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Associations between exposure to stressful life events and alcohol use disorder in a longitudinal birth cohort studied to age 30

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ABSTRACT

Background: To examine associations between measures of stressful life events exposure and alcohol abuse/dependence (AAD) from ages 18 to 30 using data from a longitudinal birth cohort ($n = 987$ to 1011).

Methods: Outcome measures included DSM-IV (American Psychiatric Association, 1994) AAD symptoms and AAD, at ages 20–21, 24–25, and 29–30 years. Exposure to a range of stressful life events was measured during the periods 18–21, 21–25, and 25–30 years using items adapted from the social readjustment rating scale (Holmes and Rahe, 1967). Data were analysed using Generalised Estimating Equation models, adjusted for non-observed sources of confounding using conditional fixed effects regression. Further analyses examined: gender × life events exposure interactions, structural equation modelling of possible reciprocal causal pathways linking stressful life events and AAD symptoms, and an alternative conceptualization of the stressful life events measure.

Results: After adjustment, those with the highest exposure to stressful life events had rates of AAD symptoms that were 2.24 ($p < .0001$) times higher, and odds of AAD that were 2.24 times higher($p < .01$), than those at the lowest level of exposure. Associations between life events exposure and AAD symptoms were stronger for females than for males ($p < .05$), with results consistent using a count measure of stressful life events. Structural equation modelling showed that the best-fitting model was one in which life events influenced AAD symptoms.

Conclusions: The results suggest that there were persistent linkages between stressful life events and AAD, providing support for a stress-reduction model of alcohol consumption.

1. Introduction

There has been increasing interest in the effect of exposure to stressful life events on alcohol consumption behaviour, with studies finding that increasing exposure to stressful life events is associated with increasing risks of alcohol abuse/dependence (AAD) (Anthenelli, 2012; Ayer et al., 2011; Blomeyer et al., 2011; Dawson et al., 2005; Jose et al., 2000; Keyes et al., 2012, 2011; King et al., 2003; Lee et al., 2012; Lemke et al., 2008; Moos et al., 2004; Perreira and Sloan, 2001; Rospenda et al., 2008; Sillaber and Henniger, 2004; Timko et al., 2008; Veenstra et al., 2006). There are several possible explanations for these consistent associations.

First, it may be that these associations arise because alcohol is used to alleviate stress and improve mood (Cooper, 1994; Cooper et al., 1995). This view is supported by research from a number of sources (Ayer et al., 2011; Bolton et al., 2009; Colder, 2001) that have shown that individuals report consuming larger amounts of alcohol in response to stress exposure. Ayer et al. (2011), using a voice diary methodology with a sample of heavy drinkers, found that higher levels of stress on any day predicted increased alcohol consumption the following day, suggesting that participants were drinking in response to stress.

Second, the association between stressful life events exposure and AAD may reflect the effects of confounding factors that increase the risks of both stressful life events and AAD. While a number of studies have controlled for possible sources of confounding (Blomeyer et al., 2011; Keyes et al., 2012, 2011), the possibility exists that these linkages may be explained by sources of non-observed confounding. One method for examining this issue is to employ fixed effects regression models (Cameron and Trivedi, 1998; Greene, 1990), which make it possible to take into account confounding by non-observed genetic and environmental factors that are correlated with stressful life events and that have a *fixed* effect on AAD over time. In the context of research into stressful

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life events and alcohol, factors that may be subsumed by the fixed effects term are all individual, family, social, and related factors that are fixed at the point of adolescence and which have a fixed effect on later stressful life events and AAD. A key feature of the fixed effects model is that, because it accounts for all time-invariant individual effects in a study, it produces less biased estimates than models employing observed confounding factors, and reduced standard errors (Allison, 2009). However, the model does not address the issue of confounders that may vary over time and to control for such confounding, the fixed effects model must be augmented by observed time-dynamic confounding factors. While such models have been used in many areas of epidemiology (Allison, 2009), they have not previously been used to examine linkages between stressful life events and AAD.

A third issue is the extent there may be gender differences in the linkages between stressful life events exposure and AAD. Several studies have found evidence that the linkages between stressful life events and AAD are stronger for women (King et al., 2003; Rospenda et al., 2008), whereas others have found evidence for stronger links amongst males (Ayer et al., 2011; Dawson et al., 2005; Lemke et al., 2008; San Jose et al., 2000), or no difference (Veenstra et al., 2007). Longitudinal data with measures of stressful life events and AAD at multiple time points may allow for more precise estimation of gender differences in the linkages between stressful life events and AAD.

Finally, it is possible that there is a reverse causal association in which AAD leads to increased susceptibility to stressful life events exposure (Brennan et al., 1999), which has been referred to as *alcohol contaminated* life events (Hart and Fazaa, 2004). One approach to addressing the issue of reverse causal processes is to employ structural equation modelling methods. These models permit the estimation of reciprocal relationships between stressful life events and AAD, with these models being compared to similar models estimating unidirectional causal pathways from stressful life events to AAD, and vice versa. These models provide an indicative guide to likely patterns of causation (Fergusson et al., 2009, 2011), but have only been infrequently used to examine the linkages between stressful life events and AAD (Dermody et al., 2013; Wills et al., 2002).

2. Methods

2.1. Participants

Data were gathered during the course of the Christchurch Health and Development Study (CHDS), a study of a birth cohort of 1265 children (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977. The cohort has been studied at birth, 4 months, 1 year and annually to age 16 years, and again at ages 18, 21, 25 and 30 years (Fergusson and Horwood, 2001; Fergusson et al., 1989). All study information was collected on the basis of signed consent from study participants and is fully confidential, and is approved by the Canterbury (NZ) Ethics Committee.

2.2. Exposure to stressful life events (ages 18–21, 21–25, and 25–30 years)

Exposure to stressful life events was assessed by questioning respondents about life events for each 12-month period over the periods 18–21, 21–25, and 25–30 years. Life events were assessed using a 30-item inventory based on the Holmes and Rahe (1967) social readjustment rating scale supplemented by custom-written survey items. These items spanned several domains, including: changes to living situation, employment/finances, death/illness, relationship problems/difficulties, problems with family members/family members' crises, problems with friends/friends' crises, crime victimisation, and other problems. All items were scored on a 0 to 4 scale with 0 representing "no event", 1 "not upset/distressed", 2 "a little upset/distressed", 3 "moderately upset/distressed", and 4 "very distressed", based on the recommendations by Brown and Harris (1978), Ormel and Wohlfarth (1991). Using this information, two measures of exposure to stressful life events were created. The first measure was a life events distress measure, which was computed by summing the 0 to 4 scaling for each item for each 12-month period, and then summing over each assessment period, resulting in a total life events distress score for the periods 18–21, 21–25, and 25–30 years (M [18–21] = 18.0, SD = 12.6, M [21–25] = 17.0, SD = 13.5; M [25–30] = 13.6, SD = 12.3). The second measure was a count of the number of stressful life events reported during each assessment period (M [18–21] = 8.5, SD = 5.5; M [21–25] = 7.6, SD = 4.9; M [25–30] = 5.9, SD = 4.5).

For the purposes of the present investigation, the total life events distress scores for each period were used to create a four-level classification of life events distress during each assessment period, representing quartiles on the total life events distress score. In addition, the count measure of life events was also used to create a four-level classification of exposure to stressful life events during each assessment period (0–5 events; 6–10 events, 11–15 events, and 16+ events).

2.3. Alcohol misuse (AAD symptoms and AAD, ages 20–21, 24–25, and 29–30 years)

At ages 21, 25 and 30 years, participants were interviewed concerning alcohol use since the previous assessment using components of the Composite International Diagnostic Interview (CIDI) (World Health Organization, 1993) to assess DSM-IV (American Psychiatric Association, 1994) symptom criteria for alcohol abuse/dependence (AAD). This information was used to create two outcome measures related to AAD for the 12-month period prior to the assessment. The first was a count measure of the number of AAD symptoms reported for the 12-month period immediately prior to the assessment (when cohort members were aged 20–21, 24–25, and 29–30 years). The second was a dichotomous classification indicating whether cohort members met DSM criteria for AAD during the 12-month period prior to the assessment.

2.4. Time-dynamic covariate factors (ages 18–21, 21–25, and 25–30 years)

In order to control for the effects of possible comorbid mental health disorders and prior AAD in the analyses, three time-dynamic covariate factors were obtained from the study database. These included: (a) prior history of AAD (ages 18–20, 18–24, and 18–29 years), (b) concurrent major depression (ages 18–21, 21–25, and 25–30); and (c) concurrent anxiety disorder (ages 18–21, 21–25, and 25–30 years). Comorbid mental health and prior AAD were controlled from age 18 because time-dynamic covariate factors in fixed effects models must be observed during the period in which both the exposure and outcome are observed. Any effects of mental health disorders or AAD prior to age 18 were accounted for by the fixed effects portion of the model. Details of these measures are given in the Online Supplement.

2.5. Statistical analyses

Associations between exposure to stressful life events and AAD outcomes: In the first stage of the analyses, the pooled associations between the categorical measure of life events distress at ages 18–21, 21–25, and 25–30 years and the two AAD outcomes (AAD symptoms; AAD) were estimated via Generalized Estimating