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 exclude 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, 2916 men were hospitalized for (n=2139) 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.

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.1–5 Heavy alcohol intake is associated with a higher rate of heart failure (HF), likely via alcohol-associated cardiomyopathy,1,5 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.

Clinical Perspective on p 427

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 drinkers,6–8,14 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.15
Starting in autumn 1997, a questionnaire about demographics, behav-
ioral and anthropometric factors, and food and beverage intake was
sent to men residing in these counties who were aged 45 to 79 years
and 48,850 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 par-
ticipants 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 consump-
tion (n=132). We excluded former drinkers (n=923) because some of
these individuals might have stopped drinking in response to underly-
ing 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 reg-
ister 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-adminis-
tered questionnaire.
Assessment of Alcohol Intake and Diet
In 1997, participants received a 96-item self-administered food fre-
cuity questionnaire (FFQ) asking about food and beverage con-
sumption over the past year.16 Men were asked to report if they never
drank alcohol, if they had quit drinking alcohol, or if they regularly
drink at least some alcohol. For current drinkers, participants report-
ed how frequently they consumed beer (2.8% alcohol), strong beer
(4.5% alcohol), wine (≥10% alcohol), fortified wine (>18% alcohol), and liquor (40% alcohol) during the past year. For each type of alcohol, there were 9 prespecified frequency options ranging from never to ≥3× 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 partici-
ient-reported frequency of consumption by alcohol composition data
and age-specific drink sizes, as previously described in a similar co-
hort of Swedish women.17 To estimate total alcohol intake (g/d), we
then summed over each type of alcohol. We defined 1 serving of alco-
hol 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 as-
sumed he never drank the alcohol type(s) with missing information,
as suggested in prior work.18
In a validation study of an FFQ similar to that used in the COSM,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 re-
call 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 via either self-report or a diagnosis of diabetes mellitus
recorded on the inpatient register. We computed total physical ac-
tivity in metabolic equivalents in hours per day based on questions
about occupational physical activity, exercise, and sedentary behav-
or.20 We calculated BMI as weight in kilograms divided by height
in meters squared (both self-reported). The questionnaire also included
questions about education (less than high school, high school, univer-
sity), cigarette smoking (current, past, never), marital status (single,
marricd/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.21 Men who were in the highest quintile for fruits, veg-
atables, 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 beverag-
es, 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 and Ascertainment 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
and 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 pri-
mary 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.22

Statistical Analyses
We categorized total alcohol intake as never drinker and into the fol-
lowing 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 pro-
portions 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 al-
cohol 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 viola-
tions 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 occur-
rence. 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, universi-
ty), BMI (linear term), cigarette smoking (current, past, never), mar-
ital status (single, married/living with someone, divorced, widower),
and component score for consistency with DASH diet (quartiles).16
In secondary analyses, we evaluated the association between HF
incidence and each alcoholic beverage type and mutually adjusted for
the 2 other beverage types. The referent group for each beverage type
was nondrinker of that specific beverage. We conducted tests for qua-
dratic trends to examine the statistical significance of the pattern of
association for each alcohol type and HF risk. We performed several
sensitivity analyses. First, we conducted an analysis using Markov
chain Monte Carlo–imputed data for BMI (n=1415, 4.0%) and physi-
cal activity (n=6907, 19.6%)7,24 and evaluated whether further adjust-
ing for physical activity, family history of MI before age 60, history
of hypertension, and history of high cholesterol would alter results.
Second, we estimated the fully adjusted model of total alcohol in-
take using a proportional substitution hazards model to account for
competing risks of non-HF death.23 Third, we tested whether the
association between alcohol consumption and HF differed by
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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 1). In the analyses by beverage type, we observed suggestions of U-shaped trends between beverage-specific intake and incident HF (Table 2).

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.
The association between alcohol intake and HF did not differ by self-reported history of hypertension ($P$ interaction=0.70), smoking status ($P$ interaction=0.37), or self-reported high cholesterol ($P$ interaction=0.08). When we defined HF as those cases 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 ($\geq$14 drinks per week) or spirits ($\geq$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 $\geq$65 years, participants who reported drinking 21 to 70 ounces of alcohol per day ($\approx$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 intake and HF.

### Table 2

<table>
<thead>
<tr>
<th>Alcohol Drinks/Wk</th>
<th>Cases</th>
<th>Person-Years</th>
<th>Model 1*</th>
<th>Model 2†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>192</td>
<td>17,038</td>
<td>1.00 (reference)</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>192</td>
<td>18,821</td>
<td>1.21 (0.99–1.48)</td>
<td>1.04 (0.85–1.28)</td>
</tr>
<tr>
<td>0.5 to &lt;1</td>
<td>157</td>
<td>21,049</td>
<td>0.94 (0.76–1.17)</td>
<td>0.86 (0.70–1.07)</td>
</tr>
<tr>
<td>1 to &lt;7</td>
<td>1,532</td>
<td>230,285</td>
<td>0.98 (0.84–1.14)</td>
<td>0.89 (0.76–1.04)</td>
</tr>
<tr>
<td>7 to &lt;14</td>
<td>628</td>
<td>111,970</td>
<td>0.89 (0.76–1.05)</td>
<td>0.81 (0.69–0.96)</td>
</tr>
<tr>
<td>14 to &lt;21</td>
<td>141</td>
<td>23,334</td>
<td>0.91 (0.73–1.13)</td>
<td>0.81 (0.65–1.02)</td>
</tr>
<tr>
<td>$\geq$21</td>
<td>74</td>
<td>7,710</td>
<td>1.30 (1.00–1.71)</td>
<td>1.12 (0.85–1.47)</td>
</tr>
</tbody>
</table>

P for quadratic trend

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Total events=2,916.

* Cox proportional hazards model accounting for age.

† In addition, adjusted for total energy intake (linear term), education (less than high school, high school, university), body mass index (linear term), dietary component score (quartiles), cigarette smoking (current, past, never), and marital status (single, married/living with someone, divorced, widower).

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### Table 3

<table>
<thead>
<tr>
<th>Drinks/Wk</th>
<th>Cases</th>
<th>Person-Years</th>
<th>Rate Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonbeer drinker*</td>
<td>352</td>
<td>31,689</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>287</td>
<td>36,264</td>
<td>1.02 (0.87–1.20)</td>
</tr>
<tr>
<td>0.5 to &lt;1</td>
<td>312</td>
<td>43,735</td>
<td>0.91 (0.78–1.07)</td>
</tr>
<tr>
<td>1 to &lt;7</td>
<td>1,620</td>
<td>263,110</td>
<td>0.90 (0.79–1.03)</td>
</tr>
<tr>
<td>7 to &lt;14</td>
<td>284</td>
<td>47,137</td>
<td>0.87 (0.73–1.03)</td>
</tr>
<tr>
<td>$\geq$14†</td>
<td>61</td>
<td>8,273</td>
<td>0.92 (0.70–1.22)</td>
</tr>
</tbody>
</table>

P for quadratic trend

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Wine

<table>
<thead>
<tr>
<th>Drinks/Wk</th>
<th>Cases</th>
<th>Person-Years</th>
<th>Rate Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonwine drinker*</td>
<td>942</td>
<td>99,975</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>728</td>
<td>110,193</td>
<td>0.92 (0.83–1.03)</td>
</tr>
<tr>
<td>0.5 to &lt;1</td>
<td>263</td>
<td>13,257</td>
<td>0.97 (0.84–1.13)</td>
</tr>
<tr>
<td>1 to &lt;7</td>
<td>881</td>
<td>192,636</td>
<td>0.89 (0.80–1.00)</td>
</tr>
<tr>
<td>7 to &lt;14</td>
<td>88</td>
<td>12,693</td>
<td>0.99 (0.79–1.25)</td>
</tr>
<tr>
<td>$\geq$14†</td>
<td>14</td>
<td>14,545</td>
<td>1.16 (0.68–1.98)</td>
</tr>
</tbody>
</table>

P for quadratic trend

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Spirits

<table>
<thead>
<tr>
<th>Drinks/Wk</th>
<th>Cases</th>
<th>Person-Years</th>
<th>Rate Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonspirits drinker*</td>
<td>770</td>
<td>83,708</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>824</td>
<td>143,347</td>
<td>0.90 (0.81–1.01)</td>
</tr>
<tr>
<td>0.5 to &lt;1</td>
<td>374</td>
<td>52,895</td>
<td>0.97 (0.84–1.11)</td>
</tr>
<tr>
<td>1 to &lt;7</td>
<td>891</td>
<td>146,424</td>
<td>0.89 (0.80–1.01)</td>
</tr>
<tr>
<td>$\geq$7†</td>
<td>57</td>
<td>3834</td>
<td>1.27 (0.96–1.69)</td>
</tr>
</tbody>
</table>

P for quadratic trend

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* Cox proportional hazards model accounting for age and adjusted for total energy intake (linear term), education (less than high school, high school, university), body mass index (linear term), dietary component score (quartiles), cigarette smoking (current, past, never), and marital status (single, married/living with someone, divorced, widower). Each model also mutually adjusted for consumption of other 2 beverage types. CI indicates confidence interval.

† Among those consuming: $\geq$14 standard drinks of beer per week, median weekly standard drinks of total alcohol: 21.2; of beer: 17.3; $\geq$14 standard drinks of wine per week, median weekly standard drinks of total alcohol: 26.9, of wine: 16.2; and $\geq$7 standard drinks of spirits per week, median weekly standard drinks of total alcohol: 20.0, of spirits: 9.6.

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In 1 of the studies that examined the association between incident HF and beer, wine, or spirits, the results were similar for all alcoholic beverage types. In another study, Klatsky et al 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 alcohol and they are at a higher risk of HF, confounding by smoking would make alcohol seem 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, but some individuals tend to under-report alcohol consumption and heavier drinkers are more likely to under-report 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, 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.

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 cancer and injury, 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

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 경우는 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 심근경색이 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 환자 및 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 경우는 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.