Drug and Alcohol REVIEW

APSAD

Drug and Alcohol Review (2020)
DOI: 10.1111/dar.13114

The link between per capita alcohol consumption and alcohol-related harm in educational groups

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Abstract

Introduction and Aims. *Research based on individual-level data suggests that the same amount of alcohol yields more harm in low-socioeconomic status (SES) groups than in high-SES groups. Little is known whether the effect of changes in population-level alcohol consumption on harm rates differs by SES-groups. The aim of this study was to elucidate this issue by estimating the association between per capita alcohol consumption and SES-specific rates of alcohol-related mortality.* ***Design and Methods.*** *Per capita alcohol consumption was proxied by Systembolaget's alcohol sales (litres 100% alcohol per capita 15+). Quarterly data on mortality and alcohol consumption spanned the period 1991Q1-2017Q4. We used two outcomes: (i) alcohol-specific mortality (deaths with an explicit alcohol diagnosis); and (ii) violent deaths. SES was measured by education. We used three educational groups: (i) low (<10 years); (ii): intermediate (10–12 years); and (iii) high (13+ years). We applied error correction modelling to estimate the association between alcohol and alcohol-specific mortality, and seasonal autoregressive integrated moving average-modelling to estimate the association between alcohol and violent deaths.* ***Results.*** *The estimated associations between per capita consumption and the two outcomes were positive and statistically significant in the two groups with low and intermediate education, but not in the high education group. There was a significant gradient in the level of association between alcohol consumption and alcohol-related harm by educational group; the association was stronger the lower the educational group.* ***Discussion and Conclusions.*** *Our findings suggest that the association between per capita consumption and alcohol-related harm was stronger the lower the educational group.* [Norström T, Landberg J. The link between per capita alcohol consumption and alcohol-related harm in educational groups. *Drug Alcohol Rev* 2020]

Key words: alcohol, mortality, time-series, Sweden, education.

Introduction

Numerous studies have substantiated a link between per capita alcohol consumption and various forms of alcohol-related harm (for reviews, see references [1,2]). These studies are typically in the form of time-series analyses that estimate the effect of a 1-l increase in total consumption on a specific outcome. However, there is a dearth of studies of this kind that analyse SES-specific harm rates as outcome. The aim of this study is to address this knowledge gap by using Swedish data to estimate whether the association between per capita alcohol consumption and alcohol-related mortality differs across educational groups.

According to the Global Burden of Disease Study 2016, alcohol use was the seventh leading risk factor for mortality and disability-adjusted life years [3]. There is a large body of research documenting a strong link between various forms of alcohol-related harm rates and per capita alcohol consumption (for reviews, see references [1,2]). A recent study, based on Swedish quarterly data spanning the period 1987–2015, showed a positive and significant association between per capita consumption and the five outcomes that were in focus (cirrhosis mortality, fatal injuries, suicide, drink driving and assaults) [4]. Another recent study [5], relying on Finnish annual data covering the period 1975–2015, revealed a significant connection between per capita alcohol consumption and alcohol-specific deaths (i.e. deaths which are 100% attributable to alcohol). The relation between per capita alcohol consumption and a large number of outcomes thus seems to be well substantiated in studies of the general population. However, an important issue is whether the relation is valid also across various sub-

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Received 10 December 2019; accepted for publication 3 May 2020.

populations, for example with different socioeconomic status (SES)—which is the focus of the present study. One reason for why this cannot be taken for granted is that SES-groups may differ in their vulnerability to alcohol (usually referred to as differential vulnerability). This refers to the notion that lower SES-groups tend to be more susceptible to the negative effects of alcohol and builds on the alcohol harm paradox, that is the observation that less privileged groups experience a disproportionally large share of alcohol-related harm, although they tend to drink less, or at least not more, than other social strata [6–9]. Two recent studies have tested the relationship between SES and alcohol-related harm for differential vulnerability at the individual level. Based on data from a large prospective Scottish cohort, Katikireddi *et al.* [8] found evidence of effect modification between SES and alcohol use implying that the same level of consumption was associated with a larger risk of alcohol-related morbidity and mortality in low compared to high socioeconomic groups. Corresponding findings are reported by Christensen *et al.* [7] on the basis of prospective Danish data. Studies at the population level, which are of greater relevance in the present context, also suggest that changes in the availability of alcohol tend to affect low-SES groups more than high-SES groups. For instance, a Finnish study [10] assessed the mortality effect of the 10% increase in per capita alcohol consumption in 2004 that resulted from the 33% cut of alcohol excise taxes. The results showed that alcohol-related mortality increased most strongly in the less privileged groups ('…the unemployed or early-age pensioners and those with low education, social class, or income') [9, p. 1110]. Another example pertains to the mortality crisis in Russia in the late 20th century, which most likely was caused by a sharp increase in alcohol consumption [11,12]. The latter was in turn in all probability induced by the almost 80% drop in the vodka price between 1990 and 1994 [13]. An interesting finding in this context is that the mortality increases '…after 1990 have predominantly affected less-educated men and women, whereas the mortality of persons with university education has improved, resulting in a sharp increase in mortality differentials across educational levels' [14, p. 1293].

The findings reviewed above only provide scattered evidence of an inverse SES-gradient in the effect of per capita alcohol consumption on alcohol-related harm. The aim of this paper is therefore to perform a more comprehensive test of this notion. More specifically, we will use Swedish quarterly time-series data to estimate the association between per capita alcohol consumption and alcohol-related mortality in three educational groups. We focus on two outcomes: (i) mortality from alcohol-specific causes of death. This outcome mainly includes causes of death that are somatic consequences of chronic heavy drinking (e.g. alcoholic liver disease); and (ii) violent deaths including the following outcomes that have well-documented associations with per capita consumption: suicide [15], homicide [16] and injuries [17]. In order to obtain stable rates we computed a composite index of these causes of death (with the addition of undetermined intent). In an analysis of Norwegian time-series data, Skog [18] found a statistically significant relation between per capita consumption and a corresponding index.

Methods

Socioeconomic status

We used education as indicator of SES. Education, together with occupation and income, is one of the main dimensions for classifying SES and is often used in studies of trends in mortality disparities over time [19]. Education has the advantage of being more stable over time and is less likely to be influenced by reversed causation than occupation and income, i.e. that a person's occupation and/or income may be adversely affected by his/hers misuse of alcohol. We used three educational groups: (i) low education (no qualifications, primary years of schooling, 9 years or less); (ii) intermediate education (upper secondary school education, 10–12 years); and (iii) high education (college or university education, 13+ years).

Alcohol consumption

Per capita alcohol consumption was proxied by Systembolaget's sales expressed as litres of alcohol (100%) per inhabitant aged 15 years and above.

Outcomes

The analyses included two outcomes:

Alcohol-specific mortality. A composite index comprising deaths with an explicit alcohol diagnosis as underlying or contributory cause of death.

Violent deaths. A composite index comprising suicide, drowning injuries, fall injuries, motor-vehicle traffic crashes, homicide and undetermined.

International Classification of Diseases (ICD) codes for the causes of death included in each outcome are listed in Table 1.

The mortality data were obtained from the National Board of Health and Welfare (Socialstyrelsen). Information on education level was linked (by Socialstyrelsen) from the Swedish Register of Education (*Utbildningsregistret*) through personal identification numbers.

We constructed age-standardised mortality rates per 100 000 in the age group 25–79 years for the three educational groups. We chose the lower age limit (25 years) because the highest level of education has normally been attained at that age. The upper age limit was motivated by the finding that the accuracy of the cause-of-death classification is poorer in higher ages [20]. All data are quarterly and span the period 1991Q1-2017Q4.

Full ethical approval was obtained from Swedish Ethical Review Authority (nr. 2017/893-31/5). The study was not preregistered, and the results should thus be regarded as exploratory.

Statistical analyses

Previous research suggests that the relation between per capita consumption and chronic outcomes (which make up the bulk of alcohol-specific mortality) includes a lag-structure, that is a part of the effect of per capita alcohol consumption on the outcome (henceforth, alcohol effect) is distributed over a longer period of time [4,21]. To accommodate this phenomenon we used error correction modelling, which is a feasible approach when the aim is to estimate short- as well as long-term effects [22]. A brief account of an empirical application should convey the basic feature of the method. It is well documented that an increase in gross domestic product/capita tends to yield an increase in traffic fatalities, likely because of the increase in road traffic due to an expansion in the economy. However, a plausible hypothesis is that there is a long-term effect of increased gross domestic product that goes in the opposite direction; that is, that economic growth creates resources for safer roads, safer vehicles and better medical care, all of which should have a decreasing impact on traffic fatality rates. A study that performed error correction modelling of time-series data for 18 countries from the Organisation for Economic Co-operation and Development gave empirical support to this notion [23]. The results thus indicated that an increase in gross domestic product/capita indeed was associated with an instantaneous short-term increase in traffic fatalities, which however was outweighed by a stronger protective long-term effect. Error correction modelling is a standard modelling tool in economics; however, it has also been applied in alcohol epidemiology, more specifically in a study estimating the relation between cirrhosis

Table 1. *Causes of death*

	ICD-9	ICD-
Alcohol-specificmortality		
Alcohol psychosis	291	
Alcohol dependence	303	
Alcohol abuse	305.0	
Alcoholic polyneuropathy	357.5	G62.1
Alcoholic cardiomyopathy	425.5	I42.6
Alcohol gastritis	535.3	K29.2
Alcoholic liver disease	571.0–571.3	K70
Alcohol poisoning	E860, E980 + 980	
Alcohol-induced pseudo-Cushing's syndrome		E24.4
Mental and behavioural disorders due to use of alcohol		F10
Degeneration of nervous system due to alcohol		G31.2
Alcoholic myopathy		G72.1
Alcohol-induced chronic pancreatitis		K86.0
Maternal care for (suspected) damage to foetus from alcohol		O35.4
Toxic effect of alcohol		T51
Evidence of alcohol involvement determined by blood alcohol level		Y90
Evidence of alcohol involvement determined by level of intoxication.		Y91
Violent deaths		
Suicide	E950-E959	X60-X84
Homicide	E960-E969	X85-Y09, Y87.1
Drowning injuries	E910	W65-W74
Fall injuries	E880-E888, E848	W00-W19
Fire injuries	E890-E899	X00-X09
Motor-vehicle traffic crashes	E810-E819	V02-V04, V12-V14, V20-V79, V89.2
Undetermined intent	E980–E989	Y10–Y34,Y87.2, Y89.9

ICD, International Classification of Diseases.

mortality and per capita consumption in Sweden [4]. Following standard specification [22] our error correction model looks as follows:

$$\Delta\text{Mortality}_{it} = \alpha + \beta_0 \Delta\text{Alcohol}_t + \beta_1 \text{Mortality}_{it-1} + \beta_2 \text{Alcohol}_{t-1} + \varepsilon_t \quad (1)$$

where Mortality_{it} is the mortality rate for education group i and Alcohol is per capita alcohol consumption (proxied by alcohol sales). The operator Δ signifies that the series is differenced. The parameter β_0 indicates the instantaneous, short-term effect of a change in alcohol consumption on mortality, while β_1 estimates the speed at which the long-term effect operates. If such an effect does exist, the estimate of β_1 should be negative and statistically significant. The model assumes that the long-term effect decays geometrically; thus $1 - (-1 \times \beta_1)$ corresponds to the lag parameter in a lag scheme with geometrically declining lag weights. The total long-term effect is calculated as $\beta_2/(-1 \times \beta_1)$. We used the Bewley transformation regression [22,24] to estimate SE and significance level of the estimated long-term effect.

Before an error correction modelling is performed it is necessary to carry out some key tests of whether the conditions for error correction modelling are fulfilled. These analyses comprised two steps; first, we tested for unit root (non-stationarity) using the augmented Dickey and Fuller test [25]. If the time-series to be analysed (i.e. per capita alcohol consumption and alcohol-specific mortality) prove to be integrated of the order I (1), the next step is to test whether they are co-integrated; that is, if there exists a linear combination of X and Y that is stationary (trend-free) around which the two series fluctuate [26]. Put simply, if two time-series are co-integrated they will not drift far away from each other in the longer term. To test for co-integration we used Johansen trace statistic method [27].

Because no lag-effect is expected in the relation between per capita consumption and violent deaths, the following simple model was applied:

$$\Delta\text{Mortality}_{it} = \beta \Delta\text{Alcohol}_t + e_{it} \quad (2)$$

Possible effects of the introduction of ICD, 10th edition (1997) were captured by a dummy variable, taking the value 0 prior to 1997Q1, and 1 otherwise.

The relation between per capita consumption and violent deaths was analysed by applying the technique of seasonal autoregressive integrated moving average-modelling [28]. Non-stationarity in the form of time trends was removed by regular or seasonal differencing. The noise (error) term, which includes explanatory variables not considered in the model, is allowed to have a temporal structure that is modelled and estimated in terms of regular and seasonal autoregressive or moving average parameters. A seasonal autoregressive integrated moving average-model is specified as: (p, d, q) (P, D, Q, M), where the first bracket represents the model's non-seasonal (regular) part, and the second bracket specifies the seasonal part. The order of the autoregressive parameter in the model's non-seasonal part is indicated by p, while d indicates the order of regular differencing, and q is the order of the moving-average parameter. The symbols in the second bracket have the corresponding seasonal significance, while M is the number of observations per year. The model residuals should not differ from white noise; this was tested using the Box-Ljung Q statistics. All statistical analyses were performed with Stata V.14 (StataCorp, College Station, TX, USA).

Results

Trends in the two SES-specific outcomes and in per capita consumption are displayed in Figure 1. The SES-gradient is especially strong for alcohol-specific mortality; the ratio between the low- and high-educational group is 5.984 (average for the whole period, Table 2). The corresponding number for violent deaths is 2.686.

We start with the analyses of the relation between alcohol and alcohol-specific mortality. The results from the Dickey–Fuller tests (Table S1, Supporting Information) indicated that all series were integrated of the first order. On this premise, we tested for co-integration. The trace statistic test indicated that the null hypothesis of no co-integration could be rejected for all SES-groups (Table S2). We thus proceeded to the error correction modelling. The outcome indicated that the instantaneous effects were non-significant, whereas the long-term effects (Table 3) were statistically significant in the two groups with low and intermediate education, but not in the high-education group. The estimates express the change in the mortality rate associated with a 1-l increase in per capita consumption. For instance, a 1-l increase in per capita consumption would yield 4.675 additional deaths per 100 000 in the low education group, while the corresponding number for the group with intermediate education is 3.458 (the estimate for the high education group was insignificant). These estimates express the total effect, that is, including the lagged effects. The estimated lag parameter (β_1 in Equation 1) was −0.394 for the low education group and −0.494 for the group with intermediate education. This means that for the first group about 87% of the total effect was realised within a year (four quarters), and 98% within 2 years. The corresponding figures for the group with intermediate education were 93% and 99%, respectively. Moreover, there was a significant gradient in the level of

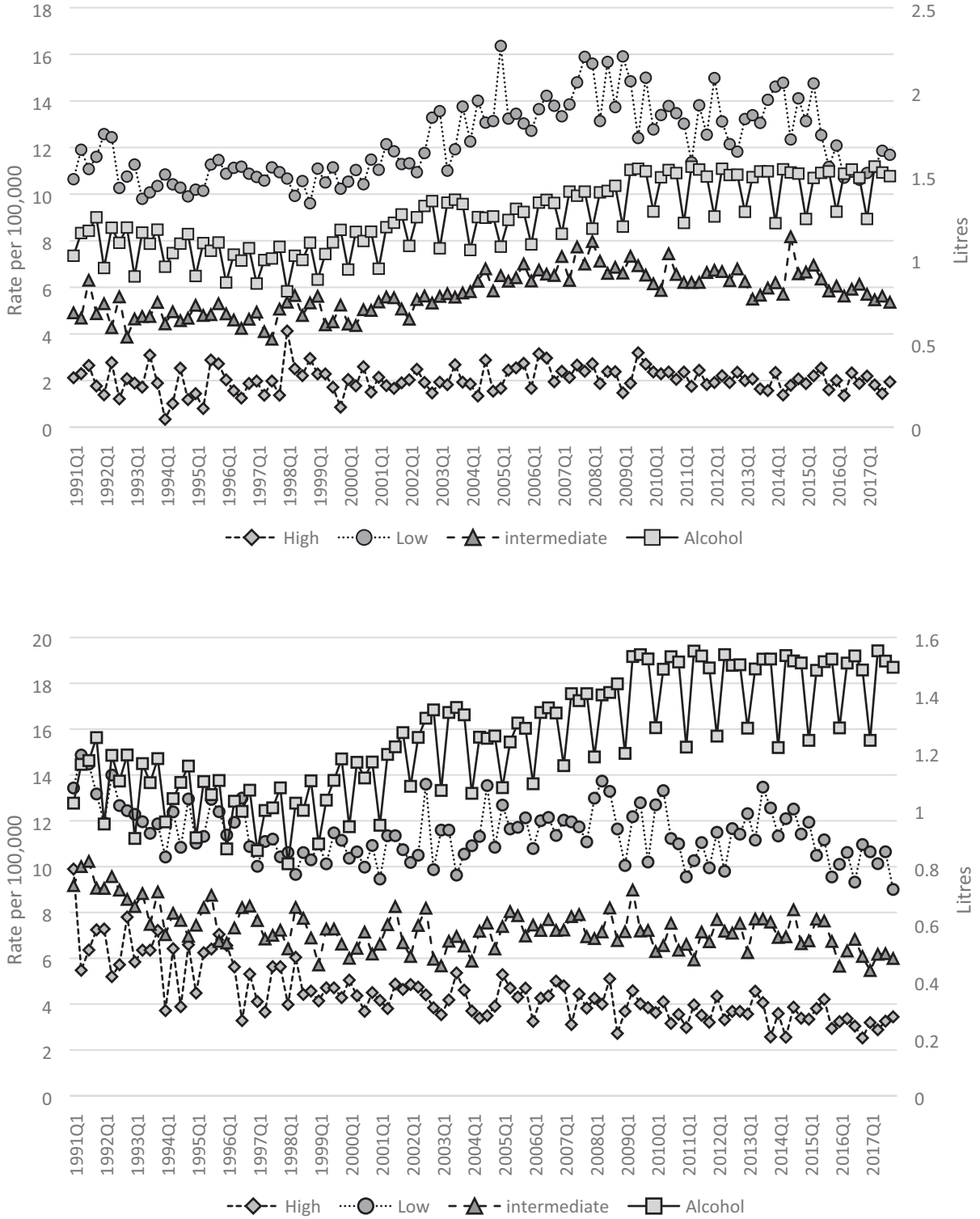

Figure 1. *Trends in per capita alcohol consumption and alcohol-specific mortality in three educational groups (a), and per capita alcohol consumption and violent deaths in three educational groups (b).*

association between per capita consumption and mortality; that is, the association was weaker the higher the educational group (F-test, $P < 0.001$). The analyses of the relation between per capita consumption and violent deaths revealed the same pattern as above; that is, statistically significant effect estimates in the two lowest

Table 2. *Descriptive statistics*

	Alcohol-specific mortality		Violent deaths	
	Mean	SD	Mean	SD
Education				
Low	12.225	1.620	9.993	1.117
Intermediate	5.741	0.929	6.136	0.751
High	2.043	0.562	3.721	0.916

educational groups, but not in the in the high education group. Here, too, the gradient in the effect estimates was significant, with a stronger effect for the group with low education (*F*-test, $P = 0.031$).

We also performed gender-specific analyses (Tables S3 and S4). By and large, the outcome was the same as reported above, with stronger alcohol effects the lower the education. However, the gradient in the estimated effects on violent deaths was not statistically significant for females. The residuals from all estimated models were white noise.

The dummy variable capturing the possible impact of the introduction of ICD-10th edition was far from statistical significance in all estimated models.

Discussion

Numerous studies suggest the presence of The Alcohol Harm Paradox [6–9], that is, that the same amount of alcohol gives rise to more harm in low-SES groups than in high-SES groups. However, to our knowledge, no study has examined whether a corresponding phenomenon exists also at the population level. Our findings, based on Swedish quarterly data, spanning the period 1991–2017, suggest that this is indeed the case. We found a statistically significant SES-gradient in the alcohol effect on alcohol-specific mortality and violent deaths; that is, a 1-l increase in per capita consumption was associated with more alcohol-related deaths (in absolute terms) in the low-educated group than in the high-educated group.

Below we discuss two, not mutually exclusive, explanations of this pattern of findings: (i) differential relations between consumption level and risk of harm; and (ii) differential consumption changes. The first explanation may reflect differences in drinking patterns or differences in vulnerability to alcohol across SES-groups at the individual level. Previous research does give some support to the notion that that low-SES groups would have more detrimental drinking habits than high-SES groups [29,30], but there seems to be marked variations across countries in this respect [31]. Analyses of Swedish data on SES-specific drinking patterns are thus a topic for future research. Although there is increasing evidence of differential vulnerability to alcohol across SES-groups, there is less knowledge of the factors and pathways underlying the disproportionate impact that alcohol have on more disadvantaged groups. Future research on the Alcohol Harm Paradox should thus focus on identifying contributing factors in this context. The other potential explanation implies that changes in consumption are

Table 3. *Estimated effects on harm rates of per capita alcohol consumption (litres 100%). Based on error correction modelling (alcohol-specific mortality) and seasonal autoregressive integrated moving average-modelling (violent deaths) of quarterly data 1991Q1-2017Q4. Whole population*

		Short-term effect			Long-term effect					
Outcome	Education	EST	SE	*P*	EST	SE	*P*	Q[a]	p(Q)	
Alcohol-specific mortality	Low	−0.127	0.752	0.865	4.675	0.683	<0.001	8.127	0.087	
	Intermediate	0.494	0.423	0.243	3.458	0.362	<0.001	7.817	0.099	
	High	−0.052	0.337	0.877	0.324	0.323	0.317	0.975	0.913	
F-test for heterogeneity		0.228		0.796	11.052		<0.001			Model
Violent deaths	Low	5.574	1.647	0.001				9.013	0.061	(1,0,0) (0,1,1,4)
	Intermediate	2.618	0.976	0.007				5.907	0.206	(1,0,0) (0,1,1,4)
	High	1.493	1.211	0.218				0.542	0.969	(1,0,0) (2,1,0,4)
F-test for heterogeneity		3.478		0.031						

[a]Box-Ljung test for residual autocorrelation (lag 4).

not synchronised across SES-groups, so that an increase in over-all consumption is disproportionally allocated to low-SES groups. This is clearly at variance with Skog's theory of the collectivity of drinking, which predicts that changes in over-all consumption take the form of shifts across the whole population due to social processes of contagion; that the individual drinker is influenced by the drinking behaviour of others [32]. However, as pointed out by Skog himself [33], exceptions to the rule of collectivity are certainly expected to occur. For instance, social barriers may hamper interaction between various socioeconomic groups, and thereby the overall synchronising process, resulting in non-parallel drinking trajectories across SES-groups. Another circumstance that may give rise to disparities in SES-specific drinking trajectories is if factors affecting consumption (e.g. real wages) evolve differently across socioeconomic groups [34,35]. However, little is known about how SES-specific drinking has evolved in Sweden during the last decades; this is thus an urgent task for future research.

Strengths and limitations

An obvious strength of our study is the use of time-series data on alcohol-related mortality for various educational groups, which made it possible to estimate SES-specific effects of per capita alcohol consumption on alcohol-related harm. A potential limitation in these analyses is that only Systembolaget's sales were used as a measure of alcohol consumption. However, a substantial portion of total consumption in Sweden comprises unrecorded alcohol (mainly travel imports). Lacking quarterly data on unrecorded alcohol consumption, we could not control for this factor by including it in our models. This implies that our estimated alcohol effects might be biased upwards (if there is a positive correlation between recorded and unrecorded alcohol consumption) or downwards (if the correlation between recorded and unrecorded consumption is negative). In this context it can be noted that there seems to be a positive SES-gradient in the consumption of alcohol brought in from abroad, which comprises the largest share of unrecorded alcohol [36]. Our definition of alcohol-specific mortality, that is including contributory in addition to underlying causes, is far from evident. An advantage of this approach is that it yields more cases, thereby more stable rates and counters the risk of under-estimating the prevalence of alcohol-specific mortality [37]. However, notwithstanding that the approach has been applied in several studies, for example [10] a potential limitation is the risk that there might be a time-trend in the propensity to include any contributory causes in the death certificate; this would create spurious trends in the mortality rates.

Conclusion

The research findings that alcohol-related harm rates are affected by changes in total alcohol consumption have provided a strong argument for maintaining a restrictive alcohol policy. However, for this notion to be viable, it needs to be refined and supplemented on the basis of new experiences. For instance, the findings from the European Comparative Alcohol Study showed that although per capita alcohol consumption 'stands out as a crucial determinant of alcohol related harm, its impact appears to be amplified or mitigated depending on the drinking culture and its drinking patterns' [38, p. 162]. The present study adds a socioeconomic dimension to our understanding of the population level association between alcohol and related harm by revealing a social gradient in the alcohol effect implying that an increase in per capita consumption is associated with a larger increase in alcohol-related mortality in the low-educated groups than in the high-educated group.

Moreover, we found a substantial social gradient in alcohol-specific mortality, with rates that were around six times higher in the low compared to the high-educational group. This finding is line with previous research showing that the social gradient in alcohol-attributable mortality tends to be larger than for other major health outcomes, for example all-cause mortality [39]. Considering the increased emphasis on decreasing social inequality in ill-health, including alcohol-related harm, it becomes increasingly important to explore which alcohol policy measures are effective for reducing or preventing social inequalities. Our findings indicate that population-based policy measures that effectively regulate per capita alcohol consumption, not only will result in decreased rates of alcohol-related harm, but also have the potential to narrow the large social inequalities of this outcome.

Acknowledgements

This study was funded by the Swedish Research Council for Health, Working life and Welfare (Forte) (grant number 2017-01769) and Systembolaget's Research Council (Systembolagets Alkoholforskningsråd) (grant number FO2017-0084).

Conflict of Interest

The authors have no conflicts of interest.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Table S1. Augmented Dickey–Fuller Test (ADF) for unit root.

Table S2. Trace statistic tests for cointegration among alcohol-specific mortality and per capita alcohol consumption.

Table S3. Estimated effects on harm rates of per capita alcohol consumption (litres 100%). Based on error correction modelling (alcohol-specific mortality) and seasonal autoregressive integrated moving average-modelling (violent deaths) of quarterly data 1991Q1-2017Q4. Females.

Table S4. Estimated effects on harm rates of per capita alcohol consumption (litres 100%). Based on error correction modelling (alcohol-specific mortality) and seasonal autoregressive integrated moving average-modelling (violent deaths) of quarterly data 1991Q1-2017Q4. Males.