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Fathers' Alcohol Consumption and Long-Term Risk for Mortality in Offspring

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Abstract

Aim: This study examined associations between fathers' alcohol consumption and risk for total and cause-specific mortality in offspring.

Short summary: We examined the associations between fathers' alcohol consumption and total and cause-specific mortality in adult offspring. Fathers' alcohol consumption was associated with increased risk of alcohol-related mortality in offspring. The association appeared to be weaker for causes of death in which alcohol plays a smaller, or less direct, role.

Methods: Data on fathers' alcohol consumption, and offspring's risky use of alcohol, smoking, mental health and contact with police/childcare authorities were collected among 46,284 men (sons) aged 18–20 years, during conscription for compulsory military training in 1969/70. Data on offspring mortality were obtained from the National Cause of Death register, 1971–2008. The mortality outcomes included total mortality, alcohol-related causes of death and violent causes of death (categorized into suicides vs violent/external causes excluding suicides).

Results: Compared to sons whose fathers never used alcohol, the risk for total and alcohol-related mortality among sons increased with the father's consumption level. The risk of violent death was significantly elevated among sons whose fathers drank alcohol occasionally or often, but the risk of suicide increased in the highest consumption category only. After adjustment for covariates, the results remained for alcohol-related mortality whereas they were significantly attenuated, or disappeared, for total mortality, violent death and suicide.

Conclusions: Fathers' alcohol consumption is associated with increased risk of alcohol-related mortality in the offspring. Alcohol use among fathers also increases the offspring's risk of later total mortality, suicide and violent death, but these associations appear to be mediated or confounded by factors related to parental drinking and/or adverse childhood psychosocial circumstances.

INTRODUCTION

Use and abuse of alcohol are associated with various types of ill health in the users, and alcohol is a major factor contributing to the overall burden of disease and mortality in the population (Rehm *et al.*, 2017). Recent years have seen growing interest in assessing how one person's alcohol use impacts those in his/her environment, referred to as alcohol's harm to others. An important topic in this research is the child's vulnerability to parents' drinking, and more

specifically, the association between parental drinking and alcohol use and related harm in offspring. While most research in this area have focused on children of parents with more severe, long-term alcohol problems, e.g. clinically diagnosed alcohol use disorders, less is known about how children are affected by alcohol consumption among parents without clinically diagnosed alcohol problems—which is the focus of the present study. Indeed, there is evidence that the former group of children have an elevated risk for a wide range of negative outcomes including, e.g. substance use, poor school performance and unemployment (Johnson and Leff, 1999; Anda *et al.*, 2002; Berg *et al.*, 2016). Still, even though the latter group of children likely has a lower risk of harm in this context, they will probably account for a substantial proportion of the accumulated harm, simply because they are more numerous (Lund *et al.*, 2015).

Research on the link between alcohol consumption among parents without clinically diagnosed alcohol problems and negative outcomes in offspring has recently been the focus of two reviews by Rossow *et al.* (2016a, 2016b). While the reviewed research show that more parental drinking is associated with more drinking and related harm in offspring, there is little knowledge concerning the degree to which parental alcohol consumption *per se* increases the risk of adverse consequences in offspring, or whether the relationship is explained by other, unmeasured, factors and mechanisms. In fact, the reviews concluded that most previous studies have limited capacity to reveal causal links between parental drinking and adverse outcomes in children, partly due to failure to identify and account for confounding factors (Rossow *et al.*, 2016a, 2016b). Further, existing studies have mainly focused on adolescent drinking behaviour as the outcome, whereas there is a lack of studies focused on other health consequences, in particular in relation to forms of alcohol use other than clinically diagnosed alcohol disorders (Rossow *et al.*, 2016a, 2016b). In a recent study based on a large cohort of Swedish men, we found a positive association between fathers' alcohol consumption and alcohol-related hospitalizations among sons during a 40-year follow-up. Interestingly, this positive association was significant across all levels of fathers' consumption, implying that the risk of long-term alcohol-related harm among children is present also at lower levels of parental consumption (Hemmingsson *et al.* 2017). Still, no study has focused on other long-term health effects in this context, e.g. psychosocial problems or severe outcomes such as total and cause-specific mortality.

Several pathways linking parental drinking to adverse consequences in offspring can be hypothesized. First, parents' level of alcohol consumption and drinking patterns may be transferred directly to children through, e.g. social learning, specific ways of communicating about alcohol and/or favourable attitudes towards alcohol use (White *et al.*, 2000; Mares *et al.*, 2011; Vermeulen-Smit *et al.*, 2012; Yap *et al.*, 2017). Second, alcohol use (in particular more problematic consumption) may impair a parent's ability to care for the children. Lack of adequate parenting, including low parent–child relationship quality and low parental support and involvement, was recently shown to be associated with later alcohol misuse among adolescents (Yap *et al.*, 2017). Moreover, poor parental care can affect the child's mental health and personality or result in adverse childhood experiences, which in turn may increase the risk that the child develops alcohol problems and other negative social and health-related outcomes later in life (Dube *et al.*, 2002; Ramchandani and Psychogiou, 2009). Inadequate parenting may also have negative effects on children's skills development and future socioeconomic position (SEP), thus, increasing the risk that children later end up in environments associated with hazardous alcohol use

and other problem behaviours (Torvik *et al.*, 2011; Berg *et al.*, 2016). Consequently, parental drinking can act by direct pathways, affecting drinking behaviour and related harm in offspring, and by indirect pathways, through which parental drinking is associated with heightened prevalence of several risk factors for later mortality in offspring. Lastly, it is possible that the association is confounded by SEP (Lund *et al.*, 2015). Low childhood SEP has been shown to be linked to an elevated risk of alcohol-related harm in early adulthood, and the relationship seems in part to be attributable to alcohol use disorders among parents in the lowest SEP groups (Gauffin *et al.*, 2013). Hence, it is likely that this relationship is mediated by parental drinking, in which case the association between parental drinking and later alcohol-related harm in children would be confounded by childhood SEP.

In the present study, we aim to overcome some of the aforementioned limitations of the previous research on the association between parental drinking and negative outcomes in offspring. To do this we use a unique prospective cohort study design that combines survey information from a full cohort of Swedish men (sons), born around 1950, with registry data on mortality. More specifically we will: (a) examine whether parental drinking, measured as fathers' alcohol consumption, is associated with long-term risk for total and cause-specific mortality (including alcohol-related causes of death) in offspring; and assess to what degree the relationship is (b) confounded by childhood SEP and (c) mediated by factors in offspring related to parental drinking and/or early-life psychosocial disadvantage, including own (risky) use of alcohol, smoking, mental health and contact with police/childcare authorities. In addition, we will calculate the proportion of all alcohol-related deaths that occur among offspring of parents with and without diagnosed alcohol disorders, and explore whether the mortality risk differs between these two groups.

METHODS

Data collection

The study was based on data from a nationwide survey of young Swedish males conscripted for compulsory military service in 1969/ 1970. Only 2–3% of all Swedish men were exempted from conscription at that time, in most cases due to severe handicaps or congenital disorders. Only men born in 1949–1951 were included in this study, accounting for 97.7% of all conscripts in 1969/1970. The remaining 2.3% were born before 1949. Thus, all men in this study were 18–20 years of age at the conscription examination and between 20 and 60 years during follow-up, 1971–2008.

All men went through a health examination and met with a physician who diagnosed any disorders according to the Swedish version of International Classification of Disease (ICD), eighth revision (ICD-8). They also met a psychologist for a structured interview. During conscription the men were asked to complete two questionnaires. The first contained questions about social background, behaviour and adjustment, psychological factors, health, etc. The second contained questions that dealt specifically with substance use, e.g. alcohol consumption and tobacco smoking.

Outcomes

We used total and cause-specific mortality as outcomes. The latter included alcohol-related causes of death and violent causes of death (categorized into suicides vs violent/external causes excluding suicides). The rationale behind the selection of types of mortality was

to include causes of death related to alcohol use and/or other social and health-related problems resulting from parental drinking.

Information on offspring mortality between 1971 (i.e. the starting year of the register) and 2008 was obtained by record linkage with the National Cause of Death register, administered at the National Board of Health and Welfare in Sweden. For alcohol-related mortality we included the following ICD codes: ICD-8: 291, 303, 571.00, 571.01, 980; ICD-9: 291, 303, 305 A, 357 F, 425 F, 535D, 570.0, 570.1, 571A-D, 790D, 980; ICD-10: E244, F10, G312, G621, G721, I426, K292, K70, K852, K864-7, O354, T51, X45, X65, Z502, Z714, Z721, Y90, Y91 (424 cases). For suicide mortality: ICD-8/9: 95 and 98; ICD-10: X60-X84, Y10-Y34 (614 cases). For violent/external causes (excluding suicides): ICD-8/9: 80–94, 96–98; ICD-10: V01-Y98 (478 cases).

Exposure: fathers' alcohol consumption

Information on parental drinking was obtained from one of the questionnaires in which the young men were asked to report their fathers' frequency of alcohol consumption. The following question was asked: 'How often does your father use alcohol?' which had four response alternatives: never, rarely, occasionally, often. This measure has previously been validated in relation to alcohol-related hospitalizations among fathers (Hemmingsson *et al.* 2017). Moreover, to deal with misclassifications in the exposure variable (see Sensitivity analysis), we identified sons whose fathers had alcohol-related diagnoses (using the same ICD-codes as above) in the National Hospital Discharge register or the National Cause of Death register during follow-up.

Covariates

In previous studies based on the same cohort, we identified a number of early life risk factors for later alcohol-related harm and other negative social and health-related outcomes, collected among the men in adolescence (Hemmingsson *et al.*, 1999; Hemmingsson and Lundberg, 2004; Lundin *et al.*, 2011). Our view is that these risk factors most likely are mediators in the association between fathers' alcohol consumption and later risk of mortality among the sons. Information on the following covariates were obtained from the conscription examination: 'risky use of alcohol' (questionnaire response of having been apprehended for drunkenness, used alcohol as 'pick me-up', been drunk often/quite often, or reported alcohol consumption of at least 250 g/week); smoking >5 cigarettes/day; 'poor emotional control' (rated by psychologist as suffering from

reduced functions due to psychosomatic symptoms, low tolerance to stress and/or anxiety, problems controlling nervousness and aggression, and incapacity for emotional commitment, corresponding to the lowest 30% in a Gaussian distribution of general emotional control); receiving a psychiatric diagnosis from a psychiatrist; and, lastly, having had contact with childcare authorities or the police (Larsson *et al.*, 2002).

Information on childhood SEP was retrieved from the 1960 census and measured as fathers' occupational class, categorized into six groups according to the Swedish socioeconomic classification of occupations: unskilled workers, skilled workers, low-level non-manual employees, intermediate non-manual employees, high-level non-manual employees and self-employed.

Information on attained SEP was obtained from the 1985 census and measured using the same approach as for childhood SEP.

Statistical analyses

Cox proportional-hazards regression was used for the analyses of associations between level of fathers' alcohol consumption and mortality in offspring. Person-time was calculated from 1 January 1971 until death, emigration or end of follow-up in 2008, whichever came first. Men who emigrated were censored on the day of first emigration regardless of any re-migration during follow-up. Associations were estimated by hazard ratios (HR), with 95% confidence intervals, in crude and adjusted models, using the stcox procedure (Cox proportional-hazards regression) in the STATA 15 SE program. All results are based on those 46,284 men (93.8%) for whom there was information on all the variables included in the final analyses. Of the 49,321 in the full cohort, 1326 (2.7%) lacked information on fathers' alcohol consumption.

First, we estimated the associations between fathers' alcohol consumption and total and cause-specific mortality in sons as adults. Second, we examined the attenuation of these associations after adding confounding or mediating variables: childhood SEP, risky use of alcohol, smoking, psychiatric diagnoses, low emotional control and contact with police/childcare authorities. Third, we replicated the analyses of fathers' alcohol consumption and alcohol-related mortality in offspring (using follow-up from 1986) and examined the attenuating effect of attained SEP, to assess its mediating role in the association between fathers' drinking and mortality (alcohol-related only) in adult offspring. In addition, we calculated the proportion of all alcohol-related deaths that occurred among offspring of a father

Table 1. Distribution of respondents, number of deaths and the explanatory variables—across the categories of fathers' consumption

Number/deaths (%)	Fathers alcohol consumption			
	Never (20.1%) 9146/539	Rarely (46.1%) 21046/1439	Occasionally (29.5%) 13362/1032	Often (4.1%) 1870/183
	%	%	%	%
Baseline covariates[a]				
Risk use of alcohol	14.7	19.4	28.0	41.1
Smoking > 5c/d	34.8	47.1	53.2	65.7
Psychiatric diagnoses	12.1	10.4	12.4	28.1
Low emotional control	29.8	27.3	30.6	52.3
Police/childcare authorities	22.4	26.7	32.5	49.1
Childhood/attained SEP				
Father unskilled worker[b]	29.6	33.9	33.2	37.3
Unskilled worker 1985	19.1	19.5	19.5	22.8

[a]Measured at age 18. [b]Measured in the 1960 census.

who was vs was not diagnosed with an alcohol-related disorder or alcohol-related cause of death during follow-up. Finally, as a sensitivity analysis, we replicated the last set of models after adding a category to the variable 'fathers' alcohol consumption', namely, fathers with diagnosed alcohol-related disorders/causes of death during follow-up. The goal of these analyses was to determine whether the main result was affected by misclassifications in the exposure variable (i.e. if fathers with alcohol-related problems were classified into the lower categories of consumption) and assess to what degree the risk of alcohol-related mortality was elevated in the group who had fathers with diagnosed alcohol-related disorders or mortality during follow-up.

RESULTS

Descriptives

Table 1 shows the distribution of the respondents, and the prevalence of the explanatory variables, across the categories of fathers' consumption. While only 4.1% of the respondents reported that their father used alcohol often, 29.5% reported the father to be an occasional drinker, 46.1% that he drank rarely, and 20.1% that he never used alcohol. Moreover, most variables show a positive association with fathers' consumption, with the lowest prevalence among sons of fathers who never used alcohol and highest prevalence among those whose fathers drank often. For instance, risky use of alcohol, smoking, psychiatric diagnoses and poor emotional control are two to three times more common in the group with fathers who drank often. Conversely, the prevalence of low childhood and low attained SEP differs little across fathers' consumption.

Relationship between fathers' alcohol consumption and total and cause-specific mortality among sons

Table 2 shows the crude and adjusted models of the association between fathers' alcohol consumption and total and cause-specific mortality among sons. For both total and alcohol-related mortality, sons whose fathers drank, regardless of level, were at increased risk of mortality compared with sons whose fathers never drank alcohol. Specifically, the risk increased with increasing consumption levels, with HR and 95% confidence intervals ranging from 1.17 (1.06, 1.29) to 1.72 (1.45, 2.04) for total mortality and from 1.40 (1.02, 1.92) to 4.01 (2.65, 6.08) for alcohol-related mortality. However, the findings for the other causes of death display a different pattern. There was an elevated risk of violent deaths (excluding suicides) among sons of fathers who drank alcohol occasionally (1.32 [1,01, 1.73]), or often (1.84 [1.2, 2.82]), whereas an increased risk of suicide was found in the highest category of fathers' consumption only (1.87 [1.30, 2.67]).

Adjusting for childhood SEP only slightly attenuated the estimates for total and alcohol-related mortality, and resulted in no change in risk of violent mortality or suicide—implying that none of the relationship is significantly confounded by childhood SEP (Table 2). Adjusting for risky use of alcohol and the baseline covariates resulted in substantial attenuations in the risk of total mortality, suicide, and violent deaths, whereas most of the elevated risk remained for alcohol-related mortality.

Additional analyses of alcohol-related mortality

Next, we conducted analyses for alcohol-related mortality including attained SEP, measured as occupational class in 1985 (Table 3). As

can be seen, adjustment for attained SEP attenuated the HRs marginally, e.g. from 4.75 (2.45, 2.25) to 4.11 (2.12, 7.98) in the category with the highest level of fathers' consumption. Moreover, a majority of the association remained (3.84 [1.97, 7.47]) after concurrent adjustment for childhood and attained SEP.

In Table 4, we calculated the proportion of all alcohol-related deaths that occur among offspring of fathers with vs without diagnosed alcohol disorders or alcohol-related deaths. The vast majority, 82%, of all alcohol-related deaths occurred among sons of fathers without clinically diagnosed alcohol problems.

Sensitivity analyses

Finally, Table 5 shows the results from a sensitivity analysis where we include the sons of fathers with clinically diagnosed alcohol problems or alcohol-related deaths as a separate category. The risk increase still mirrored that in fathers' consumption across all categories but was clearly highest among sons of fathers with diagnosed alcohol disorders or alcohol-related deaths (9.90 [5.47, 17.92]). This pattern remained also in the adjusted models. In addition, we estimated the mortality risk in the group whose fathers had clinically diagnosed alcohol problems (not shown) and found an almost 3-fold risk (2.57 [1.91, 3.46]) of alcohol-related mortality compared to the other respondents.

DISCUSSION

This study probes links between parental drinking and long-term risk for mortality in offspring, with focus on alcohol consumption other than clinically diagnosed alcohol disorders. In summary, we found that sons' risk of total and alcohol-related mortality increased with their fathers' alcohol consumption across all consumption levels. The risks of suicide and other violent deaths were also elevated, most clearly among those whose fathers often drank alcohol. After adjustment for covariates, the association with alcohol-related mortality remained, whereas the associations with total mortality, violent death, and suicide were attenuated or even disappeared. Thus, our findings imply that fathers' alcohol consumption is mainly associated with a risk of alcohol-related mortality in offspring. The association appears weaker for total mortality and violent causes of death, in which alcohol plays a smaller or more indirect role.

We found no significant evidence of a confounding or mediating effect of SEP. After adjustments for childhood and attained SEP, respectively, the sons of fathers with the highest consumption still had an almost 4-fold risk of alcohol-related mortality. In turn, this finding suggests that the previously reported relationship between childhood SEP and alcohol-related disorders in adulthood (Gauffin et al., 2013) is not mediated primarily by parental drinking.

A recent study by Berg et al. (2016) found that most of the negative effect of parental alcohol use disorders on school performance could be attributed 'to a clustering of other adverse psychosocial factors commonly appearing together with' (or as a result of) clinically diagnosed alcohol problems, e.g. mental health problems, divorce and criminality among the parents. In our analyses, the effect of fathers' drinking on their sons' total mortality, suicide and violent death basically disappeared after adjustment for offspring's risky use of alcohol and other factors measured during adolescence (including smoking, psychiatric diagnoses, low emotional control and contact with police/childcare authorities). This may indicate that this relationship can largely be attributed to indirect pathways, i.e. mediated by offspring's adverse childhood psychosocial

Table 2. The association between fathers' alcohol consumption and total- and cause-specific mortality (1973–2008) in offspring

Fathers' alcohol use	Never	Rarely		Occasionally		Often	
	HR[a]	HR	95% CI[b]	HR[a]	95% CI[b]	HR[a]	95% CI
Mortality outcomes							
Total							
Crude	1	1.17	1.06 1.29	1.33	1.20 1.48	1.72	1.45 2.04
Childhood SEP	1	1.16	1.05 1.28	1.32	1.19 1.47	1.68	1.41 1.99
Risk use alcohol	1	1.14	1.03 1.26	1.23	1.11 1.37	1.49	1.25 1.76
Baseline covariatres[c]	1	1.11	1.00 1.23	1.18	1.06 1.31	1.19	1.01 1.42
Fully adjusted[d]	1	1.11	1.00 1.22	1.16	1.04 1.29	1.16	0.98 1.38
Alcohol-related							
Crude	1	1.40	1.02 1.92	2.04	1.48 2.81	4.01	2.65 6.08
Childhood SEP	1	1.36	0.99 1.86	1.98	1.43 2.73	3.78	2.49 5.74
Risk use alcohol	1	1.31	0.95 1.80	1.70	1.23 2.34	2.89	1.90 4.41
Baseline covariatres[c]	1	1.29	0.94 1.77	1.67	1.21 2.31	2.26	1.48 3.45
Fully adjusted[d]	1	1.25	0.91 1.72	1.56	1.13 2.16	2.05	1.34 3.13
Suicide mortality							
Crude	1	1.00	0.80 1.26	1.08	0.85 1.37	1.87	1.30 2.67
Childhood SEP	1	1.02	0.82 1.28	1.12	0.88 1.42	1.90	1.32 2.72
Risk use alcohol	1	0.96	0.76 1.20	0.95	0.75 1.21	1.48	1.03 2.13
Baseline covariatres[c]	1	0.98	0.78 1.23	0.98	0.77 1.24	1.24	0.86 1.79
Fully adjusted[d]	1	0.99	0.79 1.25	0.97	0.76 1.24	1.20	0.83 1.74
Violent mortality[e]							
Crude	1	1.02	0.78 1.32	1.32	1.00 1.73	1.84	1.20 2.82
Childhood SEP	1	1.00	0.77 1.31	1.31	1.00 1.73	1.79	1.16 2.74
Risk use alcohol	1	0.97	0.75 1.27	1.17	0.89 1.54	1.48	0.96 2.27
Baseline covariatres[c]	1	0.95	0.73 1.24	1.11	0.84 1.47	1.15	0.75 1.77
Fully adjusted[d]	1	0.95	0.72 1.23	1.10	0.83 1.45	1.11	0.72 1.71

[a]Hazard ratios.

[b]95% confidence intervals.

[c]Measured at age 18: smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities.

[d]Childhood SEP, risk use of alcohol, smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities.

[e]Excluding suicides.

Table 3. The association between fathers' alcohol consumption and alcohol-related mortality (1986–2008) in offspring

Fathers' alcohol use	Never	Rarely		Occasionally		Often	
	HR[a]	HR[a]	95% CI[b]	HR[a]	95% CI[b]	HR[a]	95% CI[b]
Alcohol-related mortality							
Crude	1	1.82	1.09 3.04	3.35	2.02 5.57	4.75	2.45 9.22
Childhood SEP	1	1.73	1.03 2.90	3.20	1.92 5.33	4.37	2.25 8.51
Risk use alcohol	1	1.73	1.03 2.89	2.90	1.74 4.83	3.65	1.87 7.14
Baseline covariatres[c]	1	1.67	1.00 2.80	2.75	1.65 4.59	2.71	1.38 5.30
Attained SEP	1	1.81	1.08 3.03	3.24	1.95 5.38	4.11	2.12 7.98
Childhood + attained SEP	1	1.74	1.04 2.91	3.10	1.87 5.17	3.84	1.97 7.47
Fully adjusted[d]	1	1.64	0.98 2.74	2.63	1.58 4.39	2.52	1.28 4.94

[a]Hazard ratios.

[b]95% confidence intervals.

[c]Measured at age 18: smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities.

[d]Childhood SEP, risk use of alcohol, smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities, attained SEP.

Table 4. Distribution of alcohol-related deaths among sons to fathers with and without diagnosed alcohol use disorders/alcohol-related causes of death

Fathers' alcohol use	Never	Rarely	Occasionally	Often	Total	%
Without fathers-alc. diagnosis	36	127	97	19	283	82
With fathers-alc. diagnosis	3	9	24	12	50	18
Total	40	144	129	32	355	100

Table 5. The association between fathers' alcohol consumption (with fathers' alcohol-related diagnosis as separate category) and alcohol-related mortality (1986–2009) in offspring

Fathers' alcohol use	Never	Rarely			Occasionally			Often			Father with alc. diagnosis		
	HR[a]	HR[a]	95% CI[b]		HR[a]	95% CI[b]		HR[a]	95% CI[b]		HR[a]	95% CI[b]	
Crude	1	2.01	1.15	3.51	3.21	1.83	5.63	4.37	1.91	9.98	9.90	5.47	17.92
Childhood SEP	1	1.92	1.10	3.36	3.09	1.76	5.43	4.11	1.79	9.41	9.06	4.99	16.45
Risk use alcohol	1	1.91	1.09	3.35	2.80	1.59	4.92	3.38	1.47	7.76	8.32	4.58	15.11
Baseline risk factors[c]	1	1.86	1.06	3.25	2.68	1.52	4.71	2.60	1.13	5.99	6.90	3.79	12.57
Attained SEP	1	2.01	1.15	3.51	3.14	1.79	5.50	3.91	1.71	8.95	8.44	4.66	15.30
Childh. + attained SEP	1	1.93	1.10	3.38	3.03	1.72	5.31	3.73	1.63	8.53	7.82	4.30	14.22
Fully adjusted[d]	1	1.82	1.04	3.46	2.59	1.47	4.56	2.47	1.07	5.71	6.06	3.31	11.06

[a]Hazard ratios.

[b]95% confidence intervals.

[c]Smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities.

[d]Childhood SEP, risk use of alcohol, smoking, psychiatric diagnoses, low emotional control, contact with police/childcare authorities, attained SEP.

circumstances related to parental drinking, rather than drinking *per se*. If the covariates we have adjusted for actually are on the causal pathway between fathers' drinking and offspring mortality, it would mean that our adjusted risk estimates underestimate the true association and no such adjustments should be made. Alternatively, if the factors were outcomes of other negative childhood circumstances that also affected the fathers' alcohol consumption, e.g. parental mental health problems, it would be more correct to view them as confounders. However, in additional sensitivity analyses (not shown), we adjusted for parental suicide and hospitalization with psychiatric diagnoses, as indicators of a negative childhood family environment. Including this information in the models did not change the results. Moreover, in our analyses of alcohol-related mortality, most of the effect of fathers' alcohol use remained after adjustment for these factors—which we interpret as parental drinking having a more direct and independent impact on the long-term risk of alcohol-related mortality in offspring.

Finally, as our sensitivity analyses revealed, the sons of fathers with alcohol-related clinical diagnoses had an almost 3-fold risk of alcohol-related mortality compared to the other respondents and a 10-fold risk in relation to those whose fathers never drank alcohol. Still, the small size of this group implied that the vast majority of all cases of alcohol-related mortality occurred among sons of fathers without clinically diagnosed alcohol disorders. Consequently, in order to reach the majority of alcohol-related harm in offspring owing to parental drinking, prevention efforts aimed at children of parents with alcohol use disorders should be complemented with universal alcohol policy measures aimed at reducing the total consumption in society.

The present study had several strengths. First, it combined self-reported information on theoretically based confounding and mediating factors from pre-adulthood with information from registers for a long-term follow-up of mortality. Second, this information was obtained from sources with very little missing information. The full cohort of Swedish men born around 1950 in combination with high-quality registers provided a large and representative study population for the study. Third, no retrospective information was used in the study. Such information on early-life circumstances is known to produce bias in life-course studies within this area of research (Batty *et al.*, 2005; Kauhanen *et al.*, 2006). Some limitations of the study should also be noted. First, our measure on parental drinking was reported by the offspring. Although offspring's and parental reports on parental drinking have been found to correlate, there is some evidence that offspring tend to underestimate parental

drinking (Dielman *et al.*, 1995; Aas *et al.*, 1996). This could in turn explain why Rossow *et al.* (2016b) found that studies based on offspring's reports of parental drinking more often result in non-significant associations, compared to when alcohol use is reported by the parents themselves. Second, the measure included rather crude information on fathers' frequency of drinking, from 'never' to 'often', and no information on fathers' levels of drinking, implying that the measure to some extent may be misclassified in relation to fathers' actual consumption. However, in a previous study the measure showed a strong and dose related association with alcohol-related hospitalizations among the fathers, i.e. the risk increased with increased alcohol consumption. We regard this as a validation of the ability of our measure to reflect differences in the frequency of fathers' alcohol consumption (Hemmingsson *et al.* 2017). Third, fathers' consumption was measured at the time of conscription when the sons were at the age of 18–20 years old. It would probably have been more relevant to have this information during more sensitive periods of the life span, were parental drinking might have a stronger impact on the formation of long-term risk for negative health outcomes in offspring, e.g. infancy and early adolescence. Taken together, the limitations of our measure of exposure imply that our estimates most likely represent underestimations of the true associations at issue. Finally, our analyses included no information on maternal alcohol consumption. According to Rossow *et al.*, (2016a) studies using exposure measures from both parents separately more often find significant associations in this context, again indicating that our estimates may be underestimated. On the other hand, at the time of the conscript examination levels of alcohol consumption were substantially lower among women than among men in Sweden. Consequently, failure to account for information on mothers' drinking should not be a significant problem in this study. Moreover, with regard to other maternal variables that may have an effect on the studied outcomes, we conducted an additional sensitivity analysis (not shown) were the main models were adjusted for both paternal and maternal psychiatric diagnoses during follow-up. These models did not change the main result of the study, i.e. they did not significantly affect the association between fathers' alcohol consumption and later alcohol-related mortality in offspring.

CONCLUSION

Fathers' alcohol consumption is associated with increased long-term risk of alcohol-related mortality in offspring. Alcohol use among

fathers also increases the offspring's risk of later total mortality, suicide and violent death, but these associations appear to be mediated or confounded by factors related to parental drinking and/or adverse childhood psychosocial circumstances. Lastly, our findings highlight the importance of applying a broader public health approach to prevent the majority of harms to offspring related to parental drinking.

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CONFLICT OF INTEREST STATEMENT

None.

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