized association between health behaviors at baseline and mortality up to 20 years later assumes some degree of stability of the actual behaviors, and a more modest stability than assumed will most probably lead to an attenuation of the real associations.

An additional limitation of observational analyses examining the relationship between lifestyle and subsequent disease risk is confounding by known and unknown risk factors. For example, socioeconomic status (SES) is a complex variable encompassing a range of measures from education level through to household income, and although we adjusted for social class in the analyses, our measurement of SES may not have adequately captured this information. Therefore, in the present study, the potential for residual confounding cannot be precluded.

Because baseline data collection was performed in 1985, the proportion of the population of the United Kingdom who did not meet the national guidelines regarding physical activity, intake of fruits and vegetables, and smoking has decreased while the proportion exceeding the recommended alcohol intake has remained nearly unchanged for men and increased for women, all of which makes it reasonable to assume that a greater part of the population follows the recommendations today than did 20 years ago. The population-attributable risks calculated in this study, based on the assumptions about a causal link between the risk factors and the outcome (mortality in this study), are dependent on the risk exposure in the population. Because the prevalence of risk factors in the study population have been used as an approximation for the real exposure (because information about the prevalence of the combinations of health behaviors in the entire population is lacking), the calculated PARs might be somewhat overestimated as the exposure level in the study population probably is marginally higher than in the United Kingdom today.

In conclusion, in this contemporary population of the United Kingdom, cigarette smoking, high consumption of alcohol, low consumption of fruits and vegetables, and low levels of physical activity are associated, both independently and when combined, with increased risk of premature mortality. Modest but achievable adjustments to lifestyle behaviors are likely to have a considerable impact at both the individual and population level. Developing more efficacious methods by which to promote healthy diets and lifestyles across the population should be an important priority of public health policy.

Accepted for Publication: October 9, 2009.
Correspondence: Elisabeth Kvaavik, PhD, Department of Nutrition, University of Oslo, PO Box 1046 Blindern, N-0316 Oslo, Norway (ekvaavik@medisin.uio.no).

Author Contributions: Study concept and design: Kvaavik, Batty, and Gale. Analysis and interpretation of data: Kvaavik, Batty, Ursin, Huxley, and Gale. Drafting of the manuscript: Kvaavik, Batty, and Gale. Critical revision of the manuscript for important intellectual content: Kvaavik, Batty, Ursin, Huxley, and Gale. Statistical analysis: Kvaavik, Batty, Ursin, Huxley, and Gale. Administrative, technical, and material support: Kvaavik and Gale.

Financial Disclosure: None reported.

Funding/Support: The Health Promotion Research Trust funded the Health and Lifestyle Survey. Dr Kvaavik is funded by a grant from Norwegian Research Council. The Medical Research Council (MRC) Social and Public Health Sciences Unit receives funding from the MRC and the Chief Scientist Office at the Scottish Government Health Directorates. Dr Batty is a United Kingdom Wellcome Trust Fellow. Dr Huxley is funded by a Career Development Award from the National Heart Foundation of Australia. Catharine Gale is funded by the MRC, United Kingdom.


Additional Contributions: The Office of the Regius Professor of Physic, Cambridge University School of Clinical Medicine, and numerous research workers conducted the study, and the Economic and Social Data Service provided the data.
REFERENCES


screening uptake over baseline rates and did not incorporate a formal cost analysis, we believe that this type of approach has compelling advantages over health plan-based interventions. Within integrated health systems and, eventually, accountable care organizations, these advantages include the ability to mine clinical as opposed to claims data; the harnessing of provider-patient relationships through personalized communication from the health system where patients receive their care; and immediate access to scheduling systems. Because of their intrinsic advantages, we hope private health plans and federal payers will expand value-based contracting and pay-for-performance programs in order to support these types of “in-house” population management programs.

Thomas D. Denberg, MD, PhD
Joel S. Levine, MD

Author Affiliations: Quality and Safety, Harvard Vanguard Medical Associates/Atrius Health, Boston, Massachusetts (Dr Denberg), and Department of Medicine, University of Colorado School of Medicine, Aurora (Dr Levine).

Correspondence: Dr Denberg, University of Colorado School of Medicine, University Internal Medicine, 12631 E 17th Ave, Aurora, CO 80045 (tom.denberg@ucdenver.edu).