Alcohol and Incident Heart Failure Among Middle-Aged and Elderly Men

Cohort of Swedish Men

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**Background**—Compared with no alcohol consumption, heavy alcohol intake is associated with a higher rate of heart failure (HF) whereas light-to-moderate intake may be associated with a lower rate. However, several prior studies did not distinguish between lifelong abstainers and former drinkers, who may have changed alcohol consumption in response to diagnosis. This study aimed to investigate the association between alcohol intake and incident HF.

**Methods and Results**—We conducted a prospective cohort study of 33,760 men aged 45 to 79 years with no HF, diabetes mellitus, or myocardial infarction at baseline participating in the Cohort of Swedish Men Study. We excluded former drinkers. At baseline, participants completed a food frequency questionnaire and reported other characteristics. HF was defined as hospitalization for or death from HF, ascertained by Swedish inpatient and cause-of-death records from January 1, 1998, through December 31, 2011. We constructed Cox proportional hazards models to estimate multivariable-adjusted incidence rate ratios. During follow-up, 2,916 men were hospitalized for (n=2,139) or died (n=777) of incident HF. There was a U-shaped relationship between total alcohol intake and incident HF (P=0.0004). There was a nadir at light-to-moderate alcohol intake: consuming 7 to <14 standard drinks per week was associated with a 19% lower multivariable-adjusted rate of HF compared with never drinking (incidence rate ratio, 0.81; 95% confidence interval, 0.69–0.96).

**Conclusions**—In this cohort of Swedish men, there was a U-shaped relationship between alcohol consumption and HF incidence, with a nadir at light-to-moderate intake. Heavy intake did not seem protective. *(Circ Heart Fail. 2015;8:422–427. DOI: 10.1161/CIRCHEARTFAILURE.114.001787.)*

**Key Words:** alcohol consumption ■ cohort studies ■ epidemiology ■ heart failure

Epidemiological studies have generally found that, compared with no alcohol intake, light-to-moderate consumption is associated with a lower risk of coronary heart disease morbidity and mortality among adults.1,2 Heavy alcohol intake is associated with a higher rate of heart failure (HF), likely via alcohol-associated cardiomyopathy,3,5 but moderate consumption (≤2 drinks per day for men and ≤1 drink per day for women) may be associated with a lower rate of incident HF.

Several prospective cohort studies from the United States have shown that, compared with nondrinkers, light-to-moderate alcohol consumption is associated with a lower rate of incident HF,6–12 with a meta-analysis13 reporting that the rate of incident HF is 15% (95% confidence interval, 7%–22%) lower among those who drink <14 drinks per week than among nondrinkers. However, several prior studies did not distinguish between lifelong abstainers and former drinkers6–9,14 who may have quit because of comorbidities, such as alcoholism or other illness, and may be at higher risk of developing HF. In addition, only 3 of these studies assessed the impact of different beverage types.6,10,12 Therefore, we examined whether incident HF hospitalization or mortality is associated with total alcohol consumption or consumption of beer, wine, or spirits among a large population-based cohort of Swedish men followed for ≤14 years.

**Methods**

**Study Population**

This study consisted of 33,760 men from the Cohort of Swedish Men (COSM), a prospective study of men living in the Västmanland and Örebro counties in central Sweden. The cohort recruitment,
characteristics, and study methods have previously been described.\textsuperscript{18} Starting in autumn 1997, a questionnaire about demographics, behavioral and anthropometric factors, and food and beverage intake was sent to men residing in these counties who were aged 45 to 79 years and 48 880 responded. We excluded participants who did not provide or provided incorrect national identification numbers, who had blank questionnaires, or who had previous diagnosis of cancer (other than nonmelanoma skin cancer; n=2944). In addition, we excluded participants with missing or implausible energy intake (>3 SD from the natural log-transformed mean, n=558); missing data on more than half of the food and beverage items (n=4053); missing data on body mass index (BMI; n=1737), or no information on alcohol consumption (n=132). We excluded former drinkers (n=923) because some of these individuals might have stopped drinking in response to underlying health conditions.

In addition to excluding participants with a history of HF (n=869) at baseline, we excluded participants with a history of myocardial infarction (MI; n=1608) or diabetes mellitus (n=2266) at baseline, because they might have received dietary counseling to change their alcohol intake. HF and MI history were determined via inpatient register linkage; diabetes mellitus history was assessed by self-report and inpatient register linkage. The Regional Ethical Review Board at Karolinska Institute (Stockholm, Sweden) approved the study. Consent was implied by completion and return of the self-administered questionnaire.

### Assessment of Alcohol Intake and Diet

In 1997, participants received a 96-item self-administered food frequency questionnaire (FFQ) asking about food and beverage consumption over the past year.\textsuperscript{18} Men were asked to report if they never drank alcohol, if they had quit drinking alcohol, or if they regularly drank at least some alcohol. For current drinkers, participants reported how frequently they consumed beer (2.8% alcohol), strong beer (4.5% alcohol), wine (18% alcohol), and liquor (40% alcohol) during the past year. For each type of alcohol, there were 9 specified frequency options ranging from never to ≥3x per day. In addition, participants reported how many glasses of light beer (1.8% alcohol) they drank per day or per week during the past year.

The nutrient intake from foods and beverages was calculated by multiplying the frequency of consumption by nutrient composition data from the Swedish National Food Administration by age-specific portion sizes determined using weighted diet records. To estimate beverage-specific alcohol intake (g ethanol/d), we multiplied participant-reported frequency of consumption by alcohol composition data and age-specific drink sizes, as previously described in a similar cohort of Swedish women.\textsuperscript{13} To estimate total alcohol intake (g/d), we then summed over each type of alcohol. We defined 1 serving of alcohol as 13 g of alcohol and estimated the average number of standard alcoholic drinks per week. If a current drinker reported information about consumption of at least 1, but not all, types of alcohol, we assumed he never drank the alcohol type(s) with missing information, as suggested in prior work.\textsuperscript{10}

In a validation study of an FFQ similar to that used in the COSM,\textsuperscript{19} 248 men from the study area randomly selected from the Swedish Population Register completed an FFQ twice 1 year apart and 14 24-hour recall interviews during the year between the first and second FFQ. The Spearman correlation coefficient between the 24-hour recall interviews and the first FFQ estimate of alcohol intake was 0.81.

### Assessment of Other Covariates

We used the inpatient register to obtain information on history of MI at baseline, and we classified participants as having diabetes mellitus at baseline, and we classified participants as having diabetes mellitus because they might have received dietary counseling to change their alcohol intake. HF and MI history were determined via inpatient register linkage; diabetes mellitus history was assessed by self-report and inpatient register linkage. The Regional Ethical Review Board at Karolinska Institute (Stockholm, Sweden) approved the study. Consent was implied by completion and return of the self-administered questionnaire.

### Statistical Analyses

We categorized total alcohol intake as never drinker and into the following categories of standard drinks per week: <0.5, 0.5 to <1, 1 to <7, 7 to <14, 14 to <21, and ≥21. We reported baseline characteristics by category of alcohol intake as mean±SD or as counts with proportions for continuous and categorical characteristics, respectively. We used Cox proportional hazards models to estimate multivariable-adjusted incidence rate ratios with corresponding 95% confidence intervals for the rate of HF incidence among men who consumed alcohol compared with never drinkers. We tested for a quadratic trend by assigning each participant within a category the median drinks per week within that category. We ran a model with this variable and the squared value of this term as continuous predictors and determined the statistical significance of the squared term. We tested for violations of the proportional hazards model by including the product of alcohol intake and the natural log of survival time and did not find evidence of violations.

We chose covariates that we considered to be potential confounders because of their association with both alcohol intake and HF occurrence. In the first model, we allowed the baseline rate to vary by age. In the second model, we additionally adjusted for total energy intake (linear term), education (less than high school, high school, university), BMI (linear term), cigarette smoking (current, past, never), marital status (single, married/living with someone, divorced, widower), family history of MI before age 60 (yes, no), history of hypertension (yes, no), and history of high cholesterol (yes, no). We computed a component score for consistency of diet with the Dietary Approaches to Stop Hypertension (DASH) diet, with higher scores indicative of higher diet quality.\textsuperscript{12,13} Men who were in the highest quintile for fruits, vegetables, nuts and legumes, low-fat dairy, and whole grains received a score of 5, whereas those in the lowest quintile received a score of 1; men in the highest quintile for intake of sodium, sweetened beverages, and red and processed meats received a score of 1. We calculated the overall DASH score as the sum of the component scores.

### Follow-Up andAscertainment of HF

Participants contributed person-time from January 1, 1998, until the earliest of December 31, 2011, date of death due to causes other than HF, date of first HF hospitalization, or HF mortality. If a participant died of a non-HF cause, his follow-up time was censored. We followed patients through record linkage to Swedish inpatient and cause-of-death registers. The inpatient register includes >99% of inpatient care in Sweden.\textsuperscript{21} Hospitalization for or death from HF was defined as code 428 from the International Classification of Diseases, Ninth Edition or codes I50 or I11.0 from the International Classification of Diseases, Tenth Edition. We included the first hospitalizations or deaths with HF as the primary or secondary diagnosis and only the first HF event in the register for each individual. Among individuals with HF as a primary diagnosis (first position) in the Swedish inpatient register, 95% had HF confirmed by medical record review using European Society of Cardiology criteria; among individuals with HF as a secondary (second position) diagnosis, 76% had confirmed HF diagnosis.\textsuperscript{22}
self-reported history of hypertension, history of high cholesterol, or current smoking using likelihood ratio tests. Fourth, because unrecognized illness may influence alcohol consumption at baseline, we conducted a sensitivity analysis excluding the first 2 years of follow-up. Finally, because some of the cases with HF as secondary diagnosis might have been false positives, we conducted analyses where we defined cases of HF only based on a primary diagnosis. We performed statistical analyses using SAS version 9.3 (Cary, NC) and used the stcrreg command in Stata version 12 (Statacorp, College Station, TX) for the competing risks model. For all analyses, 2-sided $P<0.05$ was considered to be statistically significant.

**Results**

Over 14 years of follow-up of 33 760 men in the study, 2916 were hospitalized for HF for the first time ($n=2139$) or died of HF ($n=777$), corresponding to a baseline rate of 67.8 HF cases per 10 000 person-years. Most of the study participants reported drinking 1 to <7 alcoholic drinks per week. Compared with never drinkers, men who reported consuming an average of 7 to <14 drinks per week were on average younger and were more likely to be a current or former smoker and to report history of high cholesterol (Table 1).

There was a statistically significant quadratic trend, supporting a U-shaped relationship between total alcohol intake and HF ($P=0.0004$), with a nadir at light-to-moderate alcohol consumption (Table 2). In the analyses by beverage type, we observed suggestions of U-shaped trends between beverage-specific intake and incident HF (Table 3).

Results were similar when, in sensitivity analyses, we imputed physical activity and BMI and additionally adjusted for hypertension, high cholesterol, family history of MI, and imputed physical activity. Accounting for competing risks did not substantially alter the results. In analyses excluding the first 2 years of follow-up, results did not differ materially.

### Table 1. Age-Standardized Baseline Characteristics of 33 760 Participants in the Cohort of Swedish Men by Average Alcohol Intake

<table>
<thead>
<tr>
<th>Weekly Number of Drinks</th>
<th>Median Drinks/Wk (n=1396)</th>
<th>Never Drinker</th>
<th>&lt;0.5</th>
<th>≥0.5</th>
<th>1 to &lt;7</th>
<th>7 to &lt;14</th>
<th>14 to &lt;21</th>
<th>≥21 (n=630)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>63.0±9.6</td>
<td>60.4±9.5</td>
<td>59.3±9.8</td>
<td>58.2±9.2</td>
<td>57.7±8.7</td>
<td>58.4±9.0</td>
<td>57.8±9.0</td>
<td></td>
</tr>
<tr>
<td>Physical activity, MET-h/d</td>
<td>42.3±5.2</td>
<td>42.5±5.3</td>
<td>42.3±5.1</td>
<td>41.7±4.8</td>
<td>41.1±4.7</td>
<td>40.9±4.9</td>
<td>41.0±5.1</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²†</td>
<td>25.4±3.5</td>
<td>26.1±3.7</td>
<td>25.7±3.3</td>
<td>25.7±3.2</td>
<td>25.5±3.1</td>
<td>25.6±3.1</td>
<td>25.4±3.2</td>
<td></td>
</tr>
<tr>
<td>Energy intake, kcal/d</td>
<td>2950.2±931.0</td>
<td>2752.4±898.2</td>
<td>2724.0±863.2</td>
<td>2663.4±785.5</td>
<td>2774.1±772.4</td>
<td>2954.8±834.0</td>
<td>3304.5±987.9</td>
<td></td>
</tr>
<tr>
<td>Cigarette smoking†</td>
<td>Never</td>
<td>86.2</td>
<td>45.9</td>
<td>46.8</td>
<td>37.7</td>
<td>30.6</td>
<td>25.6</td>
<td>20.1</td>
</tr>
<tr>
<td></td>
<td>Past</td>
<td>7.9</td>
<td>30.3</td>
<td>30.1</td>
<td>38.5</td>
<td>44.8</td>
<td>46.9</td>
<td>42.0</td>
</tr>
<tr>
<td></td>
<td>Current</td>
<td>5.9</td>
<td>23.8</td>
<td>23.1</td>
<td>23.8</td>
<td>24.6</td>
<td>27.6</td>
<td>37.9</td>
</tr>
<tr>
<td>Marital status†</td>
<td>Single</td>
<td>11.3</td>
<td>11.8</td>
<td>8.3</td>
<td>5.7</td>
<td>4.0</td>
<td>3.6</td>
<td>6.6</td>
</tr>
<tr>
<td></td>
<td>Married/living with someone</td>
<td>83.0</td>
<td>77.7</td>
<td>82.2</td>
<td>85.2</td>
<td>87.4</td>
<td>85.6</td>
<td>82.8</td>
</tr>
<tr>
<td></td>
<td>Divorced</td>
<td>3.4</td>
<td>7.8</td>
<td>7.0</td>
<td>6.5</td>
<td>5.9</td>
<td>7.3</td>
<td>8.2</td>
</tr>
<tr>
<td></td>
<td>Widower</td>
<td>2.4</td>
<td>2.8</td>
<td>2.5</td>
<td>2.6</td>
<td>2.8</td>
<td>3.5</td>
<td>2.4</td>
</tr>
<tr>
<td>Education†</td>
<td>Less than high school</td>
<td>68.2</td>
<td>75.4</td>
<td>73.1</td>
<td>68.6</td>
<td>58.9</td>
<td>53.3</td>
<td>56.0</td>
</tr>
<tr>
<td></td>
<td>High school</td>
<td>13.2</td>
<td>10.1</td>
<td>13.9</td>
<td>14.7</td>
<td>18.2</td>
<td>18.8</td>
<td>15.1</td>
</tr>
<tr>
<td></td>
<td>University</td>
<td>18.6</td>
<td>14.5</td>
<td>13.0</td>
<td>16.8</td>
<td>23.0</td>
<td>27.9</td>
<td>28.8</td>
</tr>
<tr>
<td>Family history of MI before age 60†</td>
<td>8.9</td>
<td>13.2</td>
<td>12.1</td>
<td>12.4</td>
<td>11.5</td>
<td>12.3</td>
<td>14.4</td>
<td></td>
</tr>
<tr>
<td>History of hypertension</td>
<td>16.0</td>
<td>21.0</td>
<td>21.1</td>
<td>19.6</td>
<td>18.7</td>
<td>18.9</td>
<td>20.5</td>
<td>20.5</td>
</tr>
<tr>
<td>History of high cholesterol</td>
<td>7.1</td>
<td>9.6</td>
<td>13.0</td>
<td>12.3</td>
<td>12.6</td>
<td>12.6</td>
<td>13.2</td>
<td></td>
</tr>
<tr>
<td>DASH component score‡</td>
<td>Quartile 1</td>
<td>22.5</td>
<td>27.7</td>
<td>25.0</td>
<td>21.6</td>
<td>20.1</td>
<td>23.8</td>
<td>29.6</td>
</tr>
<tr>
<td></td>
<td>Quartile 2</td>
<td>26.5</td>
<td>22.8</td>
<td>25.0</td>
<td>24.6</td>
<td>25.2</td>
<td>24.7</td>
<td>27.2</td>
</tr>
<tr>
<td></td>
<td>Quartile 3</td>
<td>25.2</td>
<td>23.0</td>
<td>24.7</td>
<td>26.8</td>
<td>27.9</td>
<td>27.0</td>
<td>26.7</td>
</tr>
<tr>
<td></td>
<td>Quartile 4</td>
<td>25.8</td>
<td>26.5</td>
<td>25.4</td>
<td>27.0</td>
<td>26.8</td>
<td>24.5</td>
<td>16.5</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or %. BMI indicates body mass index; DASH, Dietary Approaches to Stop Hypertension; and MET, metabolic equivalent of task.

*All values (except for age and median standard drinks per week) are standardized to the age distribution in the study population at baseline.

†Missing data on smoking history (n=367), marital status (n=126), education (n=185), and family history of MI (n=7882).
‡Dietary component score derived from consistency with Dietary Approaches to Stop Hypertension diet.
The association between alcohol intake and HF did not differ by self-reported history of hypertension (interaction=0.70), smoking status (interaction=0.37), or self-reported high cholesterol (interaction=0.08). When we defined HF as those with a primary diagnosis of HF, the results were similar.

Discussion

In this large prospective cohort study, there was a U-shaped relationship between total alcohol intake and incident HF, with a nadir at light-to-moderate alcohol intake. When we examined the association separately for beer, wine, and spirits and mutually adjusted for other beverage types, we found suggestion of a U-shaped trend for beer, wine, and spirits. Although wine and spirits no longer appeared protective above 7 drinks per week, beer appeared potentially protective for 7 to 14 drinks per week. This may be because of drinking patterns; nonbeer drinkers included a higher proportion of never drinkers than did the nonwine or nonspirit drinkers. In addition, men in the highest categories of wine (≥14 drinks per week) or spirits (≥7 drinks per week) consumed fairly high levels of total alcohol, with medians of 26.9 and 20 standard drinks per week, respectively.

There are several potential mechanisms that may explain how light-to-moderate alcohol intake is associated with a lower rate of HF. Moderate alcohol consumption is associated with a lower rate of MI, a key risk factor for HF. In addition, moderate alcohol intake leads to beneficial effects on several coronary artery disease risk factors. Experimental studies have shown that within weeks, moderate alcohol consumption is associated with increased high-density lipoprotein cholesterol, apolipoprotein A-I, and adiponectin and decreased fibrinogen levels. Type 2 diabetes mellitus is a major risk factor for HF. Some observational studies have shown that moderate alcohol consumption is associated with a lower risk of type 2 diabetes mellitus, and some experimental studies have shown that it lowers insulin levels and increases insulin sensitivity.

Our findings are consistent with many but not all of the prior studies showing that moderate alcohol intake is associated with a lower rate of incident HF. For example, in a cohort of people aged ≥65 years, participants who reported drinking 21 to 70 ounces of alcohol per day (≈1.5–4 drinks) had a 47% lower rate of incident HF compared with nondrinkers (incidence rate ratio, 0.53; 95% confidence interval, 0.32–0.88). Some prior studies did not have information about whether nondrinkers were lifelong abstainers or former drinkers. Therefore, their results may have overestimated the protective benefits of alcohol because some former drinkers might have quit because of comorbidities, such as alcoholism and other illness. Similar to our study, a recent meta-analysis of alcohol and incident HF also found a nonlinear dose–response relationship between alcohol and incident HF.
In 1 of the studies that examined the association between incident and HF and beer, wine, or spirits, the results were similar for all alcoholic beverage types.6 In another study, Klatsky et al10 examined the association between alcohol and HF risk for those with and without known coronary artery disease at the time of HF. Among those without known coronary artery disease, a higher frequency of wine consumption was associated with a lower rate of HF among men but there was no benefit of a higher frequency of consuming beer or liquor. In our study, there was a suggestion of protective benefit of light intake of all alcohol types.

Our study has several limitations. First, although we adjusted for multiple potential confounders, we cannot rule out the possibility of residual or unmeasured confounding. For instance, we were only able to adjust for smoking status as never, former, or current smoker, and we did not have information on pack years or years since quitting. However, because smokers tend to drink more alcohol22 and they are at a higher risk of HF,23 confounding by smoking would make alcohol seems less protective. This is in agreement with our results; light-to-moderate alcohol intake appeared more protective in the model that adjusted for smoking than in the age-adjusted model. Second, we used an FFQ that is reproducible and valid,19 but some individuals tend to under-report alcohol consumption and heavier drinkers are more likely to underreport drinking than are lighter drinkers. Third, most of the participants reported no or moderate consumption, thereby limiting our ability to assess the impact of heavy consumption of incident HF risk. Fourth, although the accuracy of HF diagnosis in Swedish registers has previously been shown to be high,23 these registers only include HF cases that were hospitalized or died, so these findings might not be applicable to less severe cases. Furthermore, these registers do not include information about HF subtype (eg, systolic or diastolic dysfunction) or pathogenesis.

Our study also has several strengths. Because this is a large, population-based prospective cohort study, concerns about recall bias are minimized. In addition, we were able to exclude former drinkers who may have altered their alcohol consumption in response to underlying health conditions. Finally, the FFQ includes detailed information on consumption of beer, wine, and spirits and is reproducible and valid.19

In summary, in this large prospective cohort study of middle-aged and elderly Swedish men, there was a U-shaped relationship between alcohol consumption and incident HF. Light-to-moderate, but not heavy, alcohol intake was associated with a lower rate of HF hospitalization or death. Because moderate alcohol consumption also carries potential risks, such as an increased risk of cancer25 and injury,19 public health messages about the potential cardioprotective consequences of alcohol consumption should take into consideration both risks and benefits. Additional work is necessary to examine whether drinking patterns influence the relationship between alcohol consumption and incident HF.

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Disclosures
None.

References


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**CLINICAL PERSPECTIVE**

Heavy alcohol intake is associated with a higher rate of heart failure (HF). Prior observational studies have generally found that light-to-moderate intake is associated with a lower rate of HF. However, several of these studies did not exclude former drinkers, who may have changed their alcohol consumption in response to a diagnosis. Therefore, we evaluated the association between alcohol consumption and incident HF among 33,760 men aged 45 to 79 enrolled in the Cohort of Swedish Men. We excluded former drinkers and participants with history of HF, diabetes mellitus, or myocardial infarction at baseline. We saw a U-shaped relationship between alcohol consumption and incidence of HF, with a nadir at light-to-moderate alcohol intake. Although alcohol consumption is known to carry health risks, our study shows that light-to-moderate consumption may be protective against HF. Heavy consumption was not protective.
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초록

배경
알코올을 섭취하지 않는 경우와 비교하여 적거나 보통(light-to-moderate) 정도로 알코올을 섭취하는 경우에는 심부전의 발생률이 낮아질 수 있는 반면, 많은(heavy) 알코올을 섭취하는 경우에는 심부전의 발생률이 높아진다. 그러나 기존의 몇몇 연구들은 과거에만 음주를 한 사람(former drinker)을 배제하지 않았는데, 이들은 진단에 따라 음주 습관을 바꿨을 가능성이 있다. 본 연구는 알코올 섭취와 심부전 발생 간의 상관관계를 밝히고자 하였다.

방법 및 결과
스웨덴 남성 코호트에서 기존에 심부전, 당뇨병, 또는 심근 경색이 없는 45-79세의 남성 33,760명을 대상으로 전향적 코호트 연구를 수행하였다. 이때 과거에만 음주를 하였던 사람은 배제하였다. 대상자들은 연구 시작 시에 음식 빈도 설문지(food frequency questionnaire)를 작성하고, 다른 특성들에 대해 보고하였다. 심부전은 심부전에 의한 입원 및 사망으로 정의되었는데, 이는 1998년 1월 1일부터 2011년 12월 31일까지의 스웨덴 입원 환자와 사망원인 기록에 의해서 확인되었다. 그리고 Cox 비례위험 모델을 사용하여 다변수 조정된 발생률 비율을 추산하였다. 추적 기간 동안 2,916명에서 심부전이 발생하여 입원(2,139명)하거나 사망(777명)하였다. 총 알코올 섭취량과 심부전의 발생간에는 U-형의 상관관계를 보였다(P=0.0004). 적거나 보통 정도의 알코올 섭취가 U-형의 바닥에 해당하였는데, 1주에 7 이상 14 미만의 표준 음주를 하는 경우에는 전혀음주를 하지 않는 경우에 비해 19%의 낮은 다변수 보정 심부전 발생률을 보였다(발생률 비율, 0.81; 95% CI, 0.69–0.96).

결론
본 스웨덴 남성 코호트에서 알코올 섭취량과 심부전의 발생간에는 U-형의 상관관계가 있었고, 적거나 보통 정도의 알코올 섭취가 U-형의 바닥에 해당하였다. 많은 알코올을 섭취하는 경우에는 심부전 보호 효과가 없는 듯하였다.