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 to 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 ($p < .0001$) increased the risk of cannabis dependence, and cannabis dependence significantly ($p < .0001$) 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.

Keywords: cannabis use; alcohol use disorder; unemployment; longitudinal study

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 unemployment (Ross & 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; Jahoda, 1982) or Warr's vitamin model (Warr, 1987; Warr, 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 & 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 & Sindelar, 1989; Johansson, Alho, Kiiskinen, & Poikolainen, 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 & French, 2014). Fixed-effects regression models are well suited for addressing this issue of unobserved confounding (Cameron & 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 & French, 2013) and cannabis use (Popovici & 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 & 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.

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 to their varying status with regard to laws governing substance use in the study area. Considering the positive association among legal restrictions on a drug, its availability, and substance

use (Hawkins, Catalano, & Miller, 1992), cannabis might not be an easily available option for unemployed young adults.

Having longitudinal data allows us a) to add observed time-dynamic confounding factors to typical fixed-effects models and then b) to take into account possible fluctuation in the association between unemployment and substance use over the course of young adulthood (Reine, Novo, & Hammarstrom, 2004). Participants' mental health, depression and anxiety in particular, was the primary focus of time-dynamic observed confounding factors in the present analysis, considering its high comorbidity with problematic substance use (Hasin et al., 2007; Kessler et al., 2005). The central research questions addressed are: 1) Is unemployment associated with alcohol use disorder and cannabis dependence, even after taking into account unobserved time-invariant fixed sources of confounding and observed time-dynamic covariate factors?; and 2) What patterns in directions of causality do emerge? Do they support the possibility of social causation and/or the possibility of social selection?

2. Methods

2.1. Participants

Data were gathered during the course of the Christchurch Health and Development Study, which features a birth cohort of 1,265 children (635 boys, 630 girls) born in the Christchurch (New Zealand) urban region in mid-1977. The cohort has been studied at birth, 4 months, 1 year, annually to age 16, and at ages 18, 21, 25, 30, and 35 (Fergusson & Horwood, 2001; Fergusson et al., 1989). All study information was collected on the basis of signed consent from study participants, is confidential, and was approved by the Canterbury (New Zealand) Ethics Committee.

2.2. Unemployment

At each assessment at ages 21, 25, 30, and 35 years, cohort members were asked a series of questions concerning their history and patterns of employment and unemployment since the previous

assessment. One set of questions examined whether cohort members had been unemployed and looking for work for 3 or more months during any calendar year since the previous assessment. For the purposes of the present study, this information was used to classify participants during each assessment period (ages 18–21, 21–25, 25–30, and 30–35 years) as to whether they had been unemployed for three or more months during any calendar year since the previous assessment.

2.3. Alcohol use disorder (AUD)

At ages 21, 25, 30, and 35 years, participants were interviewed concerning alcohol use and problems related to 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 an alcohol use disorder (alcohol abuse or dependence; AUD). For the present study, this information was used to classify participants as to whether they met DSM criteria for an AUD during any assessment period (ages 18–21, 21–25, 25–30, and 30–35 years).

2.4. Cannabis dependence

At ages 21, 25, 30, and 35 years, participants were interviewed concerning their use of cannabis and symptoms of cannabis dependence since the previous assessment using components of the CIDI (World Health Organization, 1993) to assess DSM-IV (American Psychiatric Association, 1994) symptom criteria for cannabis dependence. For the present study, this information was used to classify participants as to whether they met DSM criteria for cannabis dependence during each assessment period (ages 18–21, 21–25, 25–30, and 30–35 years).

2.5. Time-dynamic covariate factors (major depression and anxiety disorders)

2.5.1 Major depression and anxiety disorders.

To control for any possible effects of correlated mental health disorders, time-dynamic measures of DSM-IV (American Psychiatric Association, 1994) major depression and anxiety disorders were used. At ages 21, 25, 30, and 35 years, participants were questioned regarding symptoms of major depression and a range of anxiety disorders (including generalized anxiety disorder, panic disorder, agoraphobia, social phobia, and specific phobia) using CIDI (World Health Organization, 1993) items and DSM-IV (American Psychiatric Association, 1994) diagnostic criteria. Sample members who met DSM diagnostic criteria for a major depressive episode or one or more anxiety disorders at any time during any assessment period (18–21, 21–25, 25–30, and 30–35 years) were classified using a pair of dichotomous measures as having major depression or anxiety disorder during that period.

2.5.2 Stressful life events.

Also, to control for any possible effects of stressful life events in linking unemployment, cannabis dependence and AUD, time-dynamic measures of stressful life events were used. 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, 25-30, and 30-35 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; 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 & Wohlfarth, 1991). Using this information, a measures of exposure to stressful life events was created, 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, 25-30, and 30-35 years.

2.6. Statistical analyses

2.6.1. Associations between unemployment and alcohol use disorder and cannabis dependence

In the first stage of the analyses, the pooled associations between unemployment at ages 18-21, 21-25, 25-30, and 30-35 years and both: a) AUD; and b) cannabis dependence; were estimated using Generalized Estimating Equation methods (Liang & Zeger, 1986; Zeger & Liang, 1986). One primary advantage of GEE models in the analysis of longitudinal data is that these models are flexible in terms of being able to employ a range of outcome measures with varying distributional properties (Gibbons, Hedeker, & DuToit, 2010). The GEE approach has been used extensively with data from the present cohort (e.g. Boden, Fergusson, & Horwood, 2008, 2013; Fergusson, Boden, & Horwood, 2013).

In order to begin to test the social causation hypothesis (unemployment causing substance use disorder), two population-averaged logistic regression models were fitted in which the risk of each outcome (AUD; cannabis dependence) for each assessment period was modelled as a function of unemployment (3+ months) during each assessment period. These models were of the form:

$$f(Y_{it}) = B_0 + B_1 X_{it} \qquad \text{(EQ1)}$$

where $f(Y_{it})$ was the log odds for each outcome reported by the ith subject in a given time period t and X_{it} represented unemployment exposure (less than three months; three or more months) during the time period t. In these models observations from the same individual over time were permitted to be correlated with an unstructured correlation matrix. From the fitted models, estimates of the odds ratio (OR) and 95% confidence intervals (CI) of unemployment on outcomes were calculated. In addition, these models were extended to include further terms representing a time period x unemployment interaction, in order to account for the possibility that the strength of association between unemployment and the two substance use disorder outcomes varied over time.

Then, in order to begin to address the social selection hypothesis, in the next step of the analyses, two further GEE models were fitted in which unemployment (of three or more months' duration) was the outcome measure (for which $f(Y_{it})$ was the log odds for unemployment each outcome reported by the ith subject in a given time period t), and in which X_{it} represented either: a) AUD; or b)

cannabis dependence; were the predictor of interest. These models were also augmented with either a time period x AUD interaction term, or a time period x cannabis dependence interaction term. All other aspects of the modelling remained the same as those noted above.

2.6.2. Fixed-effects model for covariate adjustment

As noted above, one key threat to validity in testing causal associations using correlational data is the possibility of uncontrolled confounding. In the present analyses, it is important to note that the four GEE models described above do not take into account possible non-observed fixed effects and observed time dynamic covariate factors that may influence the associations between unemployment and substance use disorders. To address this issue, a series of three conditional fixed effects logistic regression models were fitted to the joint data for each of the outcomes over the measurement periods. Each model used a dichotomous measure as the outcome measure (unemployment of three or more months' duration; AUD; cannabis dependence), with the other two measures of interest entered into the model as simultaneous exposures. For example, in the model using unemployment as the outcome measure, both AUD and cannabis dependence were entered as exposures. The three models employed in the present investigation were of the form:

$$f(Y_{it}) = \alpha_i + B_1 X_{it} + \Sigma B_k Z_{ikt} + \Sigma B_k Z_{ikt-1} \qquad \text{(EQ6)}$$

In this model α_i were individual-specific terms that are assumed to reflect the effects of all fixed sources of variation in the outcome Y_{it}. The fixed effects α_i were assumed to be constant over time and to be correlated with other predictors in the model. The models were also augmented by the terms Z_{ikt} , representing the set of concurrent observed time-dynamic covariates (major depression; anxiety disorder; stressful life events); and the terms $\Sigma B_k Z_{ikt-1}$, represented time period-lagged measures of AUD, cannabis dependence, and unemployment, in order to control for auto-regressive covariation. All covariate factors were entered into the models simultaneously. Finally, the models were also extended to include terms representing time period x exposure interaction.

2.7. Sample size and sample bias

The present analyses were based on samples ranging from 1011 at age 21, 1004 at age 25, 987 at age 30, and 962 at age 35 representing 79% to 82% of the surviving cohort at each age, for whom data were available concerning unemployment, alcohol use, and cannabis use were available at the assessments at ages 21, 25, 30, and 35. To examine the effects of sample losses on the representativeness of the sample, the obtained samples with complete data at each age, were compared with the remaining sample members on a series of socio-demographic measures collected at birth. This analysis suggested that there were statistically significant ($p<.01$) tendencies for the obtained samples to under-represent individuals from socially disadvantaged backgrounds characterized by low parental education, low socio-economic status and single parenthood. To address this issue, the data weighting methods described by Carlin et al. (1999) were used to examine the possible implications of selection effects arising from the pattern of missing data. These analyses produced essentially the same pattern of results to those reported here, suggesting that the conclusions of this study were unlikely to have been influenced by selection bias.

3. Results

3.1. Associations of unemployment with alcohol use disorder and cannabis dependence

Tables 1a and 1b show the cohort classified into two groups based on levels of unemployment during each assessment period (ages 18–21, 21–25, 25–30, and 30–35 years). For each level of unemployment, the tables report on the percentage of individuals who meet DSM criteria for alcohol use disorder (AUD; Table 1a) and cannabis dependence (Table 1b). Finally, the tables report on the rate of AUD and cannabis dependence for each level of unemployment, pooled over the assessment periods, and estimates of the odds ratios (OR) and 95% confidence intervals (CI) for the association between unemployment and both AUD and cannabis dependence, derived from GEE modelling (see Methods section). The Tables show that:

1. For both AUD and cannabis dependence, there were clear and statistically significant ($p < .0001$) associations with unemployment. Estimates of the OR, pooled over the four assessment periods, show that participants with three or more months of unemployment had odds of AUD that were 1.49 (95% CI: 1.18, 1.88) times higher and odds of cannabis dependence that were 3.57 (95% CI: 2.55, 4.97) times higher than those who were not unemployed for three or more months.
2. Tests of the age-by-unemployment interaction showed a statistically significant ($p < .05$) interaction between age and unemployment in predicting AUD, whereas a similar test in predicting cannabis dependence was not statistically significant ($p > .40$). The results of this analysis suggest that the strength of association between unemployment and AUD decreased as participants got older.

[INSERT TABLES 1A AND 1B HERE]

3.2. Associations of AUD and cannabis dependence with unemployment

To examine possible reverse causal effects in which substance use increased the risk of unemployment, the cohort was classified into two groups based on (a) AUD; and (b) cannabis dependence. These classifications are shown in Tables 2a and 2b, which display the percentage of cohort members reporting at least 3 months of unemployment during each assessment period (ages 18–21, 21–25, 25–30, and 30–35 years). Finally, the tables report on the rate of unemployment for each level of AUD and cannabis dependence, pooled over the assessment periods, and estimates of the ORs and 95% CIs for the association between AUD, cannabis dependence and unemployment, derived from GEE modelling (see Methods section). The Tables show that:

1. There were statistically significant ($p < .0001$) associations between unemployment (3 months or more) and both AUD and cannabis dependence. These findings are summarized by the ORs presented at the foot of each table. Individuals meeting criteria for AUD had odds of unemployment that were 1.60 (95% CI: 1.26, 2.04) times higher than those who had no symptoms of alcohol use disorder. Similarly, those meeting criteria for cannabis dependence had odds of unemployment that were 3.60 (95% CI: 2.54, 5.12) times higher than those who did not use cannabis.

2. A test of the age-by-cannabis dependence interaction showed statistically non-significant ($p > .20$) interaction between age and cannabis dependence, suggesting that the association between cannabis use and unemployment did not vary according to age. A test of the age-by-AUD interaction showed a marginally significant ($p < .10$) interaction between age and AUD, suggesting that the association between AUD and unemployment varied marginally according to age.

[INSERT TABLES 2A AND 2B HERE]

3.3. Adjustment for unobserved fixed effects, time-dynamic covariate factors, and lagged measures

As noted in the Methods section, to examine the possible causal role of substance use in increasing the risk of unemployment, and of unemployment in increasing the risk of substance use disorder, three fixed-effects logistic GEE models were fitted to the pooled data from the four assessment periods (ages 18–21, 21–25, 25–30, and 30–35 years). In all cases, the outcome measure was fitted as a function of the exposures of interest, time-dynamic covariate factors, and lagged measures. Table 3a shows the parameter estimates for the final fitted model of AUD, Table 3b shows the parameter estimates for the final fitted model of cannabis dependence, and Table 3c shows the parameter estimates for the final fitted model of unemployment. The Tables show that:

1. Unemployment was a statistically significant ($p < .01$) predictor of AUD during the period of 18 to 35 years. Estimates of adjusted ORs indicated that cohort members who had been unemployed for at least 3 months at some point during the period had odds of alcohol use disorder that were 1.41 (95% CI: 1.14, 1.76) times higher than those who had not been unemployed (Table 3a). In addition, cannabis dependence was also a statistically significant ($p < .0001$) predictor of AUD.
2. Unemployment was also a statistically significant ($p < .0001$) predictor of cannabis dependence. Estimates of the adjusted OR indicated that cohort members who had been unemployed for at least 3 months in a given year between ages 18–35 had odds of cannabis dependence that were 2.87 (95% CI: 2.00, 4.11) times higher than those who had not been unemployed (Table 3b). Also of note was that AUD was a statistically significant ($p < .0001$) predictor of cannabis dependence.

3. Both cannabis dependence ($p < .0001$) and AUD ($p < .01$) were significant predictors of unemployment during the period of 18 to 35 years. Estimates of adjusted ORs indicated that cohort members who were classified as cannabis dependent at some point during the period had odds of unemployment (3 months or more) that were 2.83 (95% CI: 1.98, 4.02) times higher than those who were not cannabis dependent. Cohort members who met criteria for an AUD had odds of unemployment that were 1.40 (95% CI: 1.12, 1.77) times higher than those who did not meet criteria for an AUD (Table 3c). A Wald chi-square comparison between cannabis dependence and AUD indicated that cannabis dependence was a significantly stronger predictor of unemployment than AUD ($X^2(1) = 9.44$, $p < .01$).

The results of these analyses suggest the possibility of reverse causal processes in the association between unemployment and both cannabis dependence and AUD, in which unemployment increases the risk of both cannabis dependence and AUD, and where both cannabis dependence and AUD increase the risk of being unemployed. In addition, there was further evidence to suggest that cannabis dependence was a stronger predictor of unemployment than AUD.

[INSERT TABLES 3A, 3B, AND 3C HERE]

4. Discussion

The present study used data from a prospective longitudinal study to evaluate the association between unemployment and substance use disorders (AUD; cannabis dependence) between ages 18 and 35. We employed a fixed-effects regression modelling strategy that was augmented by time-dynamic covariate factors in addition to time-fixed non-observed sources of confounding that fixed-effects regression models are designed to address.

Supporting the stress, frustration-aggression, and deprivation hypotheses and in line with prior studies (e.g., Redonnet et al., 2012), the analyses found consistent countercyclical associations between

unemployment status and both AUD and cannabis dependence. The experience of three or more months of unemployment resulted in increased odds of meeting the diagnostic thresholds for AUD and cannabis dependence. In our studies, no empirical evidence supported a procyclical association, by extension the income loss hypothesis, regarding unemployment status and two problematic substance use measures. Congruent with prior studies (McDonough & Berglund, 2003; Mossakowski, 2008), the current study finding suggests that the detrimental impact of an unfavourable change in one's socioeconomic status, such as unemployment, on substance use disorders might be exacerbated as unemployment becomes prolonged and chronic. A question remains, however, why such countercyclical associations exist and which specific intervening factors are critical explaining the associations in the current study. Future research that explicitly tests relative importance of potential intervening factors proposed in relevant theories (e.g., increased level of frustration, disrupted time structure) will be particularly fruitful and needed.

Results from the augmented fixed-effects regression models suggest that the linkages between unemployment and substance use disorder remained robust, even after adjusting for sources of time-invariant non-observed confounders and time-dynamic covariates. After the adjustment, participants who had been unemployed for more than three months at some point had odds of AUD that were 1.41 times higher than those who had been more consistently employed. A stronger impact was observed regarding cannabis dependence; individuals who had been unemployed for more than three months had odds of cannabis dependence that were 2.87 times higher than those who had been consistently employed. Of note, these study findings regarding cannabis dependence appear to be in contrast to prior studies reporting null findings regarding the effects of unemployment on cannabis use (Lee et al., 2015) or abuse (Melchior et al., 2015). However, these prior analyses (Lee et al., 2015; Melchior et al., 2015) operationalized cannabis use measures differently and were conducted in social and economic systems that are different than the present study—the study by Lee et al. (2015) occurred in the United States and the study by Melchior et al. (2015) occurred in France. The differences in findings may be related to differences in broader contexts and specific study design components.

The findings of the present analysis also provide support for the social selection argument. After adjustment, there was evidence that both cannabis dependence and alcohol use disorder were significantly associated with an increased risk of unemployment. Following adjustment for both non-observed fixed effects and time-dynamic covariate factors, those cohort members with cannabis dependence had odds of unemployment that were 2.83 times higher than those who were not cannabis dependent. Furthermore, those cohort members meeting criteria for an AUD had odds of unemployment that were 1.40 times higher than those who did not meet criteria for an AUD. Taken together, the results of the present study suggest the possibility of reverse causal processes in the association between unemployment and substance use, in which social causation and social selection processes are both at work.

It should also be noted that the present data suggest that the possible reverse causality between substance use disorders and unemployment might be more evident with regard to cannabis use than alcohol use. In the models of unemployment predicted by substance use disorder, both cannabis dependence and AUD were statistically significant predictors of unemployment in both unadjusted and adjusted models. However, cannabis dependence appeared to exert a bigger effect than AUD. This difference in findings between these two substances might stem from their varying status with regard to laws governing substance use in the study area. The possession and use of cannabis is illegal in New Zealand. As such, a different etiology, such as early involvement in criminal activities, might underlie its use and pathological manifestation, and thus a causal mechanism between unemployment and cannabis use might be different than that related to alcohol. It may be fruitful for future studies to test the possibility of reverse causality in regions where the use and possession of small amounts of cannabis has been legalized for adults. Irrespective of this, however, the present data show evidence supporting both the social causation and social selection arguments of the associations between unemployment and substance use.

Of interest in the adjusted analyses (Tables 3a-3c) was the consistent finding that the time-dynamic variable representing stressful life events were significantly ($p < .05$) associated with all three

outcomes in the fully adjusted models. While an in-depth examination of the associations between unemployment, life stress and substance use disorders is beyond the scope of the present investigation, it should be noted that a previous paper using CHDS data found evidence of a causal role of stressful life events in alcohol use disorder (Boden et al, 2014). Further research is needed to disentangle the potentially complex association between unemployment, general life stress, and substance use disorders.

Study findings should be interpreted in the context of a few methodological limitations. First, all the measures were based on individuals' self-reports, which are subject to possible response bias. Second, the cohort members were exposed to the most recent global recession in 2008, which may have influenced the linkage between unemployment and pathological substance use. Disentangling the effect of unemployment from the effect of recession at a macro level using multiple data points after the most recent recession may be a fruitful direction for future research. Third, although our analyses addressed sources of time-invariant unobserved and time-dynamic observed confounders and then directly assessed the possible reverse causality in the linkage of focus, possible simultaneous causation of these variables was not explicitly addressed. Finally, the Christchurch Health and Development Study features a regional community sample and thus interpretation of the study findings should be carried out with caution.

Our study makes two important contributions to the existing literature on the linkage between unemployment and pathological substance use. First, it contributes to the decades-long debate about social causation and social selection regarding the association of focus by capitalizing on a rich trove of longitudinal data and controlling for both time-variant and time-invariant confounding factors. Second, the current study advances understanding of young adulthood, which represents a particularly important period featuring concentrated vulnerability to unemployment (Edwards & Hertel-Fernandez, 2010; Taylor et al., 2012) and substance use (Johnston et al., 2014; Melchior et al., 2015; Substance Abuse and Mental Health Services Administration, 2009). To our knowledge, no prior longitudinal study has incorporated all of these strengths.

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Contributors: JMB, JOL, LJH, and GFHM conceptualized the study; JMB and LJH collected and analysed the data; and JMB, JOL, CVG, LJH, and GFHM wrote and edited the manuscript.

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Table 1a. Associations between unemployment and rates of alcohol use disorder, ages 18-21, 21-25, 25-30, and 30-35 years.

	Unemployment		
	< 3 months	3+ months	p
% AUD (ages 18-21)	24.2	42.1	
n	759	252	
% AUD (ages 21-25)	19.3	32.5	
N	809	191	
% AUD (ages 25-30)	13.5	17.1	
N	917	70	
% AUD (ages 30-35)	14.4	18.0	
n	873	89	
Pooled %	17.6	32.6	
OR (95% CI)	1 - -	1.49 (1.18-1.88)	<.0001

Test of age * unemployment interaction:

$\chi^2(1)=4.49, p<.05$

Table 1b. Associations between unemployment and rates of cannabis dependence, ages 18-21, 21-25, 25-30, and 30-35 years.

	Unemployment		
	<3 months	3+ months	p
% cannabis dependence (ages 18-21)	4.0	15.9	
n	759	252	
% cannabis dependence (ages 21-25)	4.0	17.3	
N	809	191	
% cannabis dependence (ages 25-30)	3.2	12.9	
N	917	70	
% cannabis dependence (ages 30-35)	2.6	9.0	
n	873	89	
Pooled %	3.4	15.0	
OR (95% CI)	1 - -	3.57 (2.55-4.97)	<.0001

Test of age X unemployment interaction:

$\chi^2(1)=0.63, p>.40$

Table 2a. Associations between AUD and rates of unemployment (3+ months), ages 18-21, 21-25, 25-30, and 30-35 years.

	AUD classification		
	Did not meet criteria	AUD	p
% unemployed 3+ months (ages 18-21)	20.2	36.6	
n	721	290	
% unemployed 3+ months (ages 21-25)	16.5	28.4	
N	782	218	
% unemployed 3+ months (ages 25-30)	6.8	8.8	
N	851	136	
% unemployed 3+ months (ages 30-35)	8.9	11.3	
n	820	142	
Pooled %	12.8	24.9	
OR (95% CI)	1 - -	1.60 (1.26-2.04)	<.0001

Test of age * AUD level interaction:

$\chi^2(1)=3.47$, $p<.10$

Table 2b. Associations between cannabis use and rates of unemployment (3+ months), ages 18-21, 21-25, 25-30, and 30-35 years.

	Cannabis dependence classification		p
	Did not meet criteria	Cannabis dependent	
% unemployed 3+ months (ages 18-21)	22.5	57.1	
N	941	70	
% unemployed 3+ months (ages 21-25)	16.9	50.8	
n	935	65	
% unemployed 3+ months (ages 25-30)	6.4	23.7	
n	949	38	
% unemployed 3+ months (ages 30-35)	8.7	26.7	
n	931	30	
Pooled %	13.6	44.3	
OR (95% CI)	1 - -	3.60 (2.54-5.12)	<.0001

Test of age * cannabis dependence interaction:

$\chi^2(1)=1.15, p > .20$

Table 3a. Parameter estimates for the association between alcohol use disorder, unemployment and cannabis dependence (ages 18-35) after adjustment for fixed effects, time-dynamic covariate factors, and lagged measures.

	AUD		
Predictor	β	SE	p
Unemployment	.35	.11	<.01
Cannabis dependence	.78	.17	<.0001
Major depression	.17	.11	>.10
Anxiety disorder	-.00	.12	>.90
Life stress	.02	.00	<.0001
Lagged measure of AUD	.64	.11	<.0001
Lagged measure of unemployment	.02	.06	>.60
Lagged measure of cannabis dependence	.22	.17	>.20

Table 3b. Parameter estimates for the association between cannabis dependence, unemployment and AUD (ages 18-35) after adjustment for fixed effects, time-dynamic covariate factors, and lagged measures.

	Cannabis dependence		
Predictor	β	SE	p
Unemployment	1.05	.18	<.0001
AUD	.88	.18	<.0001
Major depression	.46	.19	<.05
Anxiety disorder	-.10	.22	>.60
Life stress	.03	.01	<.0001
Lagged measure of cannabis dependence	1.86	.21	<.0001
Lagged measure of unemployment	.29	.10	<.01
Lagged measure of AUD	.18	.19	>.30

Table 3c. Parameter estimates for the association between unemployment, alcohol and cannabis (ages 18-35) after adjustment for fixed effects, time-dynamic covariate factors, and lagged measures.

	Unemployment (3+ months)		
Predictor	β	SE	p
AUD	.34	.11	<.01
Cannabis dependence	1.04	.18	<.0001
Major depression	.32	.12	<.01

Anxiety disorder	-.12	.14	>.40
Life stress	.01	.00	<.01
Lagged measure of unemployment	.53	.06	<.0001
Lagged measure of AUD	-.02	.13	>.80
Lagged measure of cannabis dependence	.04	.19	>.80