The impact of tobacco use on treatment for comorbid depression and alcohol misuse

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The impact of tobacco smoking on treatment for comorbid depression and alcohol misuse.

ABSTRACT

Background: Tobacco use is a major public health concern, and is associated with a number of mental illnesses as well as increased alcohol/other drug (AOD). Research into treatment for individuals experiencing such comorbidities is limited.

Design and Methods: Participants (n=447) were those enrolled in the Depression and Alcohol Integrated and Single-focused Interventions project (Baker et al., 2010), and the Self Help for Alcohol/other drugs and DEpression project (Kay-Lambkin, Baker, Kelly, & Lewin, 2011), who reported current depression and hazardous alcohol use at entry to the study. Smoking cessation was not targeted in, nor a goal of, treatment.

Results: After controlling for socioeconomic variables, tobacco use was not associated with higher levels of depressive symptoms at baseline; however heavy smokers (30+ cigarettes per day) consumed significantly more alcohol at baseline than did non-smokers (13 vs. 9 standard drinks per day). Baseline smoking severity did not impact on depression or alcohol use outcomes over a 12-month period. Reductions in tobacco use between baseline and 3-month follow-up were significantly associated with reductions in depression and alcohol consumption over the same time period.

Discussion and Conclusions: The study results suggest that tobacco use does not interfere with treatment for depression and alcohol use problems, and adds weight to the idea of considering specific treatment for tobacco use in the context of treatment for alcohol/other drug use.

Keywords: tobacco, depression, alcohol-related disorders, therapy

In the most recent survey into alcohol/other drug (AOD) use conducted by the Australian Institute of Health and Welfare, 19.4% of adults were current smokers, with 16.6% reporting regular daily tobacco smoking (AIHW, 2008). Younger Australians are significantly more likely to smoke tobacco, with peak tobacco smoking occurring in the 20-29 years age group, and males for the most part reporting the highest rates of daily smoking across the lifespan (AIHW, 2008). Smoking is also associated with socioeconomic status, with those from disadvantaged backgrounds being more likely to smoke, and additional links are found between smoking and geographical region as well as country of birth. People with mental health and AOD use problems are also among those groups reporting significantly higher rates of tobacco smoking than the general population (A. L. Baker et al., 2006).

Depression and smoking

In particular, the association between depression and cigarette smoking is of concern. Aside from the increased cardiovascular risk associated with both depression and tobacco smoking, compared to non-smokers, regular smokers report more frequent and severe episodes of depression, and higher suicide ideation and rates of suicide (K. Wilhelm, Wedgewood, Niven, & Kay-Lambkin, 2006). Smoking remains one of the strongest correlates of current Major Depressive Disorder (K. Wilhelm, Mitchell, Slade, Brownhill, & Andrews, 2003). Rates and severity of nicotine dependence are higher amongst those with depression, and negative affect is the most commonly cited reason for unsuccessful smoking cessation attempts (K. Wilhelm et al., 2006).

Alcohol and other drug use and smoking

AOD use has also been suggested to occur at higher rates in smokers than non-smokers (Cargill, Emmons, Kahler, & Brown, 2001). Over one-fifth of current smokers in Australia report concurrent risky consumption of alcohol, compared with 16% of ex-smokers and 8% of never smokers (ABS, 2006). There is some suggestion that tobacco smoking among AOD treatment populations is as high as 100%, with a strong relationship observed between tobacco smoking and other substance use such as cannabis, heroin and cocaine (A. L. Baker et al., 2006).

The extent of the connection between tobacco smoking and alcohol dependence is of particular note, with reports in the USA of over 80% of adolescents being treated for alcohol use disorders also smoking tobacco (K. Shelef, G. Diamond, G. Diamond, & M. Myers, 2009a). This is in comparison to approximately 25% in the general US population who report tobacco smoking alone (Dunn et al., 2010). Nicotine dependence is also more severe in individuals who drink alcohol as opposed to those who are abstinent (Cooney et al., 2007), and smoking among people who are dependent on alcohol has been shown to be associated with deaths than alcohol consumption alone (Hitsman et al., 2002).

Comorbid smoking, depression and alcohol misuse

At present, there is only a modest understanding of the interaction between depression, alcohol and tobacco smoking, and even less on what an appropriate treatment for this particular type of comorbidity might comprise (Jane-Llopis & Matytsina, 2006).

There are, however, potential difficulties in examining these issues in real world settings. For example, in psychiatric and AOD treatment settings, smoking tobacco is often viewed as a lesser evil and is ignored, perhaps at times encouraged (A. L. Baker et al., 2006), particularly in the context of poly-substance use treatment programs (Kalman, Kim, DiGirolamo, Smelson, & Ziedoni, 2010). Other concerns include the fear that a quit attempt might result in poorer mental health or AOD treatment outcomes. However, research now suggests that between 46-80% of participants in drug treatment wish to quit smoking (A. Baker et al., 2006). Other studies suggest that making a quit attempt in alcohol treatment did not negatively impact on treatment outcome (Kahler et al., 2008).

Given the limited understanding of the comorbidities of depression, alcohol and tobacco use, effective treatment for this subgroup remains a cloudy issue. Evidence based treatment for depression has been well documented (APA, 2000), as has treatment for substance use (APA, 1995), and smoking cessation (APA, 1996), with cognitive behavior therapy being a treatment of choice for each of these conditions occurring in isolation. Despite this, little evidence exists to suggest how well these treatment strategies generalize to people experiencing these conditions as they co-occur.

Comorbidity research conducted with other co-occurring disorders has investigated the changes in a secondary disorder following treatment targeting a primary disorder, exploring the generalisability of the treatment modalities utilized. For

example, CBT and other treatment strategies such as enhancing motivation, identifying triggers, learning refusal skills, and improving emotional coping skills, are areas of common ground in the treatment of substance use disorders and tobacco use (Shelef et al., 2009a). A sense of achievement in the reduction of one substance may also have a carryover effect in the reduction in the use of a secondary substance (Shelef et al., 2009a). While promising, the ability of people with comorbidity to generalize skills learned in treatment to other behaviors and issues not targeted in that treatment program has not yet been tested (Alati et al., 2004; K. Shelef, G. S. Diamond, G. M. Diamond, & M. Myers, 2009b). The current study has been designed to address this gap.

The current study

We have previously conducted two randomized controlled trials (Baker et al., 2010; Kay-Lambkin, Baker, Kelly, et al., 2011) comparing brief (1-session advice and motivational interviewing, MI) and extended (10 sessions MI/cognitive behavior therapy, CBT) interventions for comorbid depression and AOD use problems; the first large-scale randomized controlled trials of CBT for comorbid depression and AOD use problems. These studies also present the opportunity to explore the prevalence of smoking amongst this important comorbid population, and examine the impact on smoking, if any, of CBT-focused treatments for depression and AOD use problems, and vice versa. The present paper reports findings from a combination of datasets from these studies for people who met study entry criteria for at least hazardous use of alcohol and moderate depressive symptomatology. Specifically, it is hypothesized that:

(a) At baseline, heavy smokers will report higher levels of depression and alcohol use than will non-smokers;

(b) Heavy smokers will have poorer outcomes on measures of depression and alcohol use than non-smokers; and

(c) Significant reductions in smoking tobacco will be observed from pre- to post-treatment and that these reductions will be moderated by treatment condition.

METHODS

Participants

Participants were those enrolled in the Depression and Alcohol Integrated and Single-focused Interventions project (DAISI, Baker et al., 2010), and the Self Help for Alcohol/other drugs and DEpression project (SHADE, Kay-Lambkin, Baker, Kelly, et al., 2011), which commenced in 2006 and 2005 respectively. To be included in the current study, eligible participants were those with current depressive symptoms (≥ 17 on the Beck Depression Inventory II, BDI-II, Beck, Steer, & Brown, 1996) and concurrent use of alcohol in excess of recommended national guidelines in Australia at the time of the study (NHMRC, 2009), which equated to consumption above an average of four 10gm ethanol drinks per day for men, or two per day for women. Individuals who exhibited psychotic symptoms, those requiring medical detoxification, who had learning difficulties, were non-English speaking or who had organic brain disease at baseline were excluded. Participants could be using a range of other drugs, including tobacco, at entry to the study.

Assessment Measures

All assessment measures are widely used in mental health and AOD clinical and research settings, and were common to both the SHADE and DAISI projects and to all assessment timepoints. The assessments included:

(a) Demographics and treatment history: including age, gender, employment and education status.

(b) Temporal primacy of depression or alcohol use disorder (primacy): a timeline of depressive symptoms and alcohol consumption over the person's lifetime was completed to determine whether the person's first occurrence of symptoms of depression were prior or subsequent to the onset of regular alcohol use.

(c) Structured Clinical Interview for DSM-IV, Research Version (SCID-RV, First, 2001): provided a diagnostic, clinician-rated measure of depression and alcohol abuse and dependence.

(d) BDI-II (Beck et al., 1996): a 21-item self-report questionnaire used to screen for the presence of depressive symptoms over the previous two-week period.

(e) Opiate Treatment Index (OTI, Darke, Hall, Wodak, Heather, & Ward, 1992): addresses the quantity and frequency of AOD use over the month prior to assessment, three of which are relevant to the current study; alcohol, cannabis and cigarette use. For tobacco smoking, participants recalled their three most recent tobacco-smoking occasions, and estimated the number of cigarettes smoked on each of those occasions. A score was calculated based on these reports, summarizing the quantity and frequency of tobacco smoking for those use occasions falling in the same month prior to baseline. Each occasion of use equated to one day, and so the resultant score is essentially equivalent to cigarettes per day.

(f) Global Assessment of Functioning Scale (GAF, APA, 1994): provides an index of overall functioning incorporating the domains of psychological, social and occupational status.

(g) Brief Symptom Inventory (BSI, Derogatis & Melisaratos, 1983): is a self-report measure of psychopathology and general stress. The following domains of distress were included: somatization, anxiety, and phobic anxiety.

Procedures

Detailed description of the study procedures have been published for the SHADE (Kay-Lambkin, Baker, Kelly, et al., 2011) and DAISI (Baker et al., 2010) studies elsewhere. For both studies, following the provision of informed consent, eligible participants completed a baseline clinical interview with a Registered or Intern Psychologist, usually over two one-hour sessions, one week apart. All participants then received an initial session comprising advice and MI focused on depression and alcohol misuse, conducted face-to-face with a Registered or Intern Psychologist. At the conclusion of this session, participants were randomized into no further treatment (Brief Intervention (BI) condition), or conditions that ensued over the following 9 weeks. Regardless of treatment attendance, all participants were eligible for follow-up assessment, which occurred at 3-, 6-, and 12-months post-baseline, and were conducted by a blind assessor independent from the treatment phase of the projects.

The DAISI project employed four psychological treatment programs of combination MI and CBT that were conducted face-to-face with trained Psychologists:

(a) 1 60-minute integrated BI that addressed depression and alcohol use concurrently;

(b) 10 60-minute sessions of MI/CBT that targeted alcohol use, including the BI as session 1 (Alc);

(c) 10 60-minute sessions of MI/CBT that targeted depression, with the BI as session 1 (Dep); and

(d) 10 60-minute sessions of MI/CBT that addressed depression and alcohol use concurrently, with the BI as session 1 (Int).

The SHADE project examined the efficacy of three psychological treatment conditions, delivered over a 10-week period, each with a 60-minute face-to-face BI as session 1.   These treatments were:

(a) 10 60-minute sessions of MI/CBT targeting depression and alcohol use concurrently, with the BI as session1 and delivered face-to-face with a trained Psychologist (Int);

(b) 10 60-minute sessions of MI/CBT targeting depression and alcohol use concurrently, with the face-to-face BI as session 1, and ensuing sessions delivered by the SHADE computer program with weekly 15-minutes compliance checking face-to-face with a therapist (SHADE); and

(c) 10 60-minute sessions of Person Centered Therapy (PCT), with the BI as session 1, delivered face-to-face by a trained Psychologist, and limited to supportive counseling techniques with no MI/CBT strategies.

Across the SHADE and DAISI projects, the BI was identical in content and delivery mode, as was the 10-session integrated treatment program that used MI/CBT

targeting both depression and alcohol use concurrently and was delivered face-to-face by a trained therapist.

The protocols for the SHADE and DAISI projects were approved by a suitably constituted Ethics Committee of the institution within which the work was undertaken and conformed to the provisions of the Declaration of Helsinki (as revised in Tokyo 2004).

Statistical Analysis

Basic demographics were examined for the sample as a whole.

All participants received a categorization according to the severity of their consumption of tobacco at baseline assessment. The categories were based on previous research (Alati et al., 2004; Shelef et al., 2009b) and included: non-smoker (0 cigarettes per day, cpd), light (1-9 cpd), moderate (10-19 cpd) and heavy (20+ cpd). The relationship between this new smoking variable and basic demographics was examined using oneway Analysis of Variance (ANOVA) for continuous variables and chi square analysis for categorical variables.   Relevant presenting symptoms (depression alcohol consumption, anxiety, cannabis use, general functioning) were also explored for associations with smoking category using oneway ANOVA, with Bonferroni posthoc analysis used to determine the nature of any differences in these variables according to smoking category.

<u>Hypothesis (a): that heavy smokers will have higher levels of depression and alcohol use than non-smokers</u>

Analysis of CoVariance (ANCOVA) compared the newly created smoking variable with BDI-II and OTI (alcohol) scores at baseline, with all baseline variables examined above that showed a significant relationship with smoking entered in as covariates.

<u>Hypothesis (b): that heavy smokers will have poorer outcomes on measures of depression and alcohol use than non-smokers.</u>

Two repeated measures ANOVAs examined the impact of smoking category (non-smoker, light, moderate, heavy) on treatment outcomes over time (one for depression and one for alcohol use). No imputations for missing data were used, with participants providing data at each relevant timepoint included in the analysis. Baseline, 3-, 6- and 12-month BDI-II and alcohol consumption scores were included in the model, and significant baseline variables as above were included as covariates in the analysis.

<u>Hypothesis (c): that significant reductions in smoking tobacco will be observed from pre- to post-treatment and that these reductions will be moderated by treatment condition.</u>

Paired sampled t tests examined changes in tobacco consumption, based on OTI tobacco scores, between baseline and 3-months, baseline and 6-months and baseline and 12-months.

Change scores were created for the primary outcome variables (BDI-II and OTI alcohol scores) and OTI tobacco scores at three, six and twelve months relative to baseline, to examine the relationship between smoking and treatment. Positive scores on the change measures were indicative of decreases in tobacco, depression and alcohol use. Correlations were conducted between these variables to examine how changes in primary outcomes were associated with changes in smoking.

Repeated measures ANOVA examined changes in OTI tobacco scores over time (baseline, three, six and twelve months) as a function of treatment allocation (BI, Dep, Alc, Int, Comp, PCT) at each timepoint. No imputations for missing data were used, with participants providing data at each relevant timepoint included in the analysis. Baseline variables with a significant relationship to smoking were included as covariates.

RESULTS

Demographics.

Of the 447 participants in the current study at baseline, 340 (76%) completed the 3-month follow-up assessment, 342 (77%) the 6-month and 315 (70%) the 12-month follow-up assessment. Fifty eight percent (n=261) completed all three follow-up assessments. At baseline, 185 (41%) participants reported not smoking in the month prior to entry to the SHADE/DAISI studies, 63 (14%) were light smokers, 68 (15%) were moderate smokers and 131 (29%) were heavy smokers of tobacco. The mean age of the sample was 43.94 years (range 18-73). Sample characteristics are displayed in Table 1.

Significant associations were found between levels of smoking at baseline and age and education. Non-smokers were significantly older (age=47.13yrs) than were those who reported light (age=40.70, p<0.01), moderate (age=40.32, p<0.01) or heavy tobacco consumption (age=42.87, p<0.01). Heavy smokers reported significantly less involvement in education compared to light smokers, having left secondary schooling at a younger age (age=15.93 vs. 16.63yrs respectively, p<0.01). Non-smokers also left school at a significantly younger age than light smokers (age=16.09 vs.16.63, p=0.03). A significantly higher proportion of males than females were classified as heavy smokers (63% vs. 37%, χ^2_3=9.247, p=0.03). A significantly higher proportion of participants with an Independent Depressive Disorder were non-smokers, relative to those with Substance-Induced Depression (73% vs. 27%, χ^2_3=8.385, p=0.04).

Unemployed participants were significantly more likely to be light-heavy smokers (n=29-71, 47-55%) than non-smokers (n=42, 23%, p<0.01). Participants currently receiving government pensions were also significantly more likely to be light-heavy smokers (n=39-87, 63-66%) than non-smokers (n=74, 40%, χ^2_3= 27.24, p<0.01).

Associations between tobacco smoking and relevant presenting symptoms.
As can be seen in Table 2, there were significant Bonferroni corrected associations between severity of smoking and cannabis use, anxiety and general functioning.

Light and heavy smokers reported significantly higher levels of cannabis use at baseline than non-smokers. Anxiety symptoms were significantly higher among moderate and heavy smokers compared to non-smokers. Non-smokers displayed significantly higher levels of general functioning than moderate-heavy smokers.

Hypothesis (a): that heavy smokers will report higher depression and alcohol consumption at baseline.

ANCOVA compared BDI-II scores with smoking category, using age, gender, education, employment, primacy, mental health treatment, baseline cannabis use, baseline anxiety and baseline functioning as covariates. The ANOVA was not statistically significant ($F_{(3,204)}=0.935$, $p=0.425$). Significant covariates in the model were anxiety ($p<0.01$) and functioning ($p<0.01$).

The same ANCOVA model was used to examine the relationship between smoking category and alcohol consumption at baseline. The model was statistically significant ($F_{(3,205)}=3.900$, $p=0.010$), with simple contrast results indicating heavy smokers consumed significantly more alcohol than did non-smokers ($\underline{M}$(non-smokers)=9.104 standard drinks per day, $\underline{M}$(heavy smokers)=12.583 standard drinks per day, $p=0.011$). None of the covariates was significant in the model.

Hypothesis (b): that smokers will have poorer outcomes on measures of depression and alcohol use than non-smokers.

Repeated measures MANOVA, using the covariates of age, gender, education, employment, pension receipt, mental health treatment, baseline cannabis use, baseline anxiety and baseline functioning, examined depression outcomes over time according to smoking categorization at baseline. The MANOVA indicated a significant main effect of time on depression scores ($F_{(3,182)}=3.166$, $p=0.026$), with post-baseline depression scores at 3-, 6- and 12-months being 10.635, 11.656 and 12.083 BDI-II points respectively lower than baseline. However, the interaction between time, smoking status at baseline and depression was not statistically significant ($F_{(9,184)}=0.936$, $p=0.493$).

Using the same MANOVA model, there was no main effect of time on alcohol use ($F_{(3,185)}=1.470$, $p=0.224$), and no significant interaction between alcohol use over time and smoking status at baseline ($F_{(9,187)}=0.401$, $p=0.935$). The significant covariate in this model was education ($F_{(3,185)}=2.891$, $p=0.037$).

Hypothesis (c): that significant reductions in smoking tobacco will be observed from pre- to post-treatment, and that these reductions will be moderated by treatment condition.

Paired sample t tests revealed that no significant changes in tobacco use (OTI tobacco scores) occurred between baseline and 3-month follow-up (M(baseline)=10.559, M(3-months)=10.050, t=1.155, p=0.249, n=335), baseline and 6-months (M(baseline)=10.731, M(6-months)=10.594, t=0.255, p=0.799, n=338) and

baseline and 12-month ($\underline{M}$(baseline)=10.926, $\underline{M}$(12-months)=10.846, t=0.087, p=0.931, n=310) follow-up assessments.

Pearson correlations indicated a significant positive association between changes in smoking and changes in depression (r=0.14, p=0.01) between baseline and 3-months post-baseline assessments. The same was true for changes in tobacco and changes in alcohol use over the same time period (r=0.15, p<0.01). No significant relationships between changes in these three variables were found over the longer term.

Repeated measures MANOVA, with the following covariates: age, gender, employment, education, pension receipt, mental health treatment, baseline cannabis use, baseline anxiety scores, and baseline functioning, examined changes in smoking between baseline, 3-, 6- and 12-month follow-up according to treatment allocation. The ANOVA indicated that the main effect of time on smoking was not statistically significant (F(3,151)=0.245, p=0.865). None of the covariates was statistically significant, and the interaction between smoking over time and treatment allocation was not statistically significant (F(15,153)=1.714, p=0.135).

Figure 1 displays the changes in tobacco use over time, according to treatment allocation, adjusted for the covariates in the model. Participants in the integrated therapist condition reported a slight reduction of 1.53 cpd between baseline and 12-month follow-up assessment, as did those in the alcohol condition (1.49 cpd). Those in the SHADE computer condition reported a 3.62-cpd reduction over the same time period. No change in OTI tobacco scores was found for those in the BI (+0.34 cpd)

and Dep (+0.54 cpd) conditions. Participants in the PCT condition reported a 1.16-cpd increase between baseline and 12-month follow-up assessments, with a peak increase observed at 6 months post-baseline (4.29 cpd).

Insert Figure 1 about here

DISCUSSION

The aim of this study was to explore the impact of smoking tobacco on presenting symptoms and treatment outcomes for people enrolled in a trial of psychological treatment for comorbid depression and alcohol misuse. Contrary to prediction, after controlling for important socioeconomic factors relevant to smoking, heavy smokers did not report elevated levels of depression compared to non-smokers, however they did drink significantly more alcohol (13 versus 9 standard drinks per day). Smoking status at baseline did not affect depression or alcohol use outcomes over the course of the study, and tobacco use was not significantly reduced as a function of treatment for depression and alcohol misuse. These implications of these results are discussed in detail below.

Hypothesis (a): that heavy smokers will have higher levels of depression and alcohol use at baseline than will non-smokers.

We did not find an association between increased severity of depression or alcohol use at baseline and tobacco use severity. This may be due to the eligibility criteria of the study, with participants required to be experiencing at least moderate levels

of depression and hazardous use of alcohol for the month prior to baseline, thus decreasing the variability of depression and alcohol use within the sample as a whole. However, non-smokers generally reported lower levels anxiety, cannabis use and improved functioning compared to light, moderate and heavy smokers, and heavy smokers were significantly more likely to be unemployed, leave school at a younger age, and be receiving welfare payments. Light smokers reported significantly better levels of functioning and lower alcohol consumption than did moderate and heavy smokers, and generally lower anxiety, somewhat indicative of a dose-response relationship between the amount of tobacco smoked and increased severity of symptoms. This potentially illustrates the need for effective and evidence-based treatments options for smokers incorporating a wide range of intervention targets, and the potential harm reduction benefits of reduced tobacco smoking should this be a more realistic target.

The relationship between anxiety and tobacco use is worthy of mention. Our study found that moderate-heavy tobacco users at baseline reported significantly higher levels of somatization, anxiety and phobic anxiety than did their non-smoking counterparts. Alati and colleagues (2004) found an association in their study between the experience of anxiety and tobacco use, however there was no evidence of a dose-response relationship, with anxiety levels not differing across light, moderate or heavy smokers. We did not find a statistically significant dose-response relationship between anxiety levels and severity of tobacco consumption (with the exception of abstinence from tobacco being associated with significantly lower anxiety), however self-reported anxiety levels did increase as severity of smoking

increased. This finding implies that anxiety may be an important target for treatment amongst smokers of any level of severity.

As in the current study, previous studies have identified increased severity of smoking among males as compared to females (Grant, Hasin, Chou, Stinson, & Dawson, 2004). It has been suggested that the reward for smoking may be different between men and women, with women reporting more psychological rewards following smoking (e.g. relaxation) and men reporting more physiological rewards after smoking (Massak & Graham, 2008). Further, a range of socioeconomic variables, education, receiving welfare payments, and unemployment, were significantly associated with smoking severity at baseline. Smoking was heavier in those with substance-induced depression than for people with independent depressive disorders, as well as those engaged with mental health treatment services in the 12 months prior to baseline. These findings confirm the significance of smoking tobacco as not just a health, but also a social justice issue. There are also implications for the opportunistic delivery of smoking cessation interventions, particularly for people engaged with both substance use and mental health services.

Hypothesis (b): that heavy smokers will have poorer outcomes on measures of depression and alcohol use than non-smokers.

Contrary to prediction, although depressive symptomatology did significantly improve over the 12-month follow-up period, this was not moderated by baseline smoking levels. Similarly, alcohol use between baseline and 12-month follow-up was not significantly related to baseline smoking status. There is little existing literature

examining the impact of smoking levels on mental health or substance use treatment outcomes. Research investigating the impact of varying degrees of alcohol use on smoking cessation programs revealed that moderate drinkers had poorer smoking cessation outcomes when compared to heavy drinkers (Kahler et al., 2008). This was not the case in our study. Although our results were not in line with the study hypotheses, it is reassuring to observe that heavy tobacco use does not interfere with treatment outcomes for both depression and alcohol misuse. Our results did reveal, however, that reductions in tobacco use between baseline and three-month follow-up were significantly associated with reductions in depression and alcohol use over the same time period, suggesting that it is possible to make simultaneous changes on all three domains, at least in the short term. Future studies should seek to address this association, and identify mechanisms by which the short-term changes could be maintained for longer-term benefit in all three domains (depression, alcohol and smoking). Prolonged treatment may have been more successful in improving depression, alcohol and smoking, despite smoking not being specifically targeted in the treatment provided in the current study. Smoking may have been reduced further with additional targeted treatment and perhaps provision of nicotine replacement therapy.

Hypothesis (c): that significant reductions in smoking tobacco will be observed from pre- to post-treatment and that these reductions will be moderated by treatment condition.

It was anticipated that despite cigarette smoking not being directly targeted, the integrated approach to treatment taken in the current study might encourage generalization of skills to smoking behaviors, resulting in decreases in smoking.

However, there was no reduction in smoking associated with treatment for depression or alcohol problems in the present study, indicating that tobacco use needs to be specifically targeted in treatment programs.

Whilst the impact of depression and alcohol use treatment on smoking behavior has not been addressed in previous research, previous authors have reported that participants receiving treatment for smoking with skills that could be generalized to AOD use had poorer treatment outcomes compared to those who received the smoking cessation treatment alone (Burling, Burling, & Latini, 2001). It was postulated that too much emphasis was given during treatment regarding the similarities of tobacco use and other substance abuse, which may have created undue pressure for the participants, who may have found these messages overwhelming. However, this was not the trend in our study. Although not statistically significant, participants receiving an integrated CBT/MI treatment for depression and alcohol use, with arguably the greatest potential for generalization of skills, reported greater reductions in tobacco use between baseline and 12-month follow-up than did participants in other single-focused, non-specific or brief treatments. Those allocated to SHADE computer treatment reported the greatest reduction in cigarettes consumed per day of all the treatment groups. We have previously reported that the SHADE computer program was associated with significantly increased client initiative in therapy, with responsibility for learning and using treatment strategies based largely within the client (Kay-Lambkin, Baker, Lewin, & Carr, 2011). This may have also meant that the SHADE computer program had the largest potential for generalization of skills to other relevant areas of concern for the participant, including smoking tobacco. Other research has

demonstrated that a substance use-focused treatment was effective in reducing tobacco use among adolescents (Shelef et al., 2009a). The authors suggested that this might be due to the targeting of external cues in the substance use treatment, which were easily generalized by participants to their smoking behaviors. This may also explain the finding in the current study, that participants receiving alcohol-focused treatment made reductions of a similar magnitude to the integrated therapist-delivered CBT/MI in relation to cigarettes per day. Importantly, some exposure to CBT/MI strategies, regardless of focus, is important in the context of tobacco use among people with depression and alcohol use problems, with participants in the PCT condition (non-directive supportive counseling, with no CBT/MI strategies offered) reporting increases in tobacco use over the study period.

Limitations

A number of sample and methodological limitations are important to note. Given the sample was seeking treatment for depression and alcohol use problems, they may not represent the wider population with comorbid depression and alcohol use problems. The sample involved in this research was generally of Caucasian background and the findings may not be generalizable to other cultural groups. With regards to substance use, current usage may have been under-reported, and no biological verification was included to confirm smoking abstinence. We only measured smoking prospectively from baseline, and not retrospectively. Hence, we do not know how recently the non-smokers in our sample had stopped smoking, or for how long current smoking levels had been maintained by participants. Given this study was a

secondary data analysis of existing datasets that did not focus on tobacco use, key smoking outcome data was also not collected (e.g. quit attempts).

Future Directions

Notwithstanding the above limitations, the present research is amongst the first to investigate the possible relationship between levels of tobacco smoking and the severity of symptoms experienced in a comorbid adult population presenting for treatment for depression and alcohol problems.

These results have important implications for the assessment and treatment of clients within Mental Health services, Drug and Alcohol facilities and the general health care system. To effectively treat individuals with tobacco use and other comorbid disorders, we need to have a detailed understanding of the influence smoking has on the person's psychological, biological and social existence. By understanding these associations, offering comprehensive assessments around these issues, and broadening the scope of mental health and AOD treatments to include functional improvement, perhaps in the form of vocational and other life skills training, we may be able to make significant inroads into these pressing health concerns.

The issue of the generalisability of skills learned in therapy to secondary problems remains an interesting area of discussion, and is particularly relevant to the comorbidity literature. The idea of developing treatments that target multiple

conditions is also interesting to explore. The question of whether smoking is targeted within treatments for mental health or AOD use disorders raises questions about individuals becoming overwhelmed during the interventions and compromising the effectiveness of the initial treatment focus. Alternatively, acting opportunistically to target smoking has its own difficulties, including the current barriers to identifying tobacco smoking as important in the context of other (possibly more immediate) health concerns. Furthermore, targeting functional improvement in individuals experiencing comorbidities in mental health and AOD use such as vocational skills and life skills training also warrants further investigation in regards to effectiveness.

Clearly we need to focus more on the area of smoking and comorbidity, given the huge public health impact of smoking on the global community. Tobacco smoking is the single most preventable cause of disease and death worldwide. Current hindrances to maintaining strong declines in smoking rates are in part related to the reach, effectiveness and adoption of smoking cessation support. Increasing the proportion of smokers attempting to quit, together with increasing the proportion using effective strategies on those quit attempts, will add downward pressure to smoking rates.

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Table 1: Characteristics of the study sample

	N	%
Gender		
Males	234	53
Females	213	47
Marital Status		
Single, never married	130	29
Divorced/Separated/Widowed	168	38
Married/Defacto	149	33
Completed secondary school	178	40
Unemployed	174	39
Receiving Welfare	243	54
Primacy		
Independent Depression	184	41
Alcohol-induced Depression	107	24
Unable to determine	156	35
12-month Diagnosis		
Major Depressive Disorder	276	62
Alcohol Dependence	361	81
Treatment Allocation		
Brief Intervention	70	16
Depression-focused	71	16
Alcohol-focused	68	15
Integrated-focused	133	29
SHADE	56	12
Person-centered Therapy	54	12

Table 2: Levels of depression, alcohol consumption and other domains as a function of severity of tobacco consumption at baseline assessment. Note: Non-smokers=0 cigarettes per day (cpd), Light=1-9 cpd, Moderate=10-19 cpd and Heavy=20+ cpd.

Severity of Smoking	N	Mean	SD[1]	Sig.
BDI-II[2] depression				
Non-smokers	185	30.48	8.83	
Light	63	32.38	8.49	n.s.
Moderate	67	32.78	9.64	
Heavy	130	32.02	8.74	
OTI[3]-alcohol				
Non-smokers	185	9.93	7.81	
Light	63	9.13	7.08	n.s.
Moderate	68	11.06	9.69	
Heavy	131	11.33	7.74	
OTI[3]-Cannabis				
Non-smokers	184	0.29	2.37	
Light	62	3.14	9.78	Non<Light, p<0.01
Moderate	67	2.14	5.23	
Heavy	129	2.61	7.56	Non<Heavy, p<0.01
BSI[4] – somatization				
Non-smokers	154	1.00	0.82	
Light	57	1.28	0.84	
Moderate	59	1.42	0.82	Mod>Non, p<0.01
Heavy	108	1.49	0.75	Heavy>Non, p<0.01
BSI[4] - anxiety				
Non-smokers	154	1.48	1.01	
Light	57	1.70	0.93	
Moderate	59	1.92	1.09	Mod>Non, p<0.05
Heavy	108	1.90	0.89	Heavy>Non, p<0.01
BSI[4] – phobic anxiety				
Non-smokers	154	0.86	1.00	
Light	57	1.20	1.08	
Moderate	59	1.37	1.24	Mod>Non, p<0.01
Heavy	108	1.27	1.08	Heavy>Non, p<0.01
GAF[5] - functioning				
Non-smokers	167	58.78	9.00	
Light	59	57.42	10.00	
Moderate	64	54.03	10.79	Non>Mod, p<0.01
Heavy	121	53.311	11.56	Non>Heavy, p<0.01

[1] Standard Deviation
[2] Beck Depression Inventory II scores

[3] Opiate Treatment Index Scores representing average use occasions per day
[4] Brief Symptom Inventory Scores
[5] Global Assessment of Functioning scores