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Modelling possible causality in the associations between unemployment, cannabis use, and alcohol misuse

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ABSTRACT

Background: There has been considerable interest in the extent to which substance use and unemployment may be related, particularly the causal pathways that may be involved in these associations. It has been argued that these associations may reflect social causation, in which unemployment influences substance use, or that they may reflect social selection, in which substance use increases the risk of becoming and remaining unemployed. The present study sought to test these competing explanations.
Methods: Data from the Christchurch Health and Development Study, featuring a longitudinal birth cohort, were used to model the associations between unemployment and both cannabis and alcohol. Data on patterns of unemployment, involvement with cannabis, and symptoms of alcohol use disorder were examined from ages 18–35 years. The associations between unemployment and both cannabis dependence and alcohol use disorder (AUD) were modelled using conditional fixed-effects regression models, augmented by time-dynamic covariate factors.
Results: The analyses showed evidence of possible reciprocal causal processes in the association between unemployment and cannabis dependence, in which unemployment of at least three months' duration significantly ($p < 0.0001$) increased the risk of cannabis dependence, and cannabis dependence significantly ($p < 0.0001$) increased the risk of being unemployed. Similar evidence was found for the associations between unemployment and AUD, although these associations were smaller in magnitude.
Conclusions: The present findings support both social causation and social selection arguments, by indicating that unemployment plays a causal role in substance misuse, and that it is also likely that a reverse causal process whereby substance misuse increases the risk of unemployment.

1. Introduction

During the global economic crisis of 2007 and the subsequent recession, New Zealand experienced an increase in its unemployment rate. In 2006, the unemployment rate was 3.8% (Ministry of Social Development, 2008). In December 2009, the rate climbed to 6.1%, with the rate for young adults being substantially higher (16.6%; Ministry of Social Development, 2010). Historically, young adults are relatively more vulnerable to unemployment in New Zealand (Ministry of Social Development, 2010), underscoring the importance of understanding the potential impact of unemployment during this risk-prone developmental period.

Unemployment is associated with increased susceptibility to psychiatric problems, such as substance use disorder (Catalano et al., 2011; Henkel, 2011). The nature of this association has been a subject of a decades-long but yet unsettled debate (Catalano et al., 2011; Dooley et al., 1992; Henkel, 2011; Mossakowski, 2008). Two lines of thought offer explanations for the nature of this association: social causation and social selection (Boden et al., 2014; Catalano et al., 2011; Henkel, 2011; Sareen et al., 2011). First, the social causation argument (Catalano et al., 2011; Henkel, 2011; Sareen et al., 2011) posits that unemployment triggers changes in substance use. Five lines of thought and hypotheses specify the association further. Out of those five, three hypotheses suggest that unemployment can significantly *increase* one's substance use (i.e., countercyclical): a) the stress hypothesis posits that

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unemployment (Ross and Huber, 1985) might increase involvement in substance use, because an unemployed individual might use substances to cope with stress associated with unemployment (Boden et al., 2014; Catalano et al., 2011; Henkel, 2011; Mossakowski, 2008); b) the frustration-aggression hypothesis argues that an unemployed person engages in antisocial behaviours, such as problematic alcohol use as an expression of their frustration (Berkowitz, 1989); and c) the deprivation theory, such as Jahoda's latent deprivation model (Jahoda, 1981, 1982) or Warr's vitamin model (Warr, 1987, 2007), hypothesizes that an unemployed person loses all the latent beneficial elements accompanying employment, such as time structure, social contact, or status. Unstructured and increased leisure time, for example, might result in more involvement in problematic drinking. On the other hand, the remaining two lines of thought argue that unemployment can significantly *decrease* one's substance use (i.e., procyclical): a) the income loss hypothesis posits that unemployment may decrease substance use, because an unemployed person would be less likely to spend money on nonnecessity items, such as substances, to accommodate the reduction in disposable income subsequent to unemployment (Catalano et al., 2011; Henkel, 2011).; and b) the inhibition effect hypothesis (Catalano et al., 2002) suggests that particularly during the period of recession, those who perceive themselves to have a high probability of losing their job will constrain themselves from problematic drinking out of fear of losing their job. Although these five hypotheses differ in their proposed answers to the direction in the association between unemployment and substance use (i.e., countercyclical or procyclical) and/or possible intervening factors underlying the association, they share one key tenet—changes in one's employment status trigger changes in one's substance use behaviours. In contrast to the social causation argument, the social selection argument posits a possible reverse causality—preexisting substance use problems might compromise individuals' labour force participation status rather than the other way around (Hart and Fazaa, 2004; Sareen et al., 2011). For example, young adults might lose their jobs because of their binge drinking behaviours and compromised performance at work due to their excessive drinking.

Existing evidence of the linkage between unemployment and substance use problems among young adults is limited and mixed (Catalano et al., 2011; Mossakowski, 2008). Supporting the social causation argument, particularly stress hypothesis, Redonnet et al. (2012) found that unemployment increased levels of alcohol abuse among adults aged 22–35. On the other hand, although relevant studies are fewer in number and often feature samples with a wide age range, pre-existing substance use has been reported to limit the ability to attain a favourable socioeconomic status, which is in line with the social selection argument (Dooley et al., 1992; Mullahy and Sindelar, 1989; Johansson et al., 2007). A null finding has also been reported regarding the association between unemployment and cannabis abuse among participants aged 20–37 (Melchior et al., 2015), although the association was statistically significant among those without higher education. Such mixed empirical findings indicate that a consensus has not yet emerged regarding the debate of causality between unemployment and substance use (Backhans et al., 2012; Blomeyer et al., 2011; Catalano et al., 2011; Henkel, 2011; Keyes et al., 2012; Lundin et al., 2012; Sareen et al., 2011). This motivates an empirical inquiry that explicitly focuses on the issue of causality and then tests the causal nature in the association between unemployment and substance use during this critical developmental period.

In any inquiry related to causality, the critical first step is to rule out possibilities of confounders as much as possible. A widely used strategy to address the causality issue is controlling for individuals' preexisting involvement in substance use and other potential confounders (Boden et al., 2014; Lee et al., 2015). Although it is a valid way to rule out competing explanations, adjusting for possible confounders is limited in that sources of unobserved confounding are not taken into account (Boden et al., 2014; Popovici and French, 2014). Fixed-effects regression models are well suited for addressing this issue of unobserved confounding (Cameron and Trivedi, 1998; Greene, 1990). In this type of modelling, the fixed-effects terms represent all unobserved genetic, sociodemographic, individual, social, and environmental factors that have time-invariant fixed effects on unemployment and substance use disorder. A primary advantage of the fixed-effects regression model is that it generates less biased estimates than a more traditional covariate-adjusted regression model, because it accounts for all time-invariant effects (Allison, 2009). Such innovative analytic approach has been utilized in a very limited number of studies. For example, recent studies estimated typical fixed effects regreesion models to examine the association of alcohol (Popovici and French, 2013) and cannabis use (Popovici and French, 2014) with employment, and then provided supporting evidence for the possibility of social selection. However, another potentially critical source of bias is not considered in these important studies—confounding factors that vary over time, which can bias estimates in typical fixed-effects models. The present study empirically address this issue by adding observed time-dynamic confounding factors to typical fixed-effects models (Allison, 2009). This modelling strategy allows researchers to further rule out other competing explanations in the linkage between unemployment and problematic substance use, the essential step to clearly establishing causality in the linkage. This modelling strategy has been used in only a few studies examining unemployment and increased vulnerability to substance use problems (e.g., Boden et al., 2014).

Furthermore, even if possible confounding effects in an association between unemployment and substance use is minimized using augmented fixed-effects regression models, the question regarding the direction in causality might remain unresolved. One approach to directly assessing the direction of causality is to estimate a fixed-effects regression model for unemployment, predicted by substance use problems (i.e., social selection), and then another fixed regression model for substance use problems, predicted by unemployment (i.e., social causation). Such consideration has not been made yet in existing studies. For example, the recent studies by (Popovici and French, 2013, 2014) focused on the possibility of social selection and did not explicitly test the possibility of social causation. Likely patterns and directions of causation can be inferred by empirically integrating results from these models where proposed causal directionality varies.

1.1. The present study

The present study aimed to clarify the question of causality between unemployment and pathological substance use, namely alcohol use disorder and cannabis dependence, using data from a prospective longitudinal birth cohort and employing fixed-effects models augmented with time-dynamic observed confounding factors. The present analyses focused on alcohol use disorder symptoms and cannabis dependence symptoms, because these substances are the two most widely used legal and illegal substances among young adults in New Zealand (Ministry of Health, 2010, 2015). In addition, a possible reverse possibility (i.e., the administration of nicotine will trigger changes in one's employment status) is less likely, as the administration of nicotine is less likely to have detrimental effects on daily performance at work which might lead to one's job loss. It is feasible that unemployment may have differential associations with alcohol and cannabis, considering differences in availability of these two substances due