ADDICTION
SSA SOCIETY FOR THE STUDY OF ADDICTION
RESEARCH REPORT
doi:10.1111/add.15070

Socio-economic differences in all-cause mortality in people with alcohol use disorder: a prospective cohort study

Ingeborg Rossow[1], Ellen J. Amundsen[1] & Sven Ove Samuelsen[2,3]

Department of Alcohol, Tobacco and Drugs, Norwegian Institute of Public Health, Oslo, Norway,[1] Department of Mathematics, University of Oslo, Oslo, Norway[2] and Department of Chronic Diseases and Ageing, Norwegian Institute of Public Health, Oslo, Norway[3]

ABSTRACT

Background and aims While alcohol-attributable mortality rates are higher in low socio-economic status (SES) groups, less is known about SES differences in all-cause mortality in alcohol use disorder (AUD). The aim of this study was to explore whether there are SES differences in people with AUD, regarding (i) treatment admission, (ii) all-cause mortality risk and (iii) relative mortality risk. **Design and setting** A prospective cohort study in Norway, follow-up period from 2009–10 to 2013. Data on SES and mortality were obtained through linkages to national registries, using national unique ID numbers. **Participants** AUD patients (age 20+) admitted to treatment in 2009–10 (n = 11 726) and age and gender frequency-matched controls from the general population (n = 12 055). **Measurements** The SES indicator was education level (low, intermediate and high). Mortality was calculated as deaths per 1000 person-years during the 4-year observation period. **Findings** Admission to AUD treatment was elevated in the low compared with the high SES categories (OR = 3.31, 95% CI = 3.09, 3.55). Among AUD patients, mortality risk was elevated in the low SES category (HR = 1.23, 95% CI = 1.04, 1.45). Relative mortality risk from AUD was significantly higher in the high SES (HR = 8.65, 95% CI = 6.16, 12.14) compared with the low SES categories (HR = 3.29, 95% CI = 2.61, 4.15). **Conclusion** Admission to treatment for alcohol use disorders in Norway appears to decrease with increasing socio-economic status, and relative mortality risk from alcohol use disorder appears to increase with increasing socio-economic status.

Keywords Alcohol use disorder, mortality, Norway, prospective cohort, register linkage, socio-economic status, treatment.

Correspondence to: Ingeborg Rossow, Department of Alcohol, Tobacco and Drugs, Norwegian Institute of Public Health, POB 222 Skøyen, 0213 Oslo, Norway.
E-mail: Ingeborg.Rossow@fhi.no
Submitted 17 October 2019; initial review completed 8 January 2020; final version accepted 1 April 2020

INTRODUCTION

Alcohol consumption is among the most important risk factors for premature deaths and disabilities [1]. Overall consumption and drinking patterns are the two important aspects of alcohol's role in disease and injury, and thus the risk of premature death generally tends to increase with increasing overall consumption and with increasingly harmful drinking patterns [2]. This fairly simple exposure–outcome association, however, does not seem to apply equally across socio-economic status (SES) groups. While overall consumption tends to increase with SES [3], it is well demonstrated that alcohol-attributable deaths, which are often the end-points of heavy drinking careers, is elevated in lower SES groups [4]. Thus, it seems that alcohol disproportionately impacts alcohol harms in deprived individuals [5].

The elevated mortality in low SES groups is attenuated for alcohol-attributable causes of death [6,7], which are mainly seen in people with alcohol use disorders (AUD). In a meta-analysis, Probst and co-workers [6] found that the socio-economic differential in mortality risk was, on average, 1.7 times higher for alcohol-attributable causes compared to all-cause mortality. Consequently, a substantial proportion of social inequality in premature mortality is probably attributable to long-term heavy drinking and AUD.

So far, the literature on social differences in alcohol-related mortality is mainly devoted to alcohol-attributable mortality. However, deaths from alcohol-attributable causes constitute only a small fraction of all deaths: approximately 2–3% in the general population [7] and some 5–25% among heavy drinkers [8–10]. Thus, the assumption that heavy drinkers in low SES groups are more vulnerable and at higher risk of premature mortality than those in high SES groups should preferably be tested with respect to all-cause mortality.

The risk of premature mortality is higher among heavy drinkers compared to the general population, on average in the order of three- to five-fold higher [11], and thus life expectancy in AUD patients is found to be 24–28 years shorter than in the general population [12]. Relative mortality risk is higher among women than among men with AUD, and particularly high in younger people [11]. To this end, recent reviews of SES differences in alcohol-attributable morbidity and mortality [3,6,13] and of mortality among those with AUD [11] do not provide further insight into the possible SES differential in all-cause mortality among those with AUD.

In order to identify relevant literature in this respect, we reviewed the primary studies identified in Roerecke & Rehm's [11] systematic review of all-cause mortality in people with AUD, as well as relevant studies identified in reference lists and citations from these primary studies. From our literature search, it seems that few studies examined SES differences in relative mortality risk, and the studies were mainly dated and reported mixed findings. Two studies [14,15] reported no SES difference, one study [15] reported lower relative mortality risk in high SES compared to medium/low SES among females and five studies [8,9,16–18] reported increasing relative mortality risk with increasing SES. Important limitations of these studies included the lack of SES-specific denominator [8,14–17], male samples only [9,18] and little statistical power [18]. Consequently, the research literature seems sparse and inconclusive with regard to a possible SES differential in all-cause mortality in people with AUD, and even more so with respect to relative mortality risk.

Most mortality studies of heavy drinkers appear to rely on samples from AUD or similar treatments [11], which is also the case in the present study. However, interpretation of the social differences in mortality and relative mortality risk in heavy drinkers imply considerations concerning whether these reflect social differences in AUD morbidity, in survival or both [19]. Hence, in this context it is essential to assess whether AUD prevalence varies with SES. There are indications that this may well be the case. Two studies reported a higher risk of AUD in low SES groups [10,20] and several studies reported elevated risk of alcohol-attributable diseases in low SES groups [19,21–24]. Thus, while previous studies may suggest that the risk of AUD is elevated in low SES groups, direct assessments of SES differences in AUD treatment patients seem to be very different.

Against this backdrop, this study aimed at exploring whether there are socio-economic differences in people with alcohol use disorders regarding: (i) treatment admission; (ii) all-cause mortality risk; and (iii) relative mortality risk. More specifically, we also aimed at exploring these social differences in gender- and age-specific strata.

METHODS

Design and study participants

The three study aims required different study designs. We were, however, able to use the same data set for all aims, although with different perspectives. For study aim (i) (SES differences in AUD treatment admission) we applied a frequency-matched case–control design. For study aim (ii) (SES differences in mortality risk among people with AUD) we applied a prospective cohort design, while for study aim (iii) (SES differences in relative mortality risk in people with AUD) we applied a matched prospective cohort design. The data stemmed from a prospective cohort study of patients treated for alcohol use disorders (AUD patients) and a frequency-matched control population. The study was approved by the Regional Committees for Medical and Health Research Ethics in November 2014.

AUD patients were all adult patients admitted during a 2-year period to specialized alcohol treatment or psychiatric hospital with alcohol use disorder. We obtained data on these patients from the Norwegian Patient Registry (NPR), which covers the entire population of patients in the publicly financed specialist health-care system in Norway. Under this public health-care scheme, both public and private providers deliver AUD treatment. There is no information concerning the presumably small fraction of AUD patients in privately funded treatment. The current sample of patients encompasses all those who were admitted between 1 January 2009 and 31 December 2010 to treatment for substance use with alcohol-related disorders as the principal diagnosis; ICD-10 codes F10 and aged 20 years and over (n = 12 432).

A sample of controls was drawn from the Norwegian Population Registry on 1 January 2010. This sample was frequency-matched with the AUD patients by birth year and gender (n = 12 364, after removal of 66 people who were also among the AUD patients). Due to missing observations on the SES measure (n = 592) and person-years under observation (n = 423), the final computed samples consisted of 11 726 AUD patients and 12 055 controls, totalling 23 781 people. The standardized mean differences for age and gender were 0.0024 and 0.0044, respectively, suggesting successful matching.

Using national ID numbers, we obtained information on education, emigration and death for all cohort members through linkage to other national administrative registers. In Norway, the ID number is a unique 11-digit number, containing information on date of birth and gender, and allowing individual-level data from various registers to be linked.

Measures

SES

We considered SES to be a background characteristic and therefore employed information on highest completed education level as the SES indicator, as this is less likely to be affected by heavy drinking over time compared to occupation status and income. Information on completed education level as per 2009 was retrieved from the National Education Database (NUDB) and collapsed into a three-category ordinal variable; 'low' = only completion of mandatory education (i.e. 7, 9 or 10 years, depending on birth cohort), 'high' = completed college or university education and 'intermediate' = all education levels between 'low' and 'high'. Those with missing information on education were mainly immigrants (75%), and were evenly distributed between AUD patients and controls. Data on whether study participants were immigrants or not were obtained from Statistics Norway.

Mortality

Whether study participants had died during the observation period – and if so, their date of death – was obtained from the Cause of Death Registry. Mortality was calculated as number of deaths per 1000 person-years.

Person-years under observation

Number of days from study entry (date of treatment admission for AUD patients and 1 January 2010 for controls) to date of censoring (31 December 2013 or date of death or emigration) were converted to person-years. For some AUD patients (n = 423) we had data on the year of treatment but not the exact date of treatment admission, and person-years under observation were not calculated for these. Date of emigration was obtained from population data at Statistics Norway.

Analyses

First, we explored whether AUD treatment admission was associated with SES, employing logistic regression models. In this model, high SES was the reference category, and we adjusted for age (10-year age groups), gender and the interaction between them. For our estimations of absolute and relative mortality risk in AUD patients by SES, we calculated absolute risks as number of deaths per 1000 person-years and used Cox proportional hazards models to obtain mortality hazard ratios (HR). In the first analyses the focus was on absolute mortality risk, comparing the mortality among AUD patients in different SES categories, both in terms of crude mortality incidence rates and adjusted mortality hazard ratios with high SES as reference category. In the second analysis, we estimated the relative mortality risk in people with AUD, i.e. mortality among AUD patients compared to mortality in the control population, by SES categories. In the Cox regression models, we also adjusted for age, gender and the interaction between them, and the proportionality assumptions in Cox regression was tested using Schoenfeld residuals. Odds ratios (ORs) or hazard ratios (HRs) with non-overlapping 95% confidence intervals (CIs) were considered statistically significantly different [25]. In addition, differences in HR with overlapping CIs were tested by t-test. Furthermore, we carried out stratified analyses by gender and age group. Stata SE version 15.0 was used for the analysis.

We did not pre-register the analysis, and the results should be considered exploratory.

RESULTS

Table 1 presents distributions of gender, age, education level, immigrant background and number of deaths and person-years under observation for AUD patients and controls. The study cohort participants (n = 23 781) were, on average, observed during a period of almost 4 years, and the total number of person-years under observation amounted to 94 681. Among the AUD patients age at admission differed by SES categories, and the mean age at admission was 45, 50 and 51 years in the low, intermediate and high SES categories, respectively.

Table 2 shows that risk of entering AUD treatment differed by SES categories compared to the general population. The estimated adjusted OR increased with decreasing SES; compared to those with high SES, adjusted ORs were 1.67 (CI = 1.57, 1.78) and 3.31 (CI = 3.09, 3.28) for those with intermediate and low SES, respectively. Hence, there was a clear negative social gradient in AUD treatment admissions. We further explored SES differences in AUD treatment admission in age- and gender-specific strata. For both men and women, ORs increased statistically significantly with decreasing SES, and this was also the case among those under 60 years of age. Among those aged 60 years and older, OR was higher in the low—compared to the high—SES category, whereas there was no statistically significant difference between the intermediate and high SES categories (Table 2). Adjusted OR was higher in the low SES category (adjusted OR = 1.19, 95% CI = 1.03, 1.37) compared to the intermediate and high SES categories in combination (reference).

Table 1 Sample characteristics of AUD patients and controls.

	AUD patients (n = 11 726)	*Controls (n = 12 055)*
Proportion women, per cent	29.6 (3474)	29.6 (3568)
Age group distribution, per cent		
20–29 years	10.0 (1172)	9.7 (1166)
30–39 years	14.0 (1645)	14.0 (1693)
40–49 years	27.0 (3170)	27.3 (3293)
50–59 years	31.4 (3686)	31.5 (3795)
60–69 years	14.6 (1714)	14.6 (1755)
70–89 years	2.9 (339)	2.9 (353)
Education level group, per cent		
Low	39.7 (4658)	21.7 (2615)
Medium	40.4 (4731)	43.0 (5186)
High	19.9 (2337)	35.3 (4254)
Proportion immigrants, per cent	5.0 (588)	9.8 (1185)
Person years under observation (mean)	4.028	3.942
Proportion dead, per cent	9.4 (1101)	1.8 (221)

Table 2 Risk of AUD treatment admission by SES category for total cohort and for strata by gender and age group.

	SES categories		
Adjusted odds ratio[a] (95% CI)	*Low SES*	*Intermediate SES*	*High SES*
All	3.31 (3.09, 3.55) $P < 0.001$	1.67 (1.57, 1.78) $P < 0.001$	1.00 (ref)
Gender-specific odds ratio of AUD treatment by SES[b]			
Women	2.89 (2.55, 3.28) $P < 0.001$	1.53 (1.37, 1.72) $P < 0.001$	1.00 (ref)
Men	3.52 (3.24, 3.84) $P < 0.001$	1.74 (1.61. 1.88) $P < 0.001$	1.00 (ref)
Age-specific odds ratio of AUD treatment by SES[a]			
< 60 years	4.03 (3.72, 4.35) $P < 0.001$	1.84 (1.72, 1.98) $P < 0.001$	1.00 (ref)
60 + years	1.24 (1.05, 1.47) $P = 0.013$	1.07 (0.92, 1.23) $P = 0.391$	1.00 (ref)

[a]Adjusted for age in 10-year age groups, gender and the interaction between age and gender; [b]adjusted for age in 10-year age groups. AUD = alcohol use disorder; SES = socio-economic status; CI = confidence interval.

During the observation period, 1101 of the AUD patients died. Among AUD patients, crude mortality incidence was 23.3 per 1000 person-years. Adjusted all-cause mortality HR was somewhat higher in the low compared to the high SES categories; HR = 1.23 (95% CI = 1.04, 1.45). In the intermediate SES category, mortality risk did not differ from that in the high SES category (reference) (adjusted HR = 1.04, 95% CI = 0.99, 1.22) (Table 3). In both gender-specific strata, mortality risk did not differ by SES category. This was also the case among those aged under 60 years. However, among those aged 60 years and over, mortality risk was statistically significantly elevated in the low, compared to the high, SES categories (adjusted HR = 1.44, CI = 1.10, 1.89) (Table 3).

As shown in Table 4, mortality incidence (per 1000 person-years) was substantially elevated in AUD patients compared to controls (adjusted HR = 5.08, CI = 4.40, 5.87). While we observed somewhat modest SES differences in mortality incidence among AUD patients, mortality incidence among controls was more than three times higher in the low compared to the high SES categories (8.7 versus 2.4). In effect, the elevated mortality risk among AUD patients compared to controls (relative mortality risk) was higher in the high SES (adjusted HR = 8.65, CI = 6.16, 12.14) compared to the low SES categories (adjusted HR = 3.29, CI = 2.61, 4.15) (Table 4). In the intermediate SES category, relative mortality risk (adjusted RH = 5.25, CI = 4.18, 6.60) was higher compared to the low SES category and lower compared to the high SES category ($t = 2.10$, $P = 0.036$), and thus relative mortality risk increased with SES. Within gender-specific strata, a higher relative mortality risk was observed in the high compared to the low SES categories, and this

Table 3 All-cause mortality incidence and mortality hazard ratio by SES categories among AUD patients.

	SES categories		
	Low SES	*Intermediate SES*	*High SES*
Deaths	422	458	221
Person years	18 696	19 074	9397
Crude mortality incidence per 1000 person-years	22.6	24.0	23.5
Adjusted mortality hazard ratio[a]	1.23 (1.04, 1.45) $P = 0.015$	1.04 (0.88, 1.22) $P = 0.637$	1.00 (ref)
All			
Gender-specific mortality hazard ratio[b]			
Women	1.39 (0.99, 1.96) $P = 0.057$	1.04 (0.74, 1.45) $P = 0.839$	1.00 (ref)
Men	1.18 (0.98, 1.43) $P = 0.081$	1.04 (0.86, 1.25) $P = 0.696$	1.00 (ref)
Age-specific mortality hazard ratio[a]			
< 60 years	1.11 (0.90, 1.36) $P = 0.338$	0.94 (0.76, 1.15) $P = 0.534$	1.00 (ref)
60+ years[c]	1.44 (1.10, 1.89) $P = 0.009$	1.20 (0.93, 1.55) $P = 0.156$	1.00 (ref)

All other models: proportionality assumptions not rejected. [a]Adjusted for age in 10-year age groups, gender and the interaction between age and gender. [b]adjusted for age in 10-year age groups; [c]proportionality assumptions rejected. $P = 0.02$; 95% confidence interval (CI) in parentheses. AUD = alcohol use disorder; SES = socio-economic status.

was also the case among those aged under 60 years (Table 4). Among those aged 60 years and older, relative mortality risk did not differ between low and high SES ($t = 1.64$, $P = 0.101$).

DISCUSSION

This study showed that, in Norway, treatment for alcohol use disorder is more likely among those with low, compared to high, educational attainment. While people with AUD had a high mortality risk, we found a modest SES difference in mortality risk within this group. Thus, taking into account the substantial social difference in mortality risk in the general population, we found that AUD elevated the mortality risk to a much higher extent in the high compared to the low SES categories. This elevated relative mortality risk in the high SES category was further attenuated among those aged below 60 years, whereas no SES difference was observed in the older age group.

1Our study findings corroborate, to some extent, those of previous studies. While studies examining SES differences in AUD treatment admission appear to be rare, our finding of an elevated risk in the low SES category is well in line with the many previous reports of a higher risk of alcohol-attributable morbidity or AUD in low than in high SES groups, irrespective of SES indicator [3,4,10,19–24]. The modest SES difference in mortality among heavy drinkers, favouring high SES, is in line with some previous studies [10,14,15,19]. However, this literature contains mixed findings, and other studies found an increasing mortality risk with increasing SES [8,9,16,17]. Our finding of higher relative mortality risk in high SES AUDs corresponds to those in two previous studies [9,10], both of which used education level as SES indicator. If, in this study, we had used other SES indicators (e.g. income or occupational status), it is possible that we would have obtained smaller SES differences in relative mortality risk, assuming that a downward social drift in highly educated AUDs is associated with increased mortality risk. Nevertheless, it seems likely that the higher relative mortality risk in high SES AUDs mainly reflects a strong downward social gradient in mortality in the control cohort: a common finding in population studies [26].

In his 60-year follow-up of alcoholic men, Vaillant [10] found that those with high education, excellent social support, good health habits and late onset of abuse were actually more likely to remain chronic alcohol abusers compared to those in the low SES cohort. Thus, a stronger ability to continue a detrimental heavy drinking career over a longer time-period could be one possible explanation for the observed elevated relative mortality risk in the high SES category. Related to this is the possibility that high SES heavy drinkers tend to seek treatment at more advanced stages of their drinking careers than low SES heavy drinkers. This is compatible with our observation of a somewhat higher age at treatment admission among high SES compared to low SES AUD patients. Another possible explanation could be that the additional health risks in heavy drinkers (e.g. due to smoking, poor nutrition, social isolation, mental health problems etc.) occur to the same extent in high and low socio-economic groups. If this were the case, we could expect high SES AUD patients to be as vulnerable to premature death as their low SES counterparts, which is in line with our observations. However, other studies suggest that such additional health risks, including smoking and body mass index, seem to be more prevalent in low compared to high SES heavy drinkers [5,10].

Table 4 Mortality incidence among AUD patients and controls and mortality hazard ratio for AUDs by SES categories, also in gender- and age-specific strata.

	SES categories			
	Low SES	*Inter-mediate SES*	*High SES*	*All*
Crude mortality incidence AUD patients	22.3	24.0	23.5	23.3
Crude mortality incidence controls	8.7	4.4	2.4	4.6
Adjusted mortality hazard ratio[a]	3.29 (2.61, 4.15) $P < 0.001$	5.25 (4.18, 6.60) $P < 0.001$	8.65 (6.16,12.1) $P < 0.001$	5.08 (4.40, 5.87) $P < 0.001$
Gender-specific mortality hazard ratio[b]				
Women	2.99 (1.88, 4.75) $P < 0.001$	5.46 (3.18, 9.36) $P < 0.001$	24.21 (7.50, 78.15) $P < 0.001$	5.10 (3.70, 7.04) $P < 0.001$
Men	3.39 (2.59, 4.44) $P < 0.001$	5.21 (4.06, 6.70) $P < 0.001$	7.39 (5.16, 10.58) $P < 0.001$	5.08 (4.32, 5.97) $P < 0.001$
Age-specific mortality hazard ratio[a]				
< 60 years	3.85 (2.79, 5.32) $P < 0.001$	7.22 (5.12, 10.18) $P < 0.001$	13.69 (8.41, 22.29) $P < 0.001$	6.91 (5.62, 8.50) $P < 0.001$
60+ years	2.66 (1.88, 3.77) $P < 0.001$	3.84 (2.82, 5.23) $P < 0.001$	4.64 (2.89, 7.46) $P < 0.001$	3.47 (2.83, 4.28) $P < 0.001$

All models: proportionality assumptions not rejected. [a]Adjusted for age in 10-year age groups, gender and the interaction between age and gender; [b]Adjusted for age in 10 year-age groups. 95% confidence interval (CI) in parentheses. AUD = alcohol use disorder; SES = socio-economic status.

Limitations

In line with the majority of previous studies of this type [11], AUD was measured among patients in specialized health services. AUD patients are probably older and have more severe alcohol problems [27], and they are also likely to be at a higher mortality risk than people with AUD in the general population [11]. This suggests an upward bias in our estimates of relative mortality risk. While the public health-care system in Norway probably covers most of all AUD treatment, some treatment also occurs in private clinics outside the public health scheme, and it is quite possible that high SES heavy drinkers have sought such treatment to a larger extent than other heavy drinkers. If so, the observed negative social gradient in AUD treatment admission is inflated. However, such under-representation of high SES heavy drinkers in the public health care system *per se* is probably of less, if at all any, importance for the observed SES differences in absolute and relative mortality risk in AUD patients.

A general limitation with register studies is that they rely entirely on the routine use of diagnoses in the specialized health services, which may well be inaccurate to some extent, whether due to diagnostic misclassification administrative errors or both. If such inaccuracies are of negligible magnitude or randomly distributed by SED they are of less importance in our context. If, however, the use of AUD diagnoses differs systematically by SES, our results are biased. For example, if AUD tends to be diagnosed at a more advanced stage in high, compared to low, SES heavy drinkers, such differential diagnostic practice not only implies that the negative social gradient in AUD treatment admission is inflated, it will probably also imply that our estimates of both absolute and relative mortality risk are inflated in the high SES category.

CONCLUSION

Social inequalities in admission to AUD treatment and in relative mortality risk in AUD patients were found in a prospective cohort study in Norway. While AUD treatment admission decreased with increasing SES, relative mortality risk increased with SES, and these social differences were mainly seen among those aged under 60 years. To this end, the literature on social differences in AUD morbidity and relative mortality risk is small, and more studies are needed to extend our epidemiological knowledge as well as our understanding of the underlying mechanisms contributing to these differences.

Declaration of interests

None.

Acknowledgements

This study was funded by the Norwegian Institute of Public Health. The authors are most grateful for Solveig Christiansen's kind assistance in data management and supplementary statistical analyses.

References

1. Griswold M. G., Fullman N., Hawley C., Arian N., Zimsen S. R., Tymeson H. D., *et al.* Alcohol use and burden for 195 countries and territories, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 2018; **392**: 1015–35.
2. Rehm J., Shield K. D., Rehm M. X., Gmel G., Frick U. *Alcohol Consumption, Alcohol Dependence and Attributable Burden of Disease in Europe*. Toronto, CA: Centre for Addiction and Mental Health; 2012.
3. Collins S. E. Associations between socioeconomic factors and alcohol outcomes. *Alcohol Res* 2016; **38**: 83–94.
4. Schmidt L. A., Mäkelä P., Rehm J., Room R. Alcohol: equity and social determinants. In: Blas E., Kurup A. S., editors. *Equity, social determinants and public health programmes*. Geneva: Word Health Organization; 2010, pp. 11–29.
5. Bellis M. A., Hughes K., Nicholls J., Sheron N., Gilmore I., Jones L. The alcohol harm paradox: using a national survey to explore how alcohol may disproportionately impact health in deprived individuals. *BMC Public Health* 2016; **16**: 111.
6. Probst C., Roerecke M., Behrendt S., Rehm J. Socioeconomic differences in alcohol-attributable mortality compared with all-cause mortality: a systematic review and meta-analysis. *Int J Epidemiol* 2014; **43**: 1314–27.
7. Mackenbach J. P., Kulhánová I., Bopp M., Borrell C., Deboosere P., Kovács K., *et al.* Inequalities in alcohol-related mortality in 17 European countries: a retrospective analysis of mortality registers. *PLOS Med* 2015; **12**: e1001909.
8. Edwards G., Kyle E., Nicholls P., Taylor C. Alcoholism and correlates of mortality. Implications for epidemiology. *J Stud Alcohol* 1978; **39**: 1607–17.
9. Rossow I., Amundsen A. The disadvantage of being advantaged?—on a social gradient in excess mortality among alcohol abusers. *Addiction* 1996; **91**: 1821–9.
10. Vaillant G. E. A 60-year follow-up of alcoholic men. *Addiction* 2003; 98: 1043–51.
11. Roerecke M., Rehm J. Alcohol use disorders and mortality: a systematic review and meta-analysis. *Addiction* 2013; **108**: 1562–78.
12. Westman J., Wahlbeck K., Laursen T. M., Gissler M., Nordentoft M., Hällgren J., *et al.* Mortality and life expectancy of people with alcohol use disorder in Denmark, Finland and Sweden. *Acta Psychiatr Scand* 2015; **131**: 297–306.
13. Probst C., Roerecke M., Behrendt S., Rehm J. Gender differences in socioeconomic inequality of alcohol-attributable mortality: a systematic review and meta-analysis. *Drug Alcohol Rev* 2015; **34**: 267–77.
14. Sundby P. *Alcoholism and Mortality.* Oslo: Universitetsforlaget; 1967.
15. Lindberg S., Ågren G. Mortality among male and female hospitalized alcoholics in Stockholm 1962–1983. *Br J Addict* 1988; **83**: 1193–200.
16. Schmidt W., de Lint J. Social class and the mortality of clinically treated alcoholics. *Br J Addict Alcohol Other Drugs* 1970; 64: 327–31.
17. Combs-Orme T., Taylor J. R., Robins L. N. Occupational prestige and mortality in black and white alcoholics. *J Stud Alcohol* 1985; 46: 443–6.
18. Vaillant G. E., Schnurr P. P., Baron J. A., Gerber P. D. A prospective study of the effects of cigarette smoking and alcohol abuse on mortality. *J Gen Intern Med* 1991; **6**: 299–304.
19. Mäkelä P., Keskimäki I., Koskinen S. What underlies the high alcohol related mortality of the disadvantaged: high morbidity or poor survival? *J Epidemiol Community Health* 2003; **57**: 981–6.
20. Kendler K. S., Ohlsson H., Sundquist J., Sundquist K. School achievement, IQ, and risk of alcohol use disorder: a prospective, co-relative analysis in a Swedish national cohort. *J Stud Alcohol Drugs* 2017; **78**: 186–94.
21. Søndergaard G., Osler M., Andersen A. M. N., Andersen P. K., Dalton S. O., Mortensen L. H. Alcohol-related morbidity and mortality within siblings. *Addiction* 2015; **110**: 451–60.
22. Sydén L., Sidorchuk A., Mäkelä P., Landberg J. The contribution of alcohol use and other behavioural, material and social factors to socio-economic differences in alcohol-related disorders in a Swedish cohort. *Addiction* 2017; **112**: 1920–30.
23. Romelsjö A., Diderichsen F. Changes in alcohol-related inpatient care in Stockholm County in relation to socioeconomic status during a period of decline in alcohol consumption. *Am J Public Health* 1989; **79**: 52–6.
24. Hwang S. W., Agha M. M., Creatore M. I., Glazier R. H. Age-and sex-specific income gradients in alcohol-related hospitalization rates in an urban area. *Ann Epidemiol* 2005; **15**: 56–63.
25. Cumming G. Inference by eye: reading the overlap of independent confidence intervals. *Stat Med* 2009; **28**: 205–20.
26. Mackenbach J. P., Stirbu I., Roskam A.-J. R., Schaap M. M., Menvielle G., Leinsalu M., *et al.* Socioeconomic inequalities in health in 22 European countries. *N Engl J Med* 2008; **358**: 2468–81.
27. Storbjörk J., Room R. The two worlds of alcohol problems: who is in treatment and who is not? *Addict Res Theory* 2008; **16**: 67–84.