The role of alcohol use and drinking patterns in socioeconomic inequalities in mortality: a systematic review

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Summary

Background Individuals with low socioeconomic status (SES) experience disproportionately greater alcohol-attributable health harm than individuals with high SES from similar or lower amounts of alcohol consumption. Our aim was to provide an update of the current evidence for the role of alcohol use and drinking patterns in socioeconomic inequalities in mortality, as well as the effect modification or interaction effects between SES and alcohol use, as two potential explanations of this so-called alcohol-harm paradox.

Methods We did a systematic review, searching Embase, Medline, PsycINFO, and Web of Science (published between Jan 1, 2013, and June 30, 2019) for studies reporting alcohol consumption, SES, and mortality. Observational, quantitative studies of the general adult population (aged ≥15 years) with a longitudinal study design were included. Two outcome measures were extracted: first, the proportion of socioeconomic inequalities in mortality explained by alcohol use; and second, the effect modification or interaction between SES and alcohol use regarding mortality risks. This study is registered with PROSPERO (CRD42019140279).

Findings Of 1941 records identified, ten met the inclusion criteria. The included studies contained more than 400 000 adults, more than 30 000 deaths from all causes, and more than 3000 100% alcohol-attributable events. Alcohol use explained up to 27% of the socioeconomic inequalities in mortality. The proportion of socioeconomic inequalities explained systematically differed by drinking pattern, with heavy episodic drinking having a potentially significant explanatory value. Although scarce, there was some evidence of effect modification or interaction between SES and alcohol use.

Interpretation To reduce socioeconomic inequalities in mortality, addressing heavy episodic drinking in particular, rather than alcohol use in general, is worth exploring as a public health strategy.

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Introduction

The 2030 Agenda for Sustainable Development, adopted by all UN member states in 2015, provides a blueprint for global peace and prosperity.[1] At the core of this blueprint are the 17 Sustainable Development Goals. These goals recognise the interlinkages between different aspects of development, such as poverty and other deprivations, health, education, and inequality, and try to set targets to improve all of these aspects. Alcohol use is mentioned as an important determinant of health,[2] and reducing alcohol use might aid in achieving some of the major Sustainable Development Goals, such as eliminating poverty and reducing inequalities.[3] We will examine one of these postulated links—that is, the contribution of alcohol consumption to socioeconomic inequalities in health outcomes.

Socioeconomic inequalities in alcohol-attributable mortality have been documented in several, mainly high-income, countries. A meta-analysis published in 2015 found that individuals with low socioeconomic status (SES) have a two-fold to five-fold higher risk of dying from an alcohol-attributable cause of death than individuals with high SES.[4,5] This mortality gap for low compared with high SES was about one-and-a-half-times to two-times wider for alcohol-attributable mortality than for all-cause mortality.[4] Although the studies included in the respective review did not account for alcohol consumption, other investigators have shown that the prevalence of alcohol use is lower among individuals with low SES than among those with high SES.[6,7] Furthermore, in the few studies available, differences in alcohol-attributable harm could not be sufficiently explained by the amount of alcohol consumed in different socioeconomic groups.[8] The finding that people with low SES have disproportionately greater alcohol-attributable harm than people with high SES despite similar or lower amounts of alcohol consumption is commonly referred to as the alcohol-harm paradox.[9,10] One explanation for the paradox is that other behavioural risk factors (such as obesity and smoking) cluster in individuals with low SES and interact with alcohol use, resulting in exacerbated health consequences of alcohol use.[9] Differences in access to health services, variations in the safety of the drinking context, and differential drinking cultures are additional potential factors contributing to the elevated risks related to alcohol use for individuals with low SES.[11,12]

Research in context

Evidence before this study

Even though the alcohol-harm paradox is a known public health occurrence, the reasons underlying the exacerbated socioeconomic inequalities in alcohol-attributable harm remain unclear. The most recent systematic literature review of the role of alcohol consumption in the relationship between socioeconomic status (SES) and alcohol-attributable health outcomes (morbidity and mortality) was published in 2015, covering the literature up to Nov 30, 2012. The review identified only three studies for which the unique contribution of alcohol use to socioeconomic inequalities in health outcomes (incident stroke and hypertension) could be quantified and one study that investigated effect modification between SES and alcohol use with regard to breast cancer risk. The authors concluded that, at the time, the evidence was insufficient to draw any conclusions.

Added value of this study

The current study reviews the evidence put forth by ten studies published since Jan 1, 2013. Even though the evidence shows that socioeconomic differences are nearly two times higher when considering 100% alcohol-attributable events rather than all-cause mortality, this heightened socioeconomic inequality cannot be explained by differences in the amount of alcohol use. However, by systematically synthesising the evidence from all available studies, this systematic review is to the best of our knowledge the first to show that the pattern of alcohol use is pertinent in explaining socioeconomic differences in not only alcohol-attributable mortality, but also all-cause mortality.

Implications of all the available evidence

With socioeconomic inequalities in mortality increasing in many countries globally, this study adds further evidence of the necessity for an alcohol control intervention strategy that takes socioeconomic inequalities and effectiveness conditional on SES into consideration. According to the findings of the current review, public health strategies that address heavy episodic drinking patterns specifically, rather than alcohol use in general, are worth exploring as a means to reduce socioeconomic inequalities in mortality. However, further research using multiple linked data sources is warranted to identify the aetiological trajectories of the alcohol-harm paradox.

In 2015, Jones and colleagues[13] published a systematic literature review of studies published up to November, 2012, on the role of alcohol use in the observed SES differences in the risk of alcohol-attributable morbidity and mortality. The authors identified only three studies for which the unique contribution of alcohol use to socioeconomic differences in alcohol-attributable health outcomes (namely incident stroke and hypertension) could be quantified.[14–16] In addition, the authors identified one study that presented evidence on effect modification of SES on the breast cancer risk related to heavy alcohol use, finding strikingly higher odds ratios (ORs) for participants with low rather than high SES.[17] Overall, the authors concluded "A key finding of our review is the lack of studies that have explored in depth, the relationship between alcohol-attributable disease, SES, and alcohol use."[13] Given that the review by Jones and colleagues[13] is the most recent review, we aimed to provide an update of the current evidence for the role of alcohol use and drinking patterns in socioeconomic differences and the effect modification or interaction effects between SES and alcohol use, as two potential explanations of the alcohol-harm paradox with respect to mortality.

Methods

Search strategy and selection criteria

The protocol of this systematic review was published on PROSPERO (registration number CRD42019140279). We have adhered to the standards set out in Preferred Reporting Items for Systematic Reviews and Meta-Analyses.[18]

See Online for appendix

Systematic literature searches were done in Embase, Medline, PsycINFO, and Web of Science. Search terms included terms on alcohol consumption, mortality, SES, and study design (appendix p 2). Articles published between Jan 1, 2013, and June 30, 2019, were included, and no language or geographical restrictions were applied. Articles were included if they allowed for a quantification of either the role of alcohol use and drinking patterns to socioeconomic differences, or the effect modification or interaction between SES and alcohol use. Inclusion and exclusion criteria are listed in the appendix (p 3). To ensure comparability, the measurement of SES was restricted to education, occupation, employment status, income, and household assets, or combinations thereof. SES had to be measured on the individual or household level. The following outcomes were considered: (1) mortality from 100% alcohol-attributable causes of death (appendix p 4), (2) mortality from causes of death that have an alcohol-attributable fraction of 10% or more globally (appendix p 5),[2] (3) mortality from all causes, and (4) 100% alcohol-attributable events (ie, mortality and hospitalisations combined). References were screened independently by three reviewers (CK, CP, SS). To reach high agreement (Kappa>0·8),[19] two subsamples of 50 references were used to train reviewers. Uncertain cases were discussed between all three reviewers and decided in consensus.

Data analysis

Data were extracted on study and population characteristics, design, assessment of SES, assessment of the outcome, assessment of alcohol use, and summary estimates. Two types of summary estimates were considered according to the two possible explanations for the alcohol-harm paradox: the proportion of SES inequalities that can be explained by alcohol use and drinking patterns, and indicators of effect modification or interaction as specified by Knol and VanderWeele.[20] For the indicators of effect modification or interaction, stratified risk estimates were included; however, a test for statistical significance (through relative excess risk due to interaction on the additive scale or, eg, an interaction term on the multi–plicative scale) was considered stronger evidence than the stratified risk estimates. Alternatively, studies were included if they reported sufficient original data to calculate the above (for formulas, see appendix p 6). Hazards ratios, relative risks, and standardised mortality ratios were treated as equivalent measures of risk; therefore, the term risk ratio will be used from here on out. The proportion of the SES inequalities explained by alcohol use was calculated as

$$1-\frac{\ln(RR_{alcohol\ adjusted})}{\ln(RR_{minimally\ adjusted})}.$$

Minimally adjusted risk ratio (ie, adjusted for at least age and gender) was preferred over crude estimates. The risk ratio used in the numerator was adjusted for alcohol use in addition to the adjustments used in the risk ratio of the denominator. When available, CIs were reported. To avoid duplicate data, all studies were checked for overlap in the sample with regard to baseline sample and follow-up. Study quality was assessed using an adapted version of the Critical Appraisal Checklist for Cohort Studies (appendix p 4).[21] All data were extracted by one reviewer and then independently crosschecked by a second reviewer. All discrepancies were discussed, and reconciled by consensus. Risk of bias across studies (eg, publication bias) was not assessed because risk of bias can be considered low in large record linkage studies, which are included in the systematic review.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or the writing of the report. The corresponding author had full access to all data in the study and final responsibility for the decision to submit this systematic review for publication.

Results

A total of 1941 records were identified, and a total of ten studies were included in the review (figure 1; table 1). The included studies contained a sample of over 400 000 adults, more than 30 000 deaths from all causes, and over 3000 100% alcohol-attributable events. All of the included studies were from high-income countries, namely Denmark (two), the Netherlands (one), Norway (one), Scotland (two), Sweden (two), and USA (two). All but one study used baseline data from a survey or a cohort study, linked to a cause of death registry. Nandi and colleagues[22] used proxy interviews to assess follow-up information regarding the vital status of participants of a cohort study. Whereas three studies used at least two indicators of SES,[23–25] each of the remaining seven studies used a single indicator of SES. In total, six studies used education as a measure of SES,[23–28] four used occupation,[23,24,29,30] three used income,[23,25,30] and two used a combined measure that integrate multiple SES indicators such as education, income, and household assets using a statistical dimension reduction procedure.[22,31] Regarding study quality, all of the studies fulfilled at least three thirds of the quality criteria with the exception of Nordahl and colleagues.[26] Noticeably,

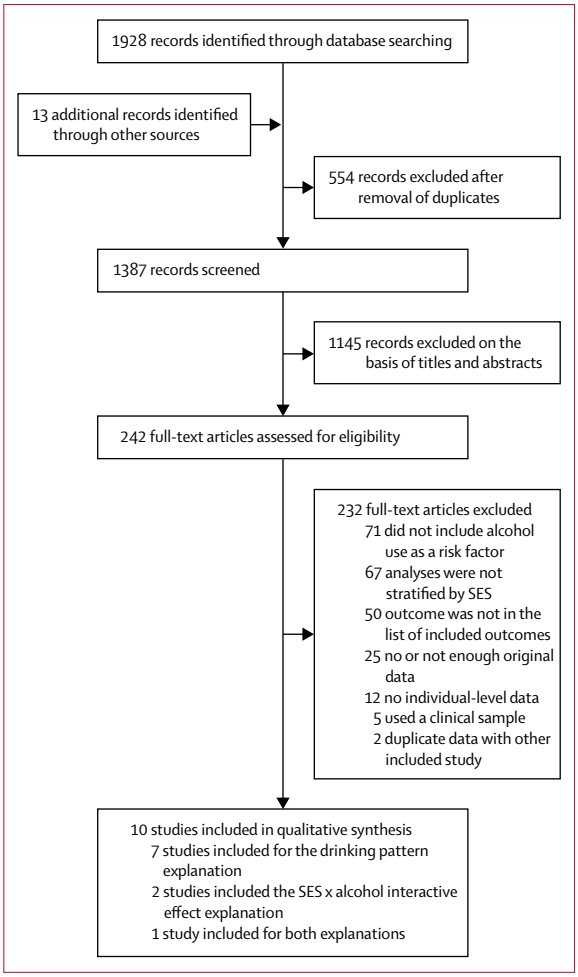

Figure 1: **Study selection**
The studies selected for review investigate two potential explanations of the alcohol-harm paradox with respect to mortality (1) the role of alcohol use and drinking patterns in socioeconomic differences in mortality, and (2) the effect modification or interaction effects between SES and alcohol use.

	Population		Study design		Exposure assessment		Outcome assessment		
	Country	Gender (age range in years)	Design	Summary estimate*	Years	Sample size	Years	Outcome	Events
Christensen and colleagues (2017)[28]	Denmark	M, W (30–70)	Longitudinal, data linkage	Effect modification or interaction	1981–1982, 1981–1983, 1982–1992, 1993–1997, 1999–2001	74 278	1981–2009	AAM or AAM and AAH combined†	302 for AAM, 1718 for AAM and AAH
Degerud and colleagues (2018)[31]	Norway	T (NA)¶	Longitudinal, data linkage	Effect modification or interaction	1987–1988, 1994–1999, 1993–2003	188 603	NA‡	ACM	21 624
Katikireddi and colleagues (2017)[23]	Scotland	T (NA)§	Longitudinal, data linkage	Effect modification or interaction, proportion explained	1995, 1998, 2003, 2008–2012	50 236	1995–2012	AAM and AAH†	1020
Mehta and colleagues (2015)[25]	USA	T (25–96)	Longitudinal, data linkage	Proportion explained	1989, 1994, 2001–02, 2011–12	3617	1986–2011	ACM	1832
Nandi and colleagues (2014)[22]	USA	T (≥50)	Cohort study, proxy interviews	Proportion explained	1992	8037	1998–2008	ACM	51
Nordahl and colleagues (2014)[26]	Denmark	M, W (30–70)	Longitudinal, data linkage	Proportion explained	1981–1983, 1982–1984, 1986–1987, 1991–1992, 1993–1997, 1999–2001	76294	1981–2009	ACM	12340
Sydén and Landberg (2017)[24]	Sweden	T (25–74)	Longitudinal, data linkage	Proportion explained	2002	21 064	2002–2007	ACM	300
Sydén and colleagues (2017)[29]	Sweden	T (25–64)	Longitudinal, data linkage	Proportion explained	2002	17 440	2002–2011	AAM and AAH†	388
van Hedel and colleagues (2018)[27]	Netherlands	M, W (15–47)	Longitudinal, data linkage	Proportion explained	1991	6099	1991–2013	ACM	NA
Whitley and colleagues (2014)[30]	Scotland	T, M, W (NA)	Longitudinal, data linkage	Proportion explained	1987–1988, 1990–1992, 1995–1997, 2000–2004, 2007–2008	1534 (1932 cohort), 1426 (1952 cohort)	1987–2011	ACM	719 (1932 cohort), 120 (1952 cohort)

M=men. W=women. T=total (both genders combined). AAM=100% alcohol-attributable mortality. AAH=100% alcohol-attributable hospitalisation. ACM=all-cause mortality. HED=heavy episodic drinking. NA=not available. *Summary estimates are referring to either of the two potential explanations with (1) proportion of inequalities explained by alcohol use and drinking patterns, and (2) effect modification or interaction between socioeconomic status and alcohol use. †AAM and AAH is referred to as "alcohol-attributable events". ‡The average follow-up time was 16·6 years (SD 4·0). §Average age of 48 years (SD 17·5). ¶Average age of 47 years (SD 11·1).

Table 1: **Characteristics of all included studies**

eight of ten studies did not report the completeness of follow-up or record linkage (appendix pp 7–8).

For clarity, we focused on the comparison between the lowest and the highest SES group. However, findings comparing the medium and the highest SES group were also assessed and showed the same overall picture.

Eight studies allowed for the quantification of the contribution of alcohol use to socioeconomic inequality (table 2).

Six studies investigated the proportion of socioeconomic inequalities in all-cause mortality that can be explained by alcohol consumption. All of these studies found elevated all-cause mortality rates among individuals with low compared with high SES, irrespective of the measure of SES. Relative socioeconomic inequalities ranged from a risk ratio of 1·58 (95% CI 1·37–1·83)[27] to a risk ratio of 3·58 (95% CI 3·52–3·63).[26] In five studies, alcohol consumption was quantified as the average number of drinks consumed per week[24,26,27,30] or per month.[25] In these studies, alcohol consumption explained between –3% (95% CI –20% to 10%; women)[27] and 14% (no CI available; both genders)[30] of the socioeconomic inequalities in mortality. Notably, the three studies that quantified the contribution of the average number of drinks consumed to socioeconomic inequality in all-cause mortality by gender[26,27,30] found a small but negative contribution among women and a small but positive contribution among men. Rather than quantifying alcohol use as the average number of drinks consumed over a particular time period, one study focused on the quantity consumed per drinking occasion,[22] whereas another study focused on heavy episodic drinking (generally defined as 60 g or more on a single occasion).[24] In doing so, these studies explained higher proportions in the socioeconomic inequality in all-cause mortality than the other studies: 17% (no CI available; both genders)[22] and 24–27% (no CI available; both genders).[24]

	Gender	SES indicator (number of levels)	Alcohol use (number of levels)	SES inequality (95% CI)*	Proportion explained by alcohol use (95% CI)†	Adjustment
All-cause mortality (outcome)						
Mehta and colleagues (2015)[25]	T	Years of education: three levels ranging from "0 to 11 years" to "16+ years of education"; income (participant and spouse): three levels ranging from "<$10 000" to "$30 000+"	Drinks per month: 0, 1–90, 91+ drinks (men); 0, 1–60, 61+ drinks (women)	Education: risk ratio 1·88 (1·48–2·39); income: risk ratio 2·09 (1·72–2·56)	Education: 9% (1% to 16%); income: 6% (–2% to 13%)	Age, gender, race
Nandi and colleagues (2014)[22]	T	Quartiles of an SES index combining information on education, occupation, labour force status, household income, and household wealth	Drinks per drinking occasion: 0, 1–2, 3–4, ≥5 drinks (past 3 months)	Risk ratio 2·84 (2·25–3·60)	17%	Age, gender, race, early-life SES
Nordahl and colleagues (2014)[26]	M, W	Educational attainment: three levels ranging from "primary and lower secondary education" to "medium-cycle university or non-university programs as well as long-cycle university programs"	Number of drinks consumed per week: 0, 1–7, 8–14, 15–21, 22–28, ≥29 drinks	Risk ratio 3·58 (3·52–3·63) for both genders combined	3% (1% to 4%) for men; –1% (–4% to –6%) for women	Age, cohort
Sydén and Landberg (2017)[24]	T	Educational attainment: three levels ranging from "primary school or less" to "post-secondary education"; occupation: six levels ranging from "unskilled workers" to "higher non-manual employees"; personal income: quintiles	Drinks per week: 0, 1–7, 8–21, ≥22 drinks (men); 0, 8–14, ≥15 drinks (women); frequency of HED: number of occasions with ≥10 drinks in the past 12 months; combined measure of the average quantity and HED (10 levels)	Education: risk ratio 1·71 (1·21–2·42); Occupation: risk ratio 1·83 (1·22–2·75); income: risk ratio 1·84 (1·19–2·83)	For drinks per week: education: 8%, occupation: 5%, income: 7%; for HED: education: 27%, occupation: 25%, income: 24%; for the combined measure: education: 22%, occupation: 18%, income: 17%	Age, gender
van Hedel and colleagues (2018)[27]	M, W	Level of education: four levels ranging from "primary education only" to "higher vocational school and university"	Weekly number of alcoholic drinks consumed: 0, 1–14, 15–21, ≥22 drinks (men); 0, 1–7, 8–14, ≥15 drinks (women)	Risk ratio 1·58 (1·37–1·83) for men; risk ratio 1·59 (1·25–2·02) for women	5% (–3% to 14%) for men; –3% (–20% to 10%) for women	Age
Whitley and colleagues (2014)[30]	T, M, W	Occupational class: manual versus non-manual	Weekly number of alcoholic drinks consumed: 0, 1–21, ≥22 drinks (men); 0, 1–14, ≥15 drinks (women)	1952 cohort: risk ratio 1·80 (1·25–2·58) for both genders combined; 1932 cohort: risk ratio 1·59 (1·30–1·95) for men; 1932 cohort: risk ratio 1·74 (1·41–2·16) for women	1952 cohort: 14% for both genders combined; 1932 cohort: 1% for men; 1932 cohort: –1% for women	Age and gender for analyses of both genders combined; age for analyses of either gender
Alcohol-attributable events (outcome)‡						
Katikireddi and colleagues (2017)[23]	T	Educational attainment: six levels ranging from "none" to "degree or above"; occupation: six levels ranging from "unskilled" to "professional"; household income: quintiles	Drinks per week: 0, 1–10, 11–20, 21–50, ≥51 drinks (men); 0, 1–7, 8–13, 14–35, ≥36 drinks (women); HED: 8 drinks per occasion (men); 6 drinks per occasion (women)	Education: risk ratio 3·76 (2·96–4·77); occupation: risk ratio 5·22 (3·28–8·30); income: risk ratio 4·41 (3·07–6·33)	Drinks per week: education: 7%, occupation: 11%, income: –6%; combined measure of average quantity and HED: education: 7%, occupation: 11%, income: –6%	Age, gender, survey wave
Sydén and colleagues (2017)[29]	T	Occupation: six levels ranging from "unskilled workers" to "higher non-manual employees"	Drinks per week: 0, 1–7, 8–21, ≥22 drinks for men and 0, 8–14, ≥15 drinks for women; frequency of HED: number of occasions with ≥10 drinks in the past 12 months; combined measure of the average quantity and HED (10 levels)	Risk ratio 4·08 (2·78–5·98)	Drinks per week: 2%; HED: 25%; combined measure: 24%	Age, gender, country of birth

SES=socioeconomic status. M=men. W=women. T=total (both genders combined). HED=heavy episodic drinking. *Comparing low versus high SES. Indicating the minimally adjusted risk ratio before adjusting for alcohol use. †CI only shown when available from the original study. ‡Includes studies that used 100% alcohol-attributable mortality exclusively or studies that used both 100% alcohol-attributable mortality and 100% alcohol-attributable hospitalisation.

Table 2: **Summary of study findings regarding the proportion of socioeconomic inequalities in all-cause mortality and alcohol-attributable events (100% alcohol-attributable mortality and 100% alcohol-attributable hospitalisation), explained by alcohol use and drinking patterns**

Two studies investigated the contribution of alcohol consumption to socioeconomic inequalities in 100% alcohol-attributable events.[23,29] The observed relative inequalities in alcohol-attributable events were considerably higher than the inequalities observed for all-cause mortality, ranging between a risk ratio of 3·76

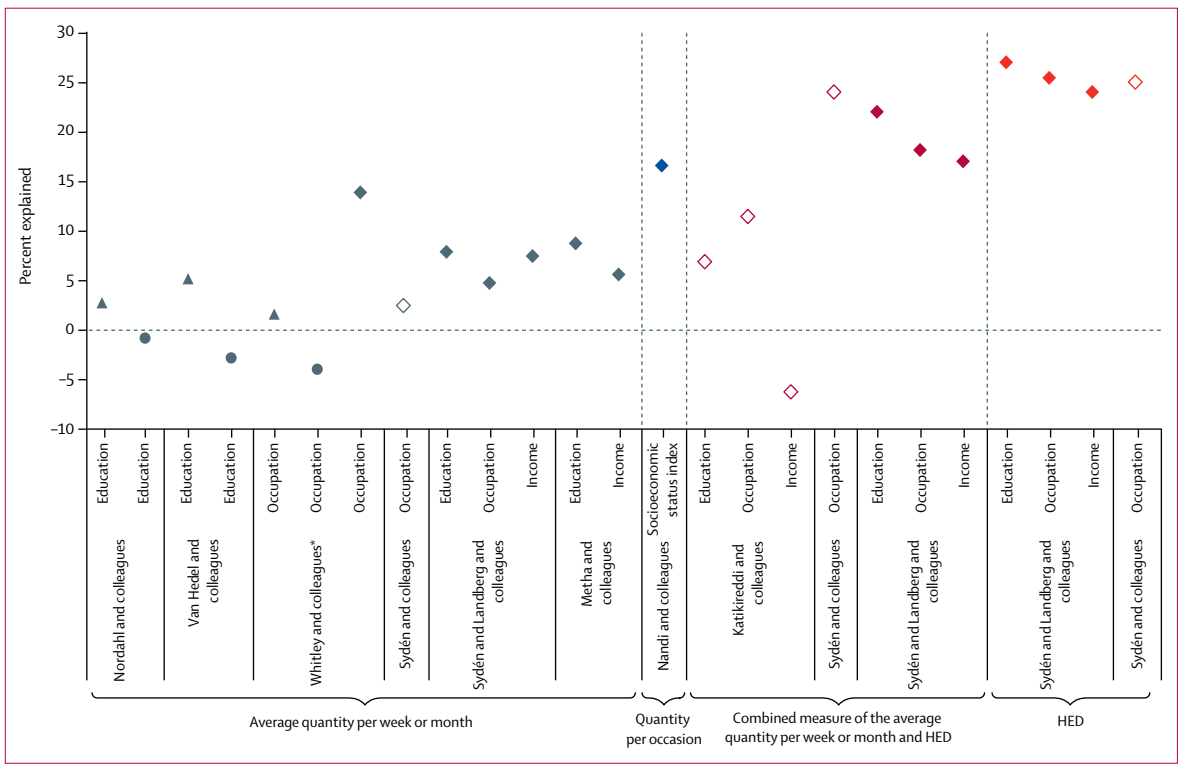

Figure 2: **Proportion of socioeconomic inequalities that can be explained by alcohol use**
Proportion of socioeconomic inequalities in all-cause mortality (solid symbols) and 100% alcohol-attributable mortality and hospitalisations (outlined symbols) explained by alcohol use, ordered by the measurement of alcohol use: average quantity of drinks consumed per week or month, usual quantity consumed per occasion, a combined measure of the average quantity per week or month and heavy episodic drinking, and heavy episodic drinking only. Estimates for men are shown as triangles, for women as circles, and for both genders combined as diamonds. HED=heavy episodic drinking. *The two left datapoints of this reference refer to the 1932 cohort, and the right data point to the 1952 cohort for which no gender-specific estimates were reported.

(95% CI 2·96–4·77),[23] when measuring SES as education level, and a risk ratio of 5·22 (95% CI 3·28–8·30), when SES was represented by occupation type.[23] As for all-cause mortality, the highest proportion explained by alcohol use was observed when accounting for heavy episodic drinking (25%; no CI available; both genders),[29] whereas accounting for the number of drinks per week only (2%; no CI available; both genders) made the smallest contribution to socioeconomic inequalities in alcohol-attributable events.[29] One outlier was observed for the proportion of inequalities explained by the combination of the usual number of drinks per week and heavy episodic drinking when using income as a measure of SES (–6%; no CI available; both genders). Figure 2 summarises the results of the eight studies that allowed for a quantification of the proportion of socioeconomic inequalities in all-cause mortality (highest *vs* lowest SES in each study) or in 100% alcohol-attributable events that can be explained by alcohol use.

Three studies investigated the joint effects of SES and alcohol use on mortality by analysing effect modification or interaction effects with regard to the outcome risk.[23,28,31] Degerud and colleagues[31] quantified SES on the basis of a combined measure of life course socioeconomic position categorised into three groups. Tests for effect modification regarding the risk of all-cause mortality were reported for alcohol consumption frequency (infrequent, once per month to once per week, 2–3 times per week, and 4–7 times per week), and the frequency of heavy episodic drinking. Alcohol use in a low or moderate frequency (compared with infrequent use) was not associated with increased mortality risks in any of the SES groups. However, highly frequent alcohol use (compared with infrequent alcohol use) was associated with an increased risk of dying (risk ratio 1·48; 95% CI 1·23–1·79) among those with low SES but not among those with high SES (adjusted for age and gender). The risk was not attenuated after adjusting for several other health behaviours and disease markers. The authors also found evidence of interaction effects for the combined effects of highly frequent alcohol use and low SES (risk ratio 1·63; 95% CI 1·32–2·01; with the reference being infrequent drinkers with high SES). When looking at the frequency of heavy episodic drinking in the past 12 months, having one or more heavy episodic drinking occasions per week (compared with no heavy episodic drinking occasions) was associated with elevated mortality risks in all SES groups, with slightly higher risks among those with low SES (risk ratio 1·61; 95% CI 1·17–2·20) than among those with high SES (risk ratio 1·33; 95% CI 1·08–1·63;

adjusting for age and gender). However, there was no evidence of interaction effects (risk ratio 1·28; 95% CI 0·89–1·85; with the reference group being individuals with high SES and no heavy episodic drinking occasions).

Christensen and colleagues[28] investigated joint effects with regard to alcohol-attributable events. SES was assessed via education, categorised into two categories; alcohol use was assessed as the number of drinks per week, categorised into three categories (men: 0–14, 15–28, and ≥29 drinks per week; women: 0–7, 8–21, and 22+ drinks per week). We calculated risk ratios related to alcohol use by SES group and by gender. Among men with high SES, having 15–28 drinks per week (compared with 0–14 drinks per week) was associated with a risk ratio of 2·10 (95% CI 2·09–2·11) for an alcohol-attributable event. Similarly, the risk ratio among men with low SES was 2·13 (95% CI 2·11–2·16). For the highest amount of alcohol use (≥29 compared with 0–14 drinks per week), the risk ratio was 4·70 (95% CI 4·66–4·74) among men with high SES, but higher among men with low SES at 5·59 (95% CI 5·47–5·70). For women with low SES, the risk associated with alcohol use was elevated at both degrees of consumption. The risk ratio for drinking 8–21 compared with 0–7 drinks per week was 1·89 (95% CI 1·88–1·90) among women with high SES, but 4·05 (95% CI 4·02–4·08) among women with low SES. The risk ratio for drinking 22 or more drinks compared with 0–7 drinks per week was 4·83 (95% CI 4·79–4·87) among women with high SES and 10·26 (95% CI 10·04–10·49) among women with low SES. In addition, the authors calculated that an excess of 289 events per 100 000 person years (95% CI 123–457) occurred among men because of interaction effects between education and alcohol consumption (adjusted for age, study cohort, and birth cohort). Among women, this number was 239 excess events per 100 000 person years (95% CI 90–388) due to the interaction.

Katikireddi and colleagues[23] dichotomised all measures of SES to analyse the joint effects of SES and alcohol consumption (on the basis of the number of drinks in the past 7 days) on alcohol-attributable events, while adjusting for age, gender, survey wave, smoking, BMI, and binge drinking in the past 7 days. In stratified analyses (simultaneous stratification by alcohol use and SES), the authors found that compared with individuals with light alcohol use and high education, excessive alcohol use (≥51 drinks per week for men and ≥36 drinks per week for women) was associated with a risk ratio of 5·26 (95% CI 3·56–7·77) among those with high education, but a risk ratio of 9·92 (95% CI 7·27–13·54) among those with low education. Similar effects were found for the other measures of SES. The authors tested for interaction effects on the multiplicative scale, which resulted in no evidence to support this postulation.[20]

Discussion

To our knowledge, this systematic review is the most comprehensive overview among the evidence currently available on two potential explanations of the alcohol-harm paradox. In line with the paradox, the identified studies showed that compared with individuals with high SES, individuals with low SES had a risk ratio of 1·6–3·6 for all-cause mortality, and a risk ratio of 3·8–5·2 for alcohol-attributable mortality. The proportion of the inequalities that could be explained by alcohol use varied systematically with the way alcohol use was accounted for. The mere quantity of alcohol consumed over a particular period had little explanatory value (–5% to 15%) with regard to explaining the observed socioeconomic inequalities. However, the usual quantity per drinking occasion or the frequency of heavy episodic drinking explained about 15–30% of the observed socioeconomic inequalities. Overall, these results indicate that differences in drinking patterns, rather than overall consumption, help to explain the alcohol-harm paradox. However, the results also show that alcohol use cannot explain the majority of the observed inequalities; reasons might include measurement error of alcohol use and the exclusion of groups with the highest alcohol consumption from household surveys.

Only three studies were identified that investigated the joint effects of SES and alcohol use.[23,28,31] All three studies provided some indication that SES modified the effects of alcohol use. However, the findings regarding interaction effects on the multiplicative scale were inconclusive. The strongest evidence for the joint effects of alcohol use and low SES on the multiplicative scale came from Christensen and colleagues.[28] Degerud and colleagues[31] also found evidence of multiplicative interaction for highly frequent alcohol use among individuals with low SES, whereas Katikireddi and colleagues[23] found no significant interaction effects. Overall, the findings indicate that more detrimental effects of alcohol consumption in individuals with low SES could contribute to the alcohol-harm paradox.

One limitation of this systematic review is that the included studies came exclusively from high-income countries, limiting global generalisability. Furthermore, there was some overlap between the data used in two of the studies included.[24,29] Another limitation is the heterogeneity in the outcomes considered in each of the studies. Because of the scarcity of studies with access to the type of data required to investigate the relationships between individual-level alcohol use, SES, and (cause-specific) mortality risk, we had to allow for broad inclusion criteria with regard to the outcome. The measurement of alcohol use is also a potential limitation because substantial underreporting often occurs, including underreporting of heavy drinking occasions. Finally, the heterogeneity and low number of studies did not allow for a meta-analysis to statistically summarise the study findings.

This study joins others in the call for further research into effect modification and interaction between SES and alcohol use. As previously proposed by Jones and colleagues,[13] future research could seek to adopt different approaches such as individual data meta-analysis.

Although some of the studies included in this review already combined data from multiple cohorts,[31] such approaches are becoming more feasible with the increasing availability of data linkage across multiple sources. Linking multiple data sources might allow to identify the causal pathways of the alcohol-harm paradox by revealing aetiological trajectories, including biomarkers regarding physiological susceptibility, exposure to environmental and psychological stressors, risk behaviours, and access to health-care services.[32] Such rich data sources will connect the currently fragmented insights and partial explanations and give rise to a comprehensive understanding of the paradox.[28] This understanding is of paramount importance to addressing the root causes of socioeconomic inequality in alcohol-attributable outcomes and beyond.

Several conclusions for interventions can be drawn on the basis of the findings of this review. A systematic review published in 2015, which examined alcohol control policies and interventions that could reduce socioeconomic inequalities, found that initiatives addressing neighbourhood planning, zoning, and licensing are among the most effective approaches to reduce socioeconomic inequalities in alcohol-attributable outcomes.[33] Research has shown that alcohol outlet density tends to be higher in areas that are deprived than in areas that are not deprived.[34,35] The regulation of alcohol outlet density can be accomplished at the local level through licensing systems and in close collaboration between law enforcement, government agencies, and health authorities. However, the current study showed that rather than addressing alcohol use in general, addressing patterns of heavy episodic drinking in particular is likely to be a more promising strategy in reducing socioeconomic differences in mortality. Minimum unit pricing has been identified as such a strategy, which should mostly affect heavy drinkers, ie, drinkers with multiple heavy drinking occasions per week.[36] First results from the implementation of this policy in Scotland show that heavy drinkers from low SES groups indeed reduced their consumption the most.[37] In addition, screening and brief intervention have been shown to be an effective approach to identifying and reducing risky alcohol use.[38] However, implementation on a large scale has been scarce,[39] and a scale-up in primary care could exacerbate health disparities, given that individuals with low SES are less likely to utilise primary care services.[40] Therefore, equal access to screening and intervention facilities would be a prerequisite for such a strategy to reduce socioeconomic inequalities.

In conclusion, the association between alcohol consumption and SES is complex, and the nature of data needed to properly explore the interaction effects between alcohol consumption and SES is still challenging to obtain. This study adds to the current body of literature showing that alcohol consumption alone is not sufficient to explain the disproportionate effect of alcohol-related harms on individuals with low SES. However, a closer look at the findings reveals the importance of accounting for drinking patterns rather than just the average quantity of alcohol consumed; albeit this interpretation is based on only two studies, and warrants further research.

Even though the evidence clearly shows that socioeconomic differences are nearly two times higher when considering 100% alcohol-attributable events rather than all-cause mortality,[4] this increased socioeconomic inequality cannot be fully explained by differences in the drinking behaviour, and there is —at this point in time— not sufficient evidence for interaction effects between SES and alcohol use. Even though addressing alcohol use and alcohol-attributable mortality remains a promising strategy to reduce health inequalities, a better understanding of the complex relationships between alcohol use, SES, and mortality risks has to be gained to effectively inform the development of population health policies.

Contributors

CP had full access to all of the data in the study and takes responsibility for the integrity of the data and accuracy of the study. CP conceptualised and designed the study and oversaw all steps of the process. CP, CK, and SS did the literature research, study selection, and data extraction. All authors contributed to the interpretation of the findings. CP and CK wrote the first draft of the manuscript. All authors contributed to the writing and revision of the manuscript and approved the final version.

Declaration of interests

We declare no competing interests.

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