Role of socioeconomic status in modifying the relationship between alcohol use and injury events: evidence from the UK Biobank

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ABSTRACT

Background Alcohol consumption is a well-established risk factor for injuries, yet the role of socioeconomic status (SES) in modifying this relationship remains underexplored. This study examines how SES influences the association between alcohol use and injury events, including both fatal and non-fatal injuries as well as the risk of repeated injuries requiring hospitalisation.

Methods Using data from the UK Biobank, we applied Cox proportional hazards models to assess whether SES modifies the relationship between different dimensions of alcohol use, average level and heavy episodic drinking (HED), and injury events. For injuries requiring hospitalisation, we fitted negative binomial regression models to estimate incidence rate ratios (IRRs) for the total number of unique injury admissions per individual.

Results Among 482 078 participants, 48 045 injuries were recorded during follow-up. A statistically significant interaction was observed between alcohol use and low education, with individuals in lower education groups showing elevated injury risk compared with the high education group, particularly among those consuming 20–40 g/day with HED (HR: 1.17, 95% CI 1.06 to 1.28) and those consuming over 40 g/day (HR: 1.13, 95% CI 1.04 to 1.23). For non-fatal injuries, similar statistically significant interactions were found (IRR 1.17, 95% CI 1.06 to 1.28; and 1.18, 95% CI 1.08 to 1.29, respectively).

Conclusion Our findings highlight that individuals with lower SES face higher risks of alcohol-related injuries. Future research should investigate specific mechanisms underlying these disparities to inform more targeted policies. Population-based pricing policies could be combined with community-based programmes and tailored health messaging to address SES-related vulnerabilities.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Previous research has studied the associations between alcohol consumption and injury risk as well as the independent effects of socioeconomic status (SES) on injury events. Alcohol use is a well-established risk factor for both intentional and unintentional injuries, and individuals from lower socioeconomic backgrounds often experience higher rates of injury due to a range of structural and environmental factors. However, the potential for SES to act as a modifier in the relationship between alcohol use and injury events remains understudied.

WHAT THIS STUDY ADDS

⇒ Our study is the first UK-based investigation to examine whether SES modifies the association between alcohol use and injury events across both fatal and non-fatal injuries. It uniquely accounts for repeated injury-related hospitalisations within individuals, an important methodological consideration often overlooked in previous research. Given the well-established link between acute alcohol use and injury risk, the inclusion of heavy episodic drinking alongside average alcohol consumption provides a more comprehensive understanding of alcohol-related injury vulnerability.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Our findings highlight socioeconomic differences in alcohol-related injury events, suggesting that individuals with lower SES may be more vulnerable to the harms of alcohol use. Future research should investigate mechanisms such as differential access to emergency care, variations in drinking contexts and exposure to high-risk environments. These findings also support the implementation of targeted, evidence-based policies to help reduce alcohol-related injuries and improve health outcomes across SES groups.

INTRODUCTION

Alcohol consumption remains a significant public health concern in the United Kingdom (UK), with substantial implications for injury events.[1] In 2021, injuries in the UK accounted for over 21 000 deaths and resulted in more than 1.2 million disability-adjusted life years.[2] Looking at motor vehicle injuries alone, there were approximately 6800 reported injury events involving at least one driver over the drink-drive limit (ie, 80 mg of alcohol per 100 mL of blood) in 2022 in Great Britain.[3] In 2023/2024, 39% of all violent crimes in England and Wales involved victims who believed the offender(s) were under the influence of alcohol.[4] Research has shown that acute alcohol consumption increases the risk of both unintentional and intentional injuries in a dose–response manner, with higher intake associated with a greater likelihood of harm.[5 6] Moreover, alcohol-related injuries contribute to long-term disability and mortality while placing a significant strain on healthcare resources.[7] Therefore, understanding the impact of alcohol consumption on injury risk is crucial for developing effective prevention strategies and policies to reduce the burden of disease and reduce the number of preventable deaths.[8] An important aspect of studying alcohol-related injuries is accounting for acute consumption patterns,[9 10] as average daily intake does not fully reflect the risks associated with heavy episodic drinking (HED, defined as consuming more than 8 units in a single session for males or more than 6 for females), which is strongly linked to impaired judgement, reduced motor coordination and increased engagement in high-risk behaviours that elevate injury risk.[11]

An additional layer of complexity in understanding the relationship between alcohol use and injuries lies in the influence of socioeconomic status (SES) in this relationship. On average, lower SES is associated with poorer health outcomes, including alcohol-related harm. Paradoxically, research has shown that individuals with lower SES suffer disproportionately higher alcohol-related harm despite similar or lower levels of alcohol use than those with higher SES.[12–14] A potential explanation for this paradox is that individuals with a lower SES may be more likely to consume alcohol in settings that are associated with higher risks of violence and injury, such as public places, bars or outdoor spaces.[15 16] While these patterns could contribute to disparities in alcohol-related harm, the underlying mechanisms remain insufficiently investigated. Combining such factors with limited access to healthcare and other resources may amplify the adverse effects of alcohol consumption in certain populations.

At the individual level, to the best of our knowledge, only two studies based on US data have assessed the SES effect modification on fatal injuries.[17 18] For fatal motor vehicle injuries and suicide, no effect modification was found. However, it was found that individuals with low SES, measured by educational attainment and family income, and frequent HED may exhibit a significantly higher risk of mortality from other unintentional injuries compared with their high SES counterparts.[17] Furthermore, in a Scottish study that analysed over 22 000 hospital cases of alcohol-related facial injuries, a common form of intentional injury, it was found that individuals from the most deprived areas were nearly seven times more likely to sustain such injuries compared with those from wealthier areas.[19] To date, no UK-based study has examined the effect modification of SES across injury events (both fatal and non-fatal) while also accounting for repeated injury-related hospitalisations within individuals. While intentional and unintentional injuries differ in their underlying mechanisms, the available hospitalisation data did not allow for this distinction; therefore, all injury-related admissions were included. Using UK Biobank (UKB) data, this study aimed to (1) assess the relationship between different dimensions of alcohol use, average level and HED, and injury events and (2) investigate whether SES modifies this relationship.

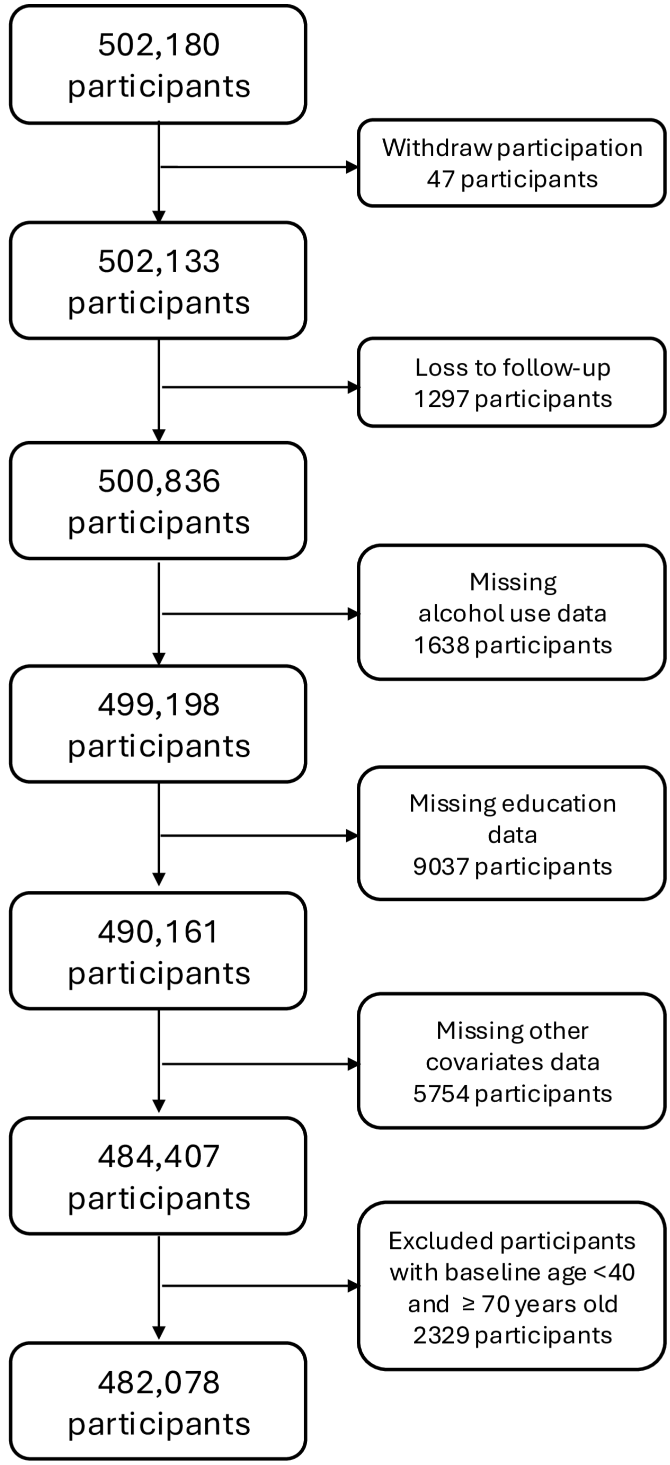

Figure 1 Selection of study participants.

METHODS

Data source and study population

The UKB is a large-scale biomedical database containing deidentified demographic, lifestyle and health data.[20] The baseline assessment for this community cohort study took place between May 2006 and July 2010 across 22 UKB assessment centres throughout the UK. Adults aged 40–69 years who lived within a 25-mile radius of any centre were invited to participate (approximately 9 million adults). Over 500 000 adults took part, resulting in a response rate of 5.5%. Baseline data have been linked to national death registries as well as hospitalisations covering admission dates, diagnoses, procedures and discharge details. Follow-up data include information on the date of mortality (up to 30 November 2022) and injury-related hospitalisations (up to 31 May 2022, for Wales; 31 August 2022, for Scotland; and 31 October 2022, for England). Participants were excluded from our study if they withdrew their participation (n=47), were lost to follow-up (n=1297), had incomplete data for our key covariates (n=16 429) or were outside of the 40-to-69-year age range (n=2329; figure 1). Additionally, the UKB distributed follow-up online questionnaires, with two of those assessing information about alcohol use in 2016 and 2022. Only a subset of participants responded to these questionnaires (n=157 235 and n=169 927, respectively).

Measures

Average daily alcohol use was derived from the self-reported monthly or weekly consumption of alcohol by beverage type at baseline. For each participant, the average grams of pure alcohol consumed per day (g/day) was calculated by dividing the number of grams of alcohol in each alcohol type reported (assuming there were 2 units (16 g) of pure alcohol in a pint of beer; 1.5 units (12 g) in a glass of red and white wine, and other alcoholic drinks; and 1 unit (8 g) in a measure of spirits and fortified wine)[21] by 30.44 for monthly reports and by 7 for weekly reports. Given that the current literature suggests HED is a stronger predictor of injury than overall alcohol consumption,[11] we accounted for this drinking pattern by imputing this variable using regression-based multiple imputation,[22] as HED was evaluated only in a subset of participants during the 2016 Mental Health Questionnaire (for definitions and detailed procedure, see online supplemental Materials Methods e1). The final alcohol use categories were lifetime abstainers and occasional drinkers (ie, past-year daily average of <1 g/day) with no HED (reference category), former drinkers (ie, past year abstention with a history of drinking), category I without HED (ie, past-year daily consumption of 1–20 g/day on average), category I and occasional drinkers with HED (ie, past-year daily consumption of 0–20 g/day on average who engage in HED), category II without HED (ie, past-year daily consumption of >20–40 g/day on average), category II with HED and category III (ie, past-year daily consumption of over 40 g/day on average).

SES was measured using educational attainment in the main analysis, and as household income in a sensitivity analysis. Participants were asked to report their qualifications, which were then categorised according to the International Standard Classification of Education (ISCED)[23]: low education (ISCED levels 1 or 2 corresponding to completion of primary education or lower secondary education, which is typically up to 9 years of education), medium education (ISCED levels 3 or 4 corresponding to completion of upper secondary or postsecondary education which is typically 10–13 years of education) and high education (reference category; ISCED level 5 or above corresponding to completion of tertiary education or equivalent professional qualifications). Household income was self-reported and categorised as: (1) high income: greater than £52 000 annually (reference), (2) middle income: £18 000–£51 999 and (3) low income: less than £18 000. Due to the high number of missing data for household income (13.7%), we treated participants with missing information as a separate category.

Fatal injuries were defined using the following International Classification of Diseases, 10th revision (ICD-10) codes as the underlying (primary) cause of death: X60-Y09, Y87.0 (ie, death by intentional injuries, including intentional self-harm—that is, suicide—and assault), V02-V04, V09.0, V09.2, V12-V14, V19.0-V19.2, V19.4-V19.6, V20-V79, V80.3-V80.5, V81.0-V81.1, V82.0-V82.1, V83-V86, V87.0-V87.8, V88.0-V88.8, V89.0, and V89.2 (ie, death by motor vehicle injuries); and V01-X59, Y40-Y86, Y88-Y89 (ie, other unintentional injuries), excluding: X40-X45, Y10-Y14, Y45, Y47, Y49 (ie, poisonings). The time to event was calculated as the years between the date of death and the baseline age for participants who died during the study period. For participants who were still alive at the time of censoring, the difference was calculated by subtracting the baseline assessment date from the censoring date.

Non-fatal injuries requiring hospitalisation were operationalised as the first-time in-patient hospital admission, identified by the distinct primary diagnosis codes recorded across all hospital inpatient records during follow-up. ICD-10 codes S00-S99 and T00-T98 were used to capture the underlying (primary) cause of admission, excluding codes T36-T65 (ie, poisoning) and T79-T88 (ie, early complications of trauma and complications of surgical and medical care). Additionally, during follow-up, participants could experience multiple episodes of non-fatal injuries; therefore, we also counted the total number of injury admissions recorded per individual. However, only the first occurrence of each distinct ICD code per patient was considered, with multiple hospitalisations for the same ICD code not being available.

Other variables that were included in the models were baseline age (continuous), sex (male (reference) and female), country (England (reference), Scotland and Wales, and ethnicity (white participants (reference) and others). The white category included British, Irish and any other white background. The Others category included 'mixed', 'Asian or Asian British', 'Black or Black British', 'Chinese' and 'Other ethnic group'.

Statistical analysis

Cox proportional hazards models with age as the timescale were used to evaluate whether SES modified the association between alcohol use and the risk of both fatal and non-fatal injuries requiring hospitalisation. Statistical interaction was assessed by including multiplicative interaction terms between alcohol use and SES.[24] We fit the models using packages *survival* and *survminer* in R software.[25]

For non-fatal injuries requiring hospitalisation, we analysed the potential overdispersion in the counts of hospitalisations per individual by calculating the variance-to-mean ratio, which was found to be greater than 1, indicating overdispersion. Consequently, we fit negative binomial models using the *MASS* package in R software.[26] Negative binomial regression is a generalisation of the Poisson regression model that includes an additional parameter, which models the overdispersion.[27] The results were reported as incidence rate ratios (IRRs). These models estimated the association between alcohol use and the rate of repeated injury hospitalisations. We tested for statistical interaction between alcohol use and education, via multiplicative term, to explore whether education modified this association.

All models were adjusted by baseline age, sex, country and ethnicity. The coefficients for the Cox proportional hazards model covariates were not provided due to concerns with "table 2 fallacy".[28] All statistical analyses were conducted in R, V.4.4.1.[29] A two-sided p value <0.05 was considered statistically significant.

Sensitivity analyses

Because the non-fatal injuries requiring hospitalisation data did not allow us to distinguish between intentional and unintentional injuries, we focused on fatal injuries for our first sensitivity analysis. We conducted Cox proportional hazard regression models to minimise potential bias that might arise from combining these distinct types of injuries in a single analysis. For our second sensitivity analysis, we used household income as an SES operationalisation to test the robustness of our findings. This decision followed the recommendations from Cubbin and Smith *et al*,[30] and Yuma-Guerrero *et al*[31] for injury research, which recommended using multiple SES indicators to provide a more

Table 1 Characteristics of study participants by education

		Education		
	Overall	Low	Medium	High
n	482 078	109 012	157 743	215 323
Sex (%)				
Male	219 139 (45.5)	49 954 (45.8)	64 132 (40.7)	105 053 (48.8)
Female	262 939 (54.5)	59 058 (54.2)	93 611 (59.3)	110 270 (51.2)
Baseline age (mean (SD))	56.5 (8.0)	58.7 (7.7)	55.7 (8.1)	55.9 (8.0)
Years follow-up (mean (SD))				
Mortality	13.4 (2.0)	13.2 (2.3)	13.4 (1.9)	13.5 (1.8)
Hospitalisations	13.1 (2.4)	13.0 (2.6)	13.1 (2.4)	13.1 (2.4)
Alcohol use (%)				
Mean grams per day (SD)	14.3 (17.3)	13.2 (18.3)	14.3 (17.3)	14.9 (16.8)
LA+Oc without HED	104 654 (21.7)	30 445 (27.9)	34 062 (21.6)	40 147 (18.6)
Former drinker	17 124 (3.6)	5609 (5.1)	4986 (3.2)	6529 (3.0)
Category I without HED	194 224 (40.3)	38 755 (35.6)	64 146 (40.7)	91 323 (42.4)
Category I with HED	39 352 (8.2)	8182 (7.5)	13 453 (8.5)	17 717 (8.2)
Category II without HED	48 118 (10.0)	9797 (9.0)	15 047 (9.5)	23 274 (10.8)
Category II with HED	41 983 (8.7)	7756 (7.1)	14 092 (8.9)	20 135 (9.4)
Category III	36 623 (7.6)	8468 (7.8)	11 957 (7.6)	16 198 (7.5)
Imputed HED (%)				
No HED	367 508 (76.2)	85 361 (78.3)	119 241 (75.6)	162 906 (75.7)
HED	114 570 (23.8)	23 651 (21.7)	38 502 (24.4)	52 417 (24.3)
Income (%)				
Low income	93 335 (19.4)	38 831 (35.6)	27 751 (17.6)	26 753 (12.4)
Medium income	214 727 (44.5)	39 005 (35.8)	79 120 (50.2)	96 602 (44.9)
High income	107 761 (22.4)	6209 (5.7)	30 318 (19.2)	71 234 (33.1)
Missing	66 255 (13.7)	24 967 (22.9)	20 554 (13.0)	20 734 (9.6)
Ethnicity (%)				
White participants	457 336 (94.9)	103 879 (95.3)	151 570 (96.1)	201 887 (93.8)
Other participants	24 742 (5.1)	5133 (4.7)	6173 (3.9)	13 436 (6.2)
Country (%)				
England	427 528 (88.7)	97 365 (89.3)	141 147 (89.5)	189 016 (87.8)
Wales	19 661 (4.1)	4403 (4.0)	6853 (4.3)	8405 (3.9)
Scotland	34 889 (7.2)	7244 (6.6)	9743 (6.2)	17 902 (8.3)

HED, heavy episodic drinking; LA, lifetime abstainers; Oc, occasional drinkers.

comprehensive analysis of the socioeconomic factors influencing injury events. In our third sensitivity analysis, we excluded all non-drinkers (both lifetime abstainers and former drinkers), using occasional drinkers without HED as the reference group, to avoid the potential bias of misclassification between drinkers and non-drinkers, ensuring that the analysis focused on the variability within the drinking population. Finally, to further explore the potential dose–response relationship, we conducted a sensitivity analysis using alcohol consumption as a continuous variable (increments of 8 g/day). This approach allowed us to assess whether risk increased with higher consumption and whether SES modified this association. HED was included in this model as a covariate.

RESULTS

Our cohort study consisted of 482 078 participants (219 139 males (45.5%) and 262 939 females (54.5%)) who were observed for a total of 6 168 423 person-years. Sample characteristics are shown in table 1. There were 48 045 injury events during the follow-up period, for a rate of 77.9 (95% CI 77.2 to 78.6) per 10 000 person-years.

In the model with no interaction term, we observed a higher risk of injury events among individuals with low education (HR 1.17, 95% CI 1.14 to 1.19) compared with those with high education. A higher risk of injury events was found among former drinkers and those with an average alcohol use exceeding 40 g/day, compared with lifetime abstainers and occasional drinkers without HED (table 2). A negative association was found among individuals in category I without HED (HR: 0.92, 95% CI 0.90 to 0.94). An increase in risk was observed with higher consumption, which varied by HED status, with individuals engaging in HED, showing higher risk across categories. A statistically significant positive interaction was observed between alcohol use and low education, with individuals in lower education groups showing elevated injury risk compared with the high education group, particularly among those consuming 20–40 g/day with HED (HR: 1.17, 95% CI 1.06 to 1.28) and those consuming over 40 g/day (HR: 1.13, 95% CI 1.04 to 1.23) (table 2).

A total of 47 485 non-fatal injuries requiring hospitalisation were recorded during follow-up. Table 3 presents results from the negative binomial regression model examining the association between alcohol use and the rate of repeated injury hospitalisations. In our main model, we observed that having a low or medium education level, being a white participant, being female and being of older age were all associated with a higher rate of non-fatal injuries requiring hospitalisation over the follow-up

Table 2 Adjusted HRs with 95% CIs for the association of alcohol use on both fatal and non-fatal injuries requiring hospitalisation, based on Cox regression models, and the effect modification between alcohol use and educational attainment

	Total N	Injury events	Main effect model		Model with interaction term	
			HR (95% CI)	P value	HR (95% CI)	P value
Educational attainment						
High education (ref)	215 323	20 017	1.00		1.00	
Medium education	157 743	15 133	1.02 (0.99 to 1.04)	0.11	1.02 (0.98 to 1.07)	0.32
Low education	109 012	12 895	1.17 (1.14 to 1.19)	<0.001	1.12 (1.07 to 1.18)	<0.001
Alcohol use						
LA+occasional drinker without HED (ref)	104 654	10 764	1.00		1.00	
Former drinker	17 124	2137	1.29 (1.23 to 1.35)	<0.001	1.27 (1.17 to 1.37)	<0.001
Category I without HED	194 224	18 355	0.92 (0.90 to 0.94)	<0.001	0.93 (0.89 to 0.97)	<0.001
Category I with HED	39 352	3392	0.97 (0.93 to 1.01)	0.11	0.95 (0.89 to 1.01)	0.11
Category II without HED	48 118	4922	1.00 (0.96 to 1.03)	0.90	0.98 (0.93 to 1.03)	0.39
Category II with HED	41 983	3942	1.04 (0.99 to 1.04)	0.06	0.98 (0.92 to 1.04)	0.49
Category III	36 623	4533	1.36 (1.31 to 1.41)	<0.001	1.30 (1.23 to 1.38)	<0.001
Interaction of alcohol use with educational attainment						
Former drinker: low education	5609	829			1.07 (0.96 to 1.19)	0.22
Category I without HED: low education	38 755	4331			1.01 (0.95 to 1.07)	0.85
Category I with HED: low education	8182	837			1.06 (0.96 to 1.17)	0.24
Category II without HED: low education	9797	1159			1.05 (0.96 to 1.14)	0.27
Category II with HED: low education	7756	909			1.17 (1.06 to 1.28)	<0.001
Category III: low education	8468	1237			1.13 (1.04 to 1.23)	<0.05
Former drinker: medium education	4986	571			0.97 (0.86, 1.09)	0.57
Category I without HED: medium education	64 146	5810			0.96 (0.91 to 1.02)	0.20
Category I with HED: medium education	13 453	1122			1.00 (0.92 to 1.10)	0.96
Category II without HED: medium education	15 047	1522			1.02 (0.94 to 1.11)	0.62
Category II with HED: medium education	14 092	1299			1.06 (0.97 to 1.15)	0.21
Category III: medium education	11 957	1413			1.03 (0.95 to 1.12)	0.53

Occasional drinker (<1 g/day), category I without HED (1–20 g/day), category I with HED (0–20 g/day), category II (20–40 g/day) and category III (>40 g/day).
HED, heavy episodic drinking; LA, lifetime abstainers; N, counts.

period. Regardless of HED, individuals who consumed less than 20 g/day on average showed a negative association, while those with category III alcohol use showed a higher rate of non-fatal injuries requiring hospitalisation (IRR 1.39, 95% CI 1.34 to 1.45). Similar significant results to those observed in the Cox model were found: individuals with low education who consumed 20–40 g/day with HED (IRR 1.17, 95% CI 1.06 to 1.28) and those consuming over 40 g/day (IRR 1.18, 95% CI 1.08 to 1.29) had higher injury rates compared with individuals with high education at the same levels of alcohol use (table 3 and figure 2). As shown in figure 2, a gradient was observed among individuals with low education, indicating a higher rate of non-fatal injuries requiring hospitalisation across various levels of alcohol use compared with their counterparts with high education.

Our dataset included a total of 293 deaths by intentional injuries (including suicide and violence) and 593 deaths by unintentional injuries (66 motor vehicle injuries, 298 falls and 164 other unintentional injuries). In our first sensitivity analysis, for fatal intentional injuries (online supplemental Materials Table e2), a statistically significant interaction was identified between education levels, with individuals in category II with HED and low education showing a higher risk of fatal intentional injuries compared with those with high education (HR: 6.14, 95% CI 1.88 to 19.98). For fatal unintentional injury (online supplemental Materials Table e3), we observed statistically significant interactions between individuals with low (HR: 0.42, 95% CI 0.20 to 0.89) and medium (HR: 0.45, 95% CI 0.21 to 0.97) education and alcohol consumption of 20–40 g/day without HED compared with lifetime abstainers and occasional drinkers without HED with high education. This suggests that, at this level of consumption, individuals with high education have a higher risk of fatal unintentional injury compared with those with low or medium education. In our second sensitivity analysis, using household income as an SES indicator, we found similar results to those seen in our main model: a higher risk of both fatal and non-fatal injuries requiring hospitalisation among individuals with low and middle income compared with high income, and among individuals with an average alcohol use of more than 20 g/day, irrespective of HED. Similarly, a statistically significant interaction between higher alcohol use and low income was found (online supplemental Materials Table e4). Our results were confirmed in our third sensitivity analysis, which excluded non-drinkers (n excluded=37 839; for results, see online supplemental Materials Table e5). This analysis further validated the effect modification of education on the relationship between alcohol use and injuries, demonstrating the robustness of our findings. Finally, when alcohol consumption was modelled continuously (per 8 g/day increments), higher consumption was associated with increased injury risk (HR 1.04, 95% CI 1.03 to 1.05). The interaction terms indicated steeper dose–response slopes in lower SES strata (medium education: HR 1.01, 95% CI 1.00 to 1.02; p = 0.035; low education: HR 1.02, 95% CI 1.01 to 1.03; p = 0.001) compared with lifetime abstainers and high education.

Table 3 Incidence rate ratios with 95% CIs from negative binomial regression for non-fatal injuries requiring hospitalisation

	Total N	Events	Main effect model		Model with interaction term	
			IRR (95% CI)	P value	IRR (95% CI)	P value
Intercept			0.02 (0.01 to 0.02)		0.02 (0.01 to 0.02)	
High education (ref)	215 323	19 781	1.00		1.00	
Medium education	157 743	14 956	1.02 (1.00 to 1.05)	<0.05	1.03 (0.98 to 1.08)	0.19
Low education	109 012	12 748	1.21 (1.18 to 1.24)	<0.001	1.16 (1.11 to 1.22)	<0.001
Baseline age (continuous)	–	–	1.03 (1.03 to 1.03)	<0.001	1.03 (1.03 to 1.04)	<0.001
Male (ref)	219 139	20 488	1.00		1.00	
Female	262 939	26 997	1.15 (1.13 to 1.17)	<0.001	1.15 (1.13 to 1.17)	<0.001
England (ref)	427 528	42 050	1.00		1.00	
Scotland	34 889	3860	1.12 (1.08 to 1.16)	<0.001	1.12 (1.08 to 1.16)	<0.001
Wales	19 661	1575	0.77 (0.73 to 0.81)	<0.001	0.77 (0.73 to 0.81)	<0.001
White participants (ref)	457 336	45 968	1.00		1.00	
Other participants	24 742	1517	0.68 (0.65 to 0.72)	<0.001	0.68 (0.65 to 0.72)	<0.001
LA+occasional drinker without HED (Ref)	104 654	10 666	1.00		1.00	
Former drinker	17 124	2105	1.29 (1.23 to 1.35)	<0.001	1.30 (1.20 to 1.41)	<0.001
Category I without HED	194 224	18 163	0.89 (0.86 to 0.91)	<0.001	0.89 (0.86 to 0.93)	<0.001
Category I with HED	39 352	3347	0.96 (0.92 to 1.00)	<0.05	0.95 (0.89 to 1.01)	0.09
Category II without HED	48 118	4866	0.97 (0.94 to 1.01)	0.10	0.96 (0.91 to 1.01)	0.12
Category II with HED	41 983	3882	1.03 (0.99 to 1.07)	0.12	0.97 (0.92 to 1.03)	0.38
Category III	36 623	4456	1.39 (1.34 to 1.45)	<0.001	1.31 (1.23 to 1.39)	<0.001
Interaction of alcohol use with educational attainment						
Former drinker: low education	5609	822			1.05 (0.94 to 1.17)	0.42
Category I without HED: low education	38 755	4283			1.00 (0.94 to 1.06)	0.98
Category I with HED: low education	8182	830			1.04 (0.94 to 1.15)	0.45
Category II without HED: low education	9797	1146			1.05 (0.96 to 1.14)	0.33
Category II with HED: low education	7756	891			1.17 (1.06 to 1.28)	<0.05
Category III: low education	8468	1219			1.18 (1.08 to 1.29)	<0.001
Former drinker: medium education	4986	556			0.91 (0.80 to 1.03)	0.14
Category I without HED: medium education	64 146	5757			0.96 (0.91 to 1.02)	0.19
Category I with HED: medium education	13 453	1102			1.00 (0.91 to 1.10)	0.97
Category II without HED: Medium education	15 047	1508			1.00 (0.92 to 1.09)	0.97
Category II with HED: medium education	14 092	1279			1.06 (0.97 to 1.16)	0.18
Category III: medium education	11 957	1391			1.05 (0.96 to 1.14)	0.31

Occasional drinker (<1 g/day), category I without HED (1–20 g/day), category I with HED (0–20 g/day), category II (20–40 g/day) and category III (>40 g/day).
HED, heavy episodic drinking; IRR, incidence rate ratio; LA, lifetime abstainers; N, counts.

DISCUSSION

Our study found associations between alcohol use and injury events as well as in the rate of non-fatal injuries requiring hospitalisation. It also underscores the role of SES, analysed for both educational attainment and household income, in modifying the relationship between alcohol use and injury events. The data showed that increased daily alcohol consumption combined with HED was associated with significantly higher injury risks, particularly among individuals with lower SES compared with lifetime abstainers and occasional drinkers with higher SES.

Our findings on the increased risk of injury events identified for former drinkers compared with lifetime abstainers and occasional drinkers align with previous research.[18 32] Lange *et al* found that former drinkers had suicide mortality rates of more than 5 and 9 deaths per 100 000 person-years for females and males, respectively, and that men had an approximately 43% greater risk of suicide compared with lifetime abstainers.[18] Similarly, a study conducted in Japan found a 6.7-fold increased risk of suicide among male former drinkers compared with occasional drinkers.[32] The elevated risk among former drinkers may be attributed to underlying health conditions that led them to stop drinking and co-occurring mental health disorders.[33] It is plausible that this group includes individuals who were heavy drinkers or had alcohol use disorders and had stopped drinking at the time of the baseline assessment but may have relapsed later. Given that the primary mechanism linking alcohol use to injury risk is acute intoxication,[5 9] the elevated risk is more likely to reflect intermittent or resumed drinking rather than lasting effects from past use.

We found a lower risk of injury events for consuming less than 20 g/day without engaging in HED compared with lifetime abstention or occasional drinking. Research has shown that such negative association is unlikely, as even low-to-moderate acute alcohol consumption can still impair cognitive and motor functions, increasing the risk of injuries.[5 9] While our data suggest a negative association, it is possible that this finding may be influenced by certain biases in the way alcohol use and injury events are reported or measured, and should not be interpreted as evidence of benefit from low level drinking. For example, the negative association could be partly due to the disconnect in time between measuring exposure and outcome in our data, which did not allow to account for acute but rather usual drinking patterns. Moreover, the relationship between alcohol consumption and injury is complex and influenced by a variety

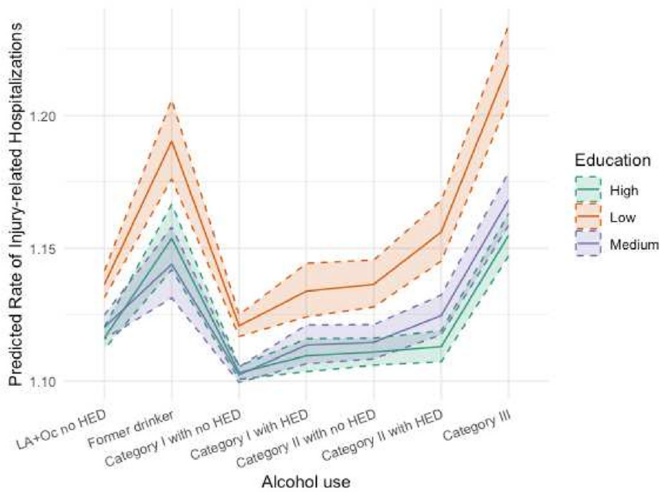

Figure 2 Predicted rate of non-fatal injuries requiring hospitalisation from negative binomial regression model with the interaction term of alcohol use and education. Reference: high education+lifetime abstainers and occasional drinkers with no HED. HED, heavy episodic drinking; LA, lifetime abstainers; Oc, occasional drinkers.

of factors, including individual susceptibility, drinking patterns and the setting in which alcohol is consumed.[7 11] Although HRs increased with higher alcohol consumption, the trend was modest, indicating that average volume alone may not fully explain injury risk. These considerations highlight the need for further investigation to better understand the nuances of alcohol-related injury risks.

Our study found that individuals consuming more than 40 g/day had both a significantly higher injury risk and an increased incidence rate of hospitalisation. Furthermore, when HED was present, the risk of injury events was strong among individuals with consumption of 20–40 g/day and low education, where a significant interaction was observed. These findings align with a substantial body of research showing that heavy alcohol consumption exacerbates the risk of both unintentional and intentional injuries.[9 11 34] The effect modification found suggests that individuals with lower SES in the UK may face barriers to accessing healthcare and specialised support services despite the availability of the NHS.[35] Factors such as longer wait times, reduced healthcare engagement and greater exposure to high-risk drinking environments may contribute to the disproportionate burden of alcohol-related injuries in this group.[36 37] Moreover, the gradient observed in non-fatal injuries requiring hospitalisation across different levels of alcohol use among those with low education compared with lifetime abstainers and occasional drinkers with high education points to a broader public health issue. To address this, policymakers can implement targeted awareness campaigns, regulate alcohol outlet density and introduce pricing policies such as increased alcohol taxes or minimum unit pricing to reduce consumption and mitigate alcohol-related harm.[38 39] Additionally, community-based interventions and local policy advocacy can provide social support and promote healthier behaviours.[40]

Our study has several strengths. First, it examines injury events, including both fatal and non-fatal injuries requiring hospitalisation, providing a comprehensive view of the relationship between alcohol use, injury events and SES. We conducted a specific analysis that accounts for multiple injuries during the follow-up period, making us the first study to address this aspect. By implementing a negative binomial regression, we were able to capture the overdispersion in the hospitalisation data and offer a valid estimate of the impact of alcohol use on injury morbidity. Additionally, we incorporated a second SES indicator in our sensitivity analysis, enhancing the robustness of our findings.[30 31] By accounting for HED status, we not only considered average alcohol consumption but also an important drinking pattern that influences injury risk. Furthermore, our deliberate selection of the reference category might have reduced classification bias within the study.

Several limitations should be acknowledged. First, our study used self-reported data, which could result in an under-reporting of alcohol use. This potential under-reporting is likely consistent across different SES groups, and, therefore, it is not expected to significantly affect the observed effect modification.[41] Second, we do not have the actual count of non-fatal injuries requiring hospitalisation per individual, as the records are based on unique types of injuries. For instance, if an individual had multiple admissions for the same ICD-10 code during the follow-up period, it was only recorded once (the first time), which may lead to an underestimation of the true effect. However, we have accounted for multiple admissions for different types of injuries. Additionally, our data on non-fatal injuries did not allow for a distinction between intentional and unintentional injuries, which might involve different causal pathways. Future studies should incorporate methods to distinguish between these types of injuries, possibly by including additional variables when constructing the cohort and follow-up questionnaires. In the mortality analysis, although we were able to differentiate between intentional and unintentional injuries, we could not further disaggregate unintentional injuries into subgroups such as motor vehicle injuries or falls due to the low number of deaths in each category. Our study also lacked sufficient power to conduct sex-specific analyses. We had to impute HED data and lacked frequency information for HED, limiting our analysis to determining whether individuals engaged in HED or not. Future studies should aim to collect detailed frequency data on HED to provide a more nuanced understanding of its impact. Furthermore, we could not account for time variations in alcohol use, as our analysis only considered baseline alcohol consumption. Finally, the cohort is not representative of the UK population, and the sample predominantly consists of white middle-aged and older adults, which limits the generalisability of our findings, particularly for younger populations. Drinking patterns and injury mechanisms can differ substantially across age groups. For example, older adults are more likely to experience falls resulting in hospitalisation,[42] whereas younger individuals may have higher rates of motor vehicle injuries.

In conclusion, this study provides valuable insights into how SES modifies the relationship between alcohol consumption and injury events. Our findings highlight that individuals with lower SES face disproportionately higher risks of alcohol-related injuries. Future research should investigate specific mechanisms underlying these disparities, such as differential access to healthcare or exposure to high-risk drinking settings. Evidence-based policies like alcohol taxation and minimum unit pricing have been shown to reduce alcohol-related harm and mortality differences by reducing affordability among high-risk groups. These measures, combined with targeted community-based programmes and tailored health messaging, can help address SES-related vulnerabilities, mitigate injury burden and promote health equity.

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Patient consent for publication Not applicable.

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