# Adverse childhood experiences and alcohol consumption in midlife and early old-age

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**Abstract -- Aims:** To examine the individual and cumulative effects of adverse childhood experiences (ACEs) on alcohol consumption in midlife and early old-age, and the role of ACEs in 10-year drinking trajectories across midlife. **Methods:** Data were from the Whitehall II study, a longitudinal British civil service-based cohort study (N=7870, 69.5% male). Multinomial logistic regression was used to examine the individual and cumulative effects of ACEs on weekly alcohol consumption. Mixed-effect multilevel modelling was used to explore the relationship between ACEs and change in alcohol consumption longitudinally. **Results:** Participants who were exposed to parental arguments/fights in childhood were 1.24 (95%CI 1.06, 1.45) times more likely to drink at hazardous levels in midlife (mean age 56 years) after controlling for covariates and other ACEs. For each additional exposure to an ACE, the risk of hazardous drinking versus moderate drinking was increased by 1.12 (95%CI 1.03, 1.21) after adjusting for sex, age, adult socio-economic status, ethnicity and marital status. No associations between ACEs and increased risk of hazardous drinking in early old age (mean age 66 years) were found. In longitudinal analyses, ACEs did not significantly influence 10-year drinking trajectories across midlife. **Conclusion:** The effect of exposure to parental arguments on hazardous drinking persists into midlife.

## INTRODUCTION

Adverse childhood experiences (ACEs) and alcohol misuse have been recognized globally as key public health issues (WHO, 2006; WHO, 2010).

ACEs refer to stressful events which an individual experiences in childhood, such as physical abuse, parental divorce or parental alcoholism (WHO, 2015). In UK, a national household survey found that the prevalence of at least one ACE was 46.4% and 8.3% had experienced 4 or more ACEs (Bellis *et al*., 2014). Statistics on child maltreatment in the UK indicate that 1 in 14 children aged 11-17 (6.9%) report being physically abused by a parent or

guardian during childhood (Radford *et al.*, 2011). The number of children affected by parental divorce is significant. In 2012, there were 99,822 children who were aged under 16 when their parents divorced (Office for National Statistics, 2014). Furthermore, the number of children living with alcohol dependent parents is largely unknown. Estimates suggest that, approximately 22% of children aged under 16 live with a hazardous drinker (Manning *et al.*, 2009).

Childhood development has effects on health and well-being throughout life (Bartley, 2012). ACEs lead to poor health outcomes and health-threatening behaviours (Shonkoff *et al.*, 2012; Anda *et al.*, 1999; Bair-Merritt *et al.*, 2006). Therefore, addressing the relationship between ACEs and alcohol use is important in designing and implementing prevention programmes and treating drinking problems at an individual and population level.

Previous studies have provided evidence on the pathways between childhood predictors and alcohol use, but most are based on adolescents and young adults. Participants with ACEs initiate alcohol use at an earlier age and are more likely to drink to cope with problems (Dube *et al.*, 2006; Rothman *et al.*, 2008). Adolescent drinking correlates with physical and sexual abuse, parental divorce, household substance abuse and mental illness of family members in childhood (Dube *et al.*, 2006; Kristjansson *et al.*, 2009; Shin *et al.*, 2009). The relationship between ACEs and risk of problem drinking has been shown to persist into young adulthood (Thompson *et al.*, 2008; Hope *et al.*, 1998; Griffin and Amodeo., 2010; Kestilä *et al.*, 2008; Timko *et al.*, 2008).

Studies linking life-course determinants of alcohol misuse in middle-aged population are limited (Dubow *et al.*, 2008). Most studies have tended to focus on parental drinking, parental divorce and physical abuse. Other ACEs such as childhood hospitalization, living in an orphanage and exposure to parental arguments have received relatively little attention (Kestilä *et al.*, 2008; Mirsal *et al.*, 2004; Spak *et al.*, 1997). Additionally, studies on alcohol

use in midlife have tended to focus on a single type of ACE and have ignored the co-occurrence of various forms of ACEs. In fact, ACEs are highly interrelated, for example, children who have alcohol dependent parents report having more parental divorce, physical abuse and family violence (Marshal, 2003; Widom and Hiller-Sturmhöfel, 2001; Gilbert *et al*., 2009). But few studies have examined the contribution of cumulative exposure to ACEs to the risk of hazardous drinking in midlife (Kauhanen *et al*., 2011). Moreover, there is a growing interest in characteristics of those who abstain from alcohol. Some studies have found that abstainers have several psychosocial disadvantages, such as low education, not being married, depression (Bell *et al*., 2014), unemployment (Naimi *et al*., 2005; Camacho *et al*., 1987) and poorer health (Ng Fat and Shelton, 2012). However, the association between ACEs and abstinence is unknown. This topic should be explored further as ACEs might play a role in non-drinking behaviour. Furthermore, the impact of ACEs on midlife and later life alcohol consumption is poorly understood. A previous study found that there are long-term effects of exposure to childhood maltreatment on heavy drinking trajectories from adolescence to young adulthood (Shin *et al*., 2013). To our knowledge, the longitudinal relationship between ACEs and change in alcohol consumption in midlife has not been described.

The aim of this study was to examine the individual and cumulative effects of ACEs on weekly alcohol consumption in midlife and early old-age, and to investigate the role of ACEs in 10-year drinking trajectories from midlife through to early old-age.

## METHODS

### Data Source

Data were drawn from phase 5 (1997-1999), phase 7 (2002-2004) and phase 9 (2007-2009) of the Whitehall II Study (Marmot and Brunner, 2005). The mean length of follow up from phases 5 to 9 was 10.45 [Standard deviation (SD):0.58] years. Middle

adulthood describes the age period of 40 to 60, and late adult transition is from 60 to 65 (Levinson, 1986). The mean age at phases 5 and 9 were 56 (SD: 6.04) and 66 (SD: 5.98) years respectively. Phase 5 provides an estimate of alcohol consumption in midlife and acts as a baseline in the assessment of 10-year drinking trajectories from midlife to late adulthood. Phase 9 represents the period of early old-age. Phase 7 was within the transition period from midlife to early old-age and was used to provide additional information to capture the change in alcohol consumption during the transition period (Singer and Willett, 2003). The Whitehall II study therefore offers an important opportunity to examine the association between ACEs and alcohol consumption in both midlife and early old-age. The University College London ethics committee approved the study. Informed consent was obtained at baseline and renewed at each contact. Whitehall II data, protocols, and other metadata are available to bona fide researchers for research purposes. Please refer to the Whitehall II data sharing policy at http://www.ucl.ac.uk/whitehallII/data-sharing.

**Adverse childhood experiences**

Six categories of ACE were studied: parental divorce, parental mental health/alcohol problems, physical abuse, hospitalization >4 weeks, living in an orphanage, and exposure to parental arguments/fights. All questions about ACEs pertained to the participants' first 16 years of life and were assessed retrospectively at phase 5 through a self-completed questionnaire.

**Alcohol consumption**

Participants were asked to report the number of alcoholic drinks they had consumed in the last seven days, in terms of spirits (measures), wine (glasses), and beer (pints). These were then converted into UK units of alcohol (in the UK, one unit of alcohol is equivalent to eight grams of ethanol) and summed to derive total weekly consumption. A conservative conversion was applied whereby a single measure of spirits and glass of wine was estimated

to contain one unit of alcohol while a pint of beer was considered to contain two. Categorical weekly alcohol consumption was used to examine the individual and cumulative effects of ACEs, while a continuous measure was used to estimate 10-year drinking trajectories in longitudinal analyses. Hazardous drinking was defined as consuming 21 or more units per week for men and 14 or more units for women (Department of Health, 1995). On the basis of weekly alcohol intake, participants were classified into 3 categories: past week non-drinkers (0 units), moderate drinkers (1-20 units for men, 1-13 units for women) and hazardous drinkers (21+units for men and 14+units for women). The reference group was moderate drinkers.

**Possible confounders**

Adjustments were made for a wide range of risk factors potentially associated with both alcohol consumption and ACEs. These included age, sex, ethnicity, smoking status, marital status, and adult socio-economic position (SEP). Age was measured on the date of questionnaire completed at phases 5 and 9. Participants were classified into two 'ethnic' categories: white or non-white.

***Smoking status*** At phases 5, 7 and 9, participants were asked to report whether they currently smoked (cigarette/cigar), whether they previously smoked, of if they have never smoked (reference category). At Phases 5, 7 and 9, participants were classified into four categories: married/cohabiting (reference category), single, divorced or widowed.

***Adult socio-economic position (SEP)*** Participants' civil service grade (or last recorded grade if the participant was no longer in the civil service at phase 5 and/or 9) defined their SEP as high (referent; administrative), intermediate (professional/executive), and low (clerical/support roles).

**Statistical analysis**

Multinomial logistic regression models were used to examine the associations between

each category of ACE, as well as number of ACEs, and weekly alcohol consumption at phases 5 (midlife), and 9 (early old-age). Relative risk ratios (RRR) and 95% confidence interval (CI) for hazardous drinking and past week non-drinking in each category of ACE were estimated. For longitudinal analyses, mixed-effect multilevel modelling was used to investigate the effect of ACEs on drinking trajectories using data from phases 5, 7 and 9 (10-year interval; coefficients from these models reflect changes in alcohol consumption per unit increase in phase; approximately 5 years). As the group of past week non-drinkers may bias the findings, a sensitivity analysis restricting these models to those who reported drinking at phase 5 was performed. To determine whether sex was a moderator in the relationship between ACEs and alcohol consumption, models were initially fitted with interactions between individual ACEs and sex. No significant interactions were observed and therefore analyses in this study were pooled with adjustment for sex. For each ACE we present two models, one adjusted only for age and sex, and another adjusted for all covariates listed above. In the multilevel models we included an interaction between time and age to control for age-related changes in alcohol consumption (Britton *et al*., 2015). Age was centred on mean age at phase 5. Complete-case methods were used to analyse data. All statistical analyses were carried out using Stata 12.1 (StataCorp, Texas).

## RESULTS

### Participants' demographic characteristics

Participants' demographic characteristics at phases 5 and 9, and weekly alcohol consumption at phases 5, 7 and 9 are presented in Table 1. Nearly one-third of participants in this study were women. The mean age of participants at phases 5 and 9 was 56 (SD: 6.04; range from 45 to 69) and 66 (SD: 5.98; range from 55 to 80) years, respectively. Over 90% of participants were white, over 75% were married/cohabitated. A large proportion of participants were in high or intermediate SEP groups in adulthood. Mean weekly alcohol

consumption declined from phases 5 to 9.

**Adverse childhood experiences (ACEs)**

Women were more likely to report experiencing exposure to arguments/fights, parental divorce, parental mental health/alcohol problems and physical abuse (Table 2). The most common ACE across all participants was exposure to parental arguments/fights while the least common was living in an orphanage.

**Individual ACE and weekly alcohol consumption**

Participants who experienced physical abuse in childhood were 1.43 (95%CI 1.01, 2.04) times more likely to be hazardous drinkers than moderate drinkers in midlife after adjusting for age and sex (Table 3). However, in the multivariable adjusted model, the association was no longer statistically significant. Participants who were exposed to parental arguments/fights during childhood were 1.20 (95%CI 1.03, 1.38) times more likely to drink at a hazardous level in midlife than be moderate drinkers after adjusting for covariates. This association was robust in a sensitivity analysis adjusting for all other ACEs and covariates (data not shown).

Similarly, participants who experienced physical abuse in childhood were 1.60 (95%CI 1.08, 2.35) times more likely to report no consumption in past week in midlife, compared with moderate drinking. However, adjustment for covariates attenuated the effect to non-significant (RRR=1.32; 95%CI 0.87, 2.01). Exposure to parental arguments/fights was associated with past week non-drinking in midlife (RRR=1.22; 95%CI 1.04, 1.44). In the multivariable adjusted model, participants who were exposed to parental arguments/fights during childhood were 1.21 (95%CI 1.01, 1.45) times more likely to not drink in the past week in midlife, compared with moderate drinking.

Associations between ACEs and hazardous drinking/-past week non-drinkers at phase 9 were also examined (data not shown). Participants who lived in an orphanage during childhood had a lower risk of hazardous drinking in early old-age (RRR=0.28; 95%CI 0.086,

0.93), after adjusting for covariates. However, in the multivariable adjusted model, hazardous drinking in early old-age was not significantly associated with exposure to parental arguments/fights, parental divorce, parental mental health/alcohol problems, physical abuse and hospitalization >4 weeks. A significant association was observed between physical abuse in childhood and past week non-drinkers in early old age. Participants who were exposed to physical abuse in childhood were more likely to be past week non-drinkers in early old-age, compared with moderate drinking (RRR=1.73; 95%CI 1.18, 2.54). However, adjustment for covariates attenuated the effect (RRR=0.95; 95%CI 0.53, 1.70).

**Cumulative effect of ACEs on alcohol consumption**

For each one category increase in the number of ACEs a participant was exposed to during childhood, the risk of hazardous drinking versus moderate drinking increased by 1.09 (95%CI 1.01, 1.18) (Table 4). The association remained significant after adjusting for sex and age (RRR=1.10; 95%CI 1.02, 1.19). After further adjustment for adult SEP, ethnicity, marital status and smoking status, the association was no longer statistically significant (RRR=1.08; 95%CI 0.99, 1.17).

At phase 9, for each additional exposure to an ACE, the risk of hazardous drinking versus moderate drinking increased by 1.07 (95%CI 0.97, 1.18) in early old-age. However, this association was not statistically significant (data not shown).

**Longitudinal effect of ACEs on drinking trajectories from phases 5 to 9**

Both marital status and the interaction between sex and ACEs were not included in the final mixed-effect multilevel regression models as there was no evidence of any improvement in the model fit upon their inclusion according to likelihood ratio tests (Table 5). Alcohol consumption tended to decrease with increasing age. There was significant variation in both the slope and intercept of participants' drinking trajectories, indicating heterogeneity between participants' 10-year drinking trajectories. Participants who reported parental

arguments/fights had a slower rate of decline in alcohol consumption than those with no reports (β=0.039; 95%CI -0.34, 0.42) over 10-year interval, but this was not statistically significant (p=0.84). Those who experienced hospitalization (β= -0.12; 95%CI -0.57, 0.33) or living in an orphanage (β= -0.15; 95%CI -1.45, 1.15) made greater decline in alcohol consumption than those with no ACEs over 10-year interval when all covariates were held constant. As participants who reported hospitalization or living in an orphanage during their childhood made greater reductions on average in alcohol consumption over 10-year interval, they may mask the cumulative effects of number of ACEs experienced which were associated with slower decline in alcohol consumption in mixed-effect multilevel regression modelling. When examining the cumulative effect of ACEs, these two ACEs were excluded in calculating the total number of ACE score. Those who experienced an increasing the number of ACEs had a slower rate of decline in alcohol consumption than those who did not experience any ACEs (β=0.10; 95%CI -0.14, 0.34) over the 10-year interval, however, this effect was not statistically significant (p=0.42).

**DISCUSSION**

Among the six categories of ACEs examined in this study, only exposure to parental arguments/fights was associated with hazardous drinking in midlife, after controlling for a range of covariates. Exposure to parental arguments may act as a stressor to children, and children are more likely to have negative emotional reactions such as anger and aggression (Jenkins, 2000). At the same time, prolonged inter-parental conflict can influence parenting practices (Krishnakumar and Buehler, 2000). Parents who are in marital strife may spend less time monitoring or supervising their children (Cox *et al*., 2001). Parental factors such as parental monitoring and parental involvement are associated with delayed alcohol initiation and lower levels of alcohol use by offspring (Ryan *et al*., 2010).

Exposure to parental arguments/fights was also associated with both hazardous drinking

and past week non-drinking in midlife. This finding might provide insights into the role of childhood adversity to alcohol abstinence as well as poorer health profiles among non-drinkers. Several studies have shown that abstainers have worse health profiles than moderate drinkers (Ng Fat and Shelton, 2012; Fekjær, 2013). Moderate consumption of alcohol is alleged to confer protective effects for multiple cardiovascular outcomes and all-cause mortality (Movva and Figueredo, 2013; Ronksley *et al*., 2011; Roerecke and Rehm, 2012). However, the underlying mechanism between abstinence and worse health profiles is still poorly understood (Fekjær, 2013). Non-drinkers may have poor health outcomes due to factors other than not drinking. Future research should examine the interrelationship between adverse childhood experiences, abstinence and poor health outcomes. In this study, the definition of non-drinkers was broad and based on the previous week only. It includes lifetime abstainers, ex-drinkers, and occasional drinkers who did not drink in the week prior to participation. A more robust categorisation of abstaining would be required in future studies.

A significant association between the continuous ACE score and risk of hazardous drinking in midlife was observed in this study. Exposure to multiple ACEs was associated with an increased risk of hazardous drinking in midlife. This result supports previous studies (Bellis *et al*., 2014; Kauhanen *et al*., 2011; Dube *et al*., 2002), but to our knowledge, this is the first study to examine the role of ACEs in drinking trajectories across midlife longitudinally. The decline in alcohol consumption with increasing age is consistent with other longitudinal studies (Karlamangla *et al*., 2006; Moore *et al*., 2005). Although ACEs play a role in heavy drinking trajectories from adolescence to young adulthood (Shin *et al*., 2013), this study found that ACEs did not impact on the rate of change in alcohol consumption from during an approximately 10 years period from midlife into early old-age. The pathway from ACEs to adoption of drinking may be interrupted by resilience. Interaction

between genetic factors, life experiences, personal characteristics and social environment over the time may explain the resilience from ACEs (Collishaw *et al*., 2007; Liem *et al*., 1997). Adult experiences provide important turning points for individuals to counter ACEs (Rutter, 2006). Through life experiences, maturation may reduce the impacts of ACEs among adults and develop more adaptive strategies to cope with stress (Draper *et al*., 2008).

Some potential limitations of this study should be considered when interpreting the results. Firstly, the retrospective self-reported nature of ACEs raises the potential for recall bias. Participants may not be able to remember all the details in childhood fully or not willing to divulge the childhood adversities, resulting in those who truly had ACEs being misclassified as belonging to the unexposed group. Additionally, only six categories of ACE were offered in the questionnaire. Other important adverse experiences were not included, for example, being bullied by peers in childhood (Swahn *et al*., 2011; Topper *et al*., 2011). Some categories are very subjective, for example, exposure to parental arguments/fights, and some experiences are more adverse than others, for example, individuals who reported parental separation or divorce were likely to have suffered from physical abuse in childhood (Dong *et al*., 2004). This study did not capture the frequency or severity of ACEs which participants had experienced. Secondly, alcohol consumption could be either over- or under-reported by participants (Bellis *et al*., 2015). As both exposure (ACEs) and outcome (weekly alcohol consumption) were possibly underestimated, this may bias our findings towards the null hypothesis. Therefore, the strength of the relationships between ACEs and risk of hazardous drinking found in this study are likely to be conservative. Thirdly, the use of data from the Whitehall II study may limit the generalizability of the findings from this study. It is not fully representative of the general population, primarily consisting of white-collar, men of predominantly high-to-intermediate socioeconomic position. The proportion of participants reporting 2 or more ACEs (9.1%) in the Whitehall II study was lower than that of population

studies (23.7%) (Bellis *et al.*, 2014). However, aetiological findings from the cohort have been shown to be consistent with those obtained from general population samples suggesting this bias may not be as substantial as previously thought (Batty *et al.*, 2014). Bias may result from differential loss to follow-up in longitudinal studies (Hernán *et al.*, 2004). Illness and death could lead participants to drop-out not only before phase 5 in the Whitehall II study (the baseline of this study) but even if participants took part at phase 5, drop-out may be occurred in subsequent phases. ACEs and hazardous drinking are associated with poor health outcomes and death (Room *et al.*, 2005; Shonkoff *et al.*, 2012). Participants with ACEs and/or hazardous drinking may be more likely to drop-out, resulting in systematic differences between who remain and those who drop-out. Loss to follow-up may increase the potential for selection bias in the remaining sample, and mask the effects of ACEs on hazardous drinking in early old-age and longitudinally. Furthermore, some covariates that were included as confounders in our models could conceivably be argued to be mediators of the association between ACEs and alcohol consumption, for example experiencing ACEs could affect the socioeconomic group or marital status a participant belongs to in adulthood, which in turn might affect drinking - so therefore controlling for these factors may represent over-adjustment (Schisterman *et al.*, 2009). However, the age/sex adjusted estimates are similar to the multivariable adjusted estimates, suggesting that these variables are unlikely to explain a substantial proportion of the association between ACEs and alcohol intake in midlife/old-age.

This study is one of few to examine the relationship between ACEs and hazardous drinking in midlife, and the first to explore the impacts of ACEs on change in alcohol consumption from midlife to early old age. Target prevention and intervention programmes can help to reduce the occurrence of ACEs which may have favourable knock-on advantages in lowering the risk of hazardous drinking as well as negative health outcomes.

## CONCLUSION

The impact of exposure to parental arguments on hazardous drinking persists into midlife, but not early old-age. A significant association between continuous ACE score and risk of hazardous drinking in midlife was also observed. Our findings highlight that on-going efforts to prevent ACEs may also help to reduce hazardous drinking in midlife.

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**Table 1** Demographic characteristics and weekly alcohol consumption of participants.

| Covariates | | Phase 5 | | | | | | Phase 9 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Men | | Women | | Total | | Men | | Women | | Total | |
| Mean age (years, SD) | | 5473 | 55.69(5.99) | 2397 | 56.55(6.11) | 7870 | 55.95(6.04) | 4759 | 65.82(5.90) | 2002 | 66.43(6.13) | 6761 | 66.00(5.98) |
| Ethnicity N(%) | White | 5473 | 5116(93.48) | 2397 | 2070(86.36) | 7870 | 7186(91.31) | 4759 | 4472(93.97) | 2002 | 1746(87.21) | 6761 | 6218(91.97) |
| | Non-white | | 357(6.52) | | 327(13.64) | | 684(8.69) | | 287(6.03) | | 256(12.79) | | 543(8.03) |
| Marital status N(%) | Married/cohabitated | 4892 | 4156(84.96) | 2029 | 1269(62.54) | 6921 | 5425(78.38) | 4702 | 3917(83.30) | 1944 | 1082(55.66) | 6646 | 4999(75.22) |
| | Single | | 438(8.95) | | 380(18.73) | | 818(11.82) | | 386(8.21) | | 365(18.78) | | 751(11.30) |
| | Divorce | | 231(4.72) | | 234(11.53) | | 465(6.72) | | 240(5.10) | | 235(12.09) | | 475(7.15) |
| | Widowed | | 67(1.37) | | 146(7.20) | | 213(3.08) | | 159(3.38) | | 262(13.48) | | 421(6.33) |
| Adult SEP N(%) | High(administrative) | 5415 | 2776(51.27) | 2358 | 447(18.96) | 7773 | 3223(41.46) | 3102 | 1739(56.06) | 1258 | 318(25.28) | 4360 | 2057(47.18) |
| | Intermediate (professional/executive) | | 2325(42.94) | | 1071(45.42) | | 3396(43.69) | | 1210(39.10) | | 594(47.22) | | 1804(41.38) |
| | Low(clerical/support) | | 314(5.80) | | 840(35.62) | | 1154(14.85) | | 153(4.93) | | 346(27.50) | | 499(11.44) |
| Smoking status N(%) | Current-smoker | 5057 | 484(9.57) | 2141 | 283(13.22) | 7198 | 767(10.66) | 4480 | 273(6.09) | 1859 | 113(6.08) | 6339 | 386(6.09) |
| | Ex-smoker | | 2204(43.58) | | 700(32.70) | | 2904(40.34) | | 2235(49.89) | | 750(40.34) | | 2985(47.09) |
| | Never-smoker | | 2369(46.85) | | 1158(54.09) | | 3527(49.00) | | 1972(44.02) | | 996(53.58) | | 2968(46.82) |

| Weekly alcohol consumption | Phase 5 | | | Phase 7 | | | Phase 9 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Men (N=4979) | Women (N=2098) | Total (N=7077) | Men (N=4759) | Women (N=1981) | Total (N=6740) | Men (N=4612) | Women (N=1902) | Total (N=6514) |
| Mean alcohol units (SD) | 16.16 (16.18) | 7.14 (9.25) | 13.49 (15.05) | 14.25 (13.80) | 6.04 (7.33) | 11.84 (12.82) | 12.23 (12.20) | 5.40 (7.14) | 10.24 (11.40) |
| Moderate drinkers N(%) (1-20 units for men, 1-13 units for women) | 2962 (59.49) | 1140 (54.34) | 4102 (57.96) | 3044 (63.96) | 1050 (53.00) | 4094 (60.74) | 3078 (66.74) | 984 (51.74) | 4062 (62.36) |
| Hazardous drinkers N(%) (21+ untis for men, 14+ units for women) | 1443 (28.98) | 371 (17.68) | 1814 (25.63) | 1148 (24.12) | 327 (16.51) | 1475 (21.88) | 897 (19.45) | 264 (13.88) | 1161 (17.82) |
| Past week non-drinker (0 units) N(%) | 574 (11.53) | 587 (27.98) | 1161 (16.41) | 567 (11.91) | 604 (30.49) | 1171 (17.37) | 637 (13.81) | 654 (34.38) | 1291 (19.82) |

Phase 5= Middle adulthood

Phase 7= Transition period from middle adulthood to early old-age

Phase 9= Early old-age

**Table 2** Prevalence of adverse childhood experiences.

| Category of ACEs | Prevalence | | | n |
|---|---|---|---|---|
| | Men N(%) | Women N(%) | Total N(%) | |
| Exposure to parental arguments/fights | 886(18.36) | 495(24.37) | 1381(20.14) | 6858 |
| Parental divorce | 189(3.91) | 108(5.33) | 297(4.33) | 6861 |
| Parental mental health/alcohol problems | 275(5.70) | 145(7.15) | 420(6.13) | 6855 |
| Physical abuse | 91(1.89) | 86(4.24) | 177(2.58) | 6853 |
| Hospitalization > 4 weeks | 640(13.24) | 272(13.35) | 912(13.28) | 6870 |
| Living in an orphanage | 62(1.29) | 35(1.73) | 97(1.42) | 6840 |
| ACE score | | | | |
| 0 | 3294(68.88) | 1222(61.19) | 4516(66.62) | 6779 |
| 1 | 1089(22.77) | 559(27.99) | 1648(24.31) | |
| 2 | 300(6.27) | 166(8.31) | 466(6.87) | |
| ≥3 | 99(2.07) | 50(2.50) | 149(2.20) | |

ACEs= Adverse childhood experiences

**Table 3** Relationship between ACEs and weekly alcohol consumption after adjusting for covariates at phase 5.

| ACEs | No covariates added | | Sex + Age | | Multivariable adjusted (Sex + Age + Adult SEP + Ethnicity + Marital status + Smoking Status) | |
|---|---|---|---|---|---|---|
| | N | RRR (95%CI) | N | RRR(95%CI) | N | RRR(95%CI) |
| Hazardous drinking | | | | | | |
| Exposure to parental arguments/ fights | 6775 | 1.22* (1.07;1.41) | 6775 | 1.25* (1.09;1.44) | 6476 | 1.20*(1.03;1.38) |
| Parental divorce | 6779 | 1.01 (0.77;1.34) | 6779 | 1.05 (0.79;1.38) | 6480 | 0.97(0.72;1.31) |
| Parental mental health/ alcohol problems | 6772 | 1.00 (0.79;1.27) | 6772 | 1.00 (0.79;1.27) | 6472 | 0.95(0.75;1.22) |
| Physical abuse | 6771 | 1.35 (0.95;1.92) | 6771 | 1.43* (1.01;2.04) | 6474 | 1.37(0.94;1.98) |
| Hospitalization >4 weeks | 6788 | 1.02 (0.86;1.20) | 6788 | 1.04 (0.88;1.23) | 6487 | 1.05(0.88;1.24) |
| Living in an orphanage | 6759 | 0.72 (0.43;1.22) | 6759 | 0.76 (0.45;1.28) | 6465 | 0.75(0.43;1.32) |
| Past week non-drinkers | | | | | | |
| Exposure to parental arguments/ fights | 6775 | 1.22* (1.04;1.44) | 6775 | 1.13 (0.96;1.33) | 6476 | 1.21* (1.01;1.45) |
| Parental divorce | 6779 | 1.11 (0.81;1.53) | 6779 | 1.03 (0.74;1.42) | 6480 | 0.90 (0.64;1.28) |
| Parental mental health/ alcohol problems | 6772 | 0.98 (0.74;1.30) | 6772 | 0.93 (0.70;1.24) | 6472 | 1.04 (0.77;1.42) |
| Physical abuse | 6771 | 1.60* (1.08;2.35) | 6771 | 1.34 (0.90;1.99) | 6474 | 1.32 (0.87;2.01) |
| Hospitalization >4 weeks | 6788 | 1.07 (0.89;1.30) | 6788 | 1.05 (0.86;1.28) | 6487 | 1.04 (0.84;1.28) |
| Living in an orphanage | 6759 | 1.02 (0.59;1.76) | 6759 | 0.95 (0.54;1.65) | 6465 | 0.86 (0.47;1.57) |

Base outcome: moderate drinking
Reference group for adult socioeconomic position (SEP): high
Reference group for smoking status: never-smoker
*p <0.05

RRR= Relative risk ratios
CI= Confidence interval
ACEs=Adverse childhood experiences
Phase 5=Middle adulthood

**Table 4** Association between number of ACEs and weekly alcohol consumption at phase 5.

| Number of ACEs experienced (0 as the reference group) | No covariates added (N:6699) RRR(95%CI) | Adjusted for Sex + Age (N:6699) RRR(95%CI) | Multivariable adjusted (Sex + Age + Adult SEP + Ethnicity + Marital status + Smoking status) (N:6407) RRR(95%CI) |
|---|---|---|---|
| Hazardous drinking | | | |
| 1 | 1.18* (1.03;1.35) | 1.22* (1.07;1.40) | 1.17* (1.01-1.34) |
| 2 | 1.10 (0.87;1.38) | 1.12 (0.89;1.41) | 1.11 (0.88-1.41) |
| ≥3 | 1.21 (0.83;1.78) | 1.24 (0.84;1.82) | 1.13 (0.76-1.67) |
| Continuous ACEs score | 1.09* (1.01;1.18) | 1.10* (1.02;1.19) | 1.08 (0.99-1.17) |
| Past week non-drinkers | | | |
| 1 | 1.15 (0.98;1.35) | 1.06 (0.90;1.24) | 1.11 (0.93-1.31) |
| 2 | 1.22 (0.94;1.59) | 1.12 (0.86;1.45) | 1.15 (0.87-1.53) |
| ≥3 | 1.27 (0.82;1.99) | 1.19 (0.76;1.88) | 1.21 (0.74-1.97) |
| Continuous ACEs score | 1.11* (1.01;1.22) | 1.06 (0.96;1.16) | 1.08 (0.98-1.19) |

Base outcome: moderate drinking

Reference group for adult socioeconomic position (SEP): high

Reference group for smoking status: never-smoker

*p <0.05

RRR= Relative risk ratios

CI= Confidence interval

ACEs=Adverse childhood experiences

Phase 5=Middle adulthood

**Table 5** Fixed effects from multilevel analyses of the role of ACEs on drinking trajectories from phases 5 to 9

| ACEs | Unconditional model | | | Multivariable adjusted model^ | | |
|---|---|---|---|---|---|---|
| | N | Slope | Coefficient for interaction with slope† | N | Slope | Coefficient for interaction with slope† |
| Exposure to parental arguments/fights | 5744 | -2.15(-2.32;-1.98)** | 0.038 (-0.34;0.42) | 5686 | -2.19(-2.36;-2.02)** | 0.039 (-0.34;0.42) |
| Parental divorce | 5745 | -2.18(-2.33;-2.03)** | 0.44 (-0.31;1.20) | 5686 | -2.22(-2.37;-2.06)** | 0.45 (-0.31;1.21) |
| Parental mental health/alcohol problem | 5740 | -2.18(-2.33;-2.02)** | 0.36 (-0.27;0.99) | 5680 | -2.21(-2.37;-2.05)** | 0.31 (-0.32;0.95) |
| Physical abuse | 5740 | -2.15(-2.30;-2.00)** | 0.084 (-0.90;1.07) | 5680 | -2.19(-2.34;-2.03)** | 0.16 (-0.83;1.15) |
| Hospitalization > 4weeks | 5749 | -2.12(-2.28;-1.96)** | -0.15 (-0.60; 0.30) | 5689 | -2.16(-2.32;-2.00)** | -0.12 (-0.57;0.33) |
| Living in an orphanage | 5726 | -2.14(-2.29;-1.99)** | -0.33 (-1.63;0.96) | 5671 | -2.18(-2.33;-2.02)** | -0.15 (-1.45;1.15) |
| Number of ACEs experienced | 5698 | -2.18(-2.35;-2.01)** | 0.10 (-0.14;0.34) | 5641 | -2.22(-2.39;-2.05)** | 0.10 (-0.14;0.34) |

Reference group for adult socioeconomic position (SEP): high

Reference group for smoking status: never-smoker

^Adjusted for sex, age, adult SEP, ethnicity and smoking status

† Change in alcohol consumption per increase in phase (approximately 5 years)

**p ≤0.001

ACEs=Adverse childhood experiences

Phase 5= Middle adulthood

Phase 9= Early old-age