UTILIZING A DIATHESIS-ANXIETY MODEL TO UNDERSTAND COMORBID ANXIETY AND DEPRESSION IN A CLINICAL ADULT POPULATION.

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Preface

The data for this study was collected by researchers from the Christchurch Psychotherapy for Depression Study (CPDS) in which participants were randomised to receive either Interpersonal Psychotherapy (IPT) or Cognitive Behavioural Therapy (CBT) for depression. I was not involved in the implementation of the treatment within the CPDS, or the collection of data throughout the study. My contribution to this study was to prepare, analyse and interpret the data collected at baseline from this sample. These analyses were based upon the current and expanding literature surrounding the comorbidity of anxiety and depression. Thus, this formed the basis of my thesis.

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Abstract

It is well documented that anxiety and depression often co-occur, and that with comorbidity comes an increase in personal and societal costs. However, it is not entirely clear as to the mechanism of their comorbid relationship. The purpose of this study is to investigate whether a diathesis-anxiety model could be a possible explanation for frequently occurring comorbid anxiety and depression. The diathesis-anxiety model proposes that cognitive vulnerabilities interact with anxiety symptoms resulting in depressive symptoms. The cognitive variables of sociotropy, autonomy, rumination and dysfunctional attitudes were examined within a clinical adult population. On completion of regression modelling of the diathesis-anxiety model, as well as a reverse diathesis-anxiety model to test for an alternative temporal relationship of anxiety and depression, it was clear that no interaction effects were found for the selective cognitive variables. Whilst the current study did not support the diathesis anxiety model, past research has shown some significant results. The implications and future directions for research are discussed.

Introduction

Depression

Depression is described by the Diagnostic and Statistical Manual of Mental disorders, fifth edition (DSM-5; American Psychiatric Association, 2013) as the culmination of a depressed mood or loss of interest or pleasure plus four or more other symptoms. These include; weight change, sleeping difficulties (insomnia or hypersomnia), psychomotor agitation or retardation, fatigue, feelings of worthlessness or guilt, decreased concentration, and suicidal thoughts, ideation or plans. The DSM-5 (American Psychiatric Association, 2013) also includes a minimum time that symptoms must have been occurring for, that being two weeks. These symptoms are also known as Major Depressive Disorder (MDD).

Depression is one of the most commonly diagnosed mental disorders amongst adults (Richards, 2011). A nation-wide New Zealand household survey in 2003-2004 of 12,992 people aged 16 years and above found a lifetime prevalence of 16% for MDD (Oakley Browne, Wells, Scott, & McGee, 2006). The age of onset for depression is also wide-ranging, however a study of 3,896 outpatients found the mean age of onset for MDD to be 26 years of age (Zisook et al., 2007).

MDD is also known to have a very chronic course such that a 2010 review found the rate of recurrence after remission of an episode of MDD to be up to 85% in specialised mental health care and primary care populations (Hardeveld, Spijker, De Graaf, Nolen & Beekman, 2010). Furthermore, rates in the general population for relapse are approximately 35% (Hardeveld et al., 2010). Due to the chronicity and prevalence of depression, a large number of individuals are likely to experience significant disability from the disorder. Also, a number of subclinical residual symptoms often linger after the depressive episodes causing additional disability and promoting further relapse (Hardeveld et al., 2010). Disability due to depression includes negative impacts on psychosocial functioning such as work/employment

and spousal relationship impacts (Judd et al., 2000), exacerbation of pre-existing medical conditions (Gaynes, Burns, Tweed & Erickson, 2002), decreased health-related quality of life comparable to the chronic health conditions of arthritis, diabetes, and hypertension (Gayne et al., 2002), and higher risk of alcohol abuse or dependence, suicide attempts, educational underachievement and unemployment (Fergusson & Woodward, 2002). Whilst it is clear that depression has a large impact on the individual and their living circumstances, it is important to recognize that depression also has a large direct economic cost on society due to treatment and healthcare costs, as well as the substantial indirect costs of morbidity, absenteeism and decreased productivity (Richards, 2011).

Methodological Issues Regarding the Study of Depression

There is still a lot that is unknown in regards to the etiology of depression, such that depression is understood today as a multifactorial disorder with a combination of risk factors (de Jonge, Wardenaar & Wichers, 2015). The current limitations in understanding the etiology of depression may lay in the methodological issues of depression research. These issues are somewhat reflected in the current diagnostic assessment of depression. Depression as described by the DSM-5, is based on a description of symptoms, their presence and their quantitative change over time. Each of the depressive symptoms described is not qualitatively, but quantitatively different from experiences all individuals have at some point in their lives and therefore depression is often not detected or misdiagnosed (Wong & Licinio, 2001). Consequently, if this occurs in clinical practise it is also highly likely to be occurring in research resulting in incorrectly labelled samples.

Further methodological issues with researching depression include the difficulties in comparing studies due to differing definitions of depression. Depression can be viewed as a symptom, defined by a single isolated component of the syndrome that is regarded as part of

the normal range of human emotion or as a syndrome, such as MDD. It is important to distinguish in research whether the study is measuring depression as a syndrome or as a symptom in order to accurately compare findings and identify targets for treatment. For example, in the treatment of eating disorders the occurrence of depression symptoms is likely to improve with an eating disorder-focussed treatment, however the occurrence of a depression syndrome indicates the likely requirement of specific management of that syndrome (Steiger & Israel, 2010). In much the same way, depression as a symptom is a normal reaction to grief and does not require treatment, however if the symptoms persist past two months and other syndrome symptoms are also present (i.e. suicidal ideation, psychomotor retardation) then a major depressive episode is able to be diagnosed and treatment is recommended (Zisook & Shuchter, 2001). Therefore, the definition of depression is critical for understanding the importance of its manifestation.

Additional issues regarding defining depression arises in research due to the range of instruments used to measure the occurrence of depression. This is because different measures are based on different DSM criteria and whilst psychometric instruments may be able to detect depression, they are not able to diagnose a depressive disorder and therefore caution needs to be taken when comparing samples of "depressed" individuals. Furthermore, the description of a sample, whether it be community, student or clinical is critical when comparing research findings as the area of depression has been heavily reliant on student samples since the 1970s (Gotlib, 1984). This was due to the assumption that mild depression in university students in quantitatively analogous to clinical depression in psychiatric patients however this has since been challenged (Gotlib, 1984).

Whilst there are issues that need to be considered when studying depression regarding the definition, as highlighted above, it is also important to consider the impact that assessment methods have on ratings of depression. Of particular importance is the

comparison of self-reported and clinician-rated measures. When comparing measures of self-reported depression with clinician-rated depression it is generally found that the individual rates their symptoms as more severe than the clinician does (Domken, Scott & Kelly, 1994). It is likely that a number of demographic, clinical and personality variables play a role in the differences between self-reported and clinician-rated measures of depression including possible influences of age, subtypes of depressive presentations, severity of symptoms, education levels and personality variables such as neuroticism, extraversion and agreeableness (Enns, Larsen & Cox, 2000). Domken et al. (1994) found similar findings for neuroticism and severity, whilst Sayer et al.'s (1993) own research agrees with the findings regarding the influence of age and education.

Furthermore, a 2012 study by Uher et al. demonstrated that the self-report and clinician-rated measures of outcomes for depression are not equivalent, but rather are complimentary. Even more importantly, Carter, Framptom, Mulder, Luty and Joyce (2010) have shown that only a moderate correlation exists between the self-report Beck Depression Inventory (BDI-II) and the clinician-rated Montgomery-Asberg Depression Rating Scale (MADRS) when compared using an outpatient sample of individuals with primary depression. These results show that the two measures of depression are not measuring the same aspects of depression symptomology and therefore interpreting them in research and practice should be done so with caution.

Comparing and investigating differences between measures of self-report and clinician-rated depression is important clinically as differences in perceptions of the problem between the individual and the clinician may undermine treatment, collaboration and compliance (Domken et al., 1994). Furthermore, clinicians interpreting self-report measures from patients need to be aware of influencing variables (Carter et al., 2010). Therefore, the use of only one mode of measurement may result in the loss of clinically significant

information. In regards to research, the use of multimodal assessment methods for depression are important as researchers should be aware of individual variables that could influence ratings wether that be over- or under-estimation of symptoms (Enns et al., 2000).

Anxiety

According to the DSM-5 (American Psychiatric Association, 2013) anxiety disorders include those disorders that have features of excessive fear and anxiety and related behavioural disturbances. By this definition, anxiety is a disorder but it is also a symptom component of anxiety disorders. Therefore, like depression, anxiety has similar methodological issues to depression, as it can be either a syndrome (such as Generalised Anxiety Disorder, Social Phobia, etc.) or a symptom. In the symptom form the DSM-5 (American Psychiatric Association, 2013) broadly describes anxiety as the "anticipation of future threat", however this can present itself in the emotional, cognitive, behavioural and somatic domains of an individual.

Whilst mood disorders typically have their onset in early adolescence, anxiety states tend to have their onset in childhood with higher rates in girls than boys, however both panic and obsessive-compulsive disorders are considerably rare in children under 12 years of age (Merikangas, Nakamura, & Kessler, 2009). A recent systematic review and meta-regression on international data found the prevalence on anxiety disorders to be approximately 7.3% worldwide (Baxter, Scott, Vos, & Whiteford, 2013). Another recent survey of 8,841 Australian residents aged 16-85 years old as part of the Australian National Survey of Mental Health and Wellbeing found the 12-month prevalence of anxiety disorder to be 11.8% whilst lifetime prevalence was at 20.0% (McEvoy, Grove, & Slade, 2011). A comparable New Zealand study also found a similar lifetime prevalence of anxiety disorders of 24.9% (Oakley

Browne et al., 2006) indicating that a large number of the population is affected in some way by anxiety disorders in their lifetime.

Anxiety is a debilitating condition with all anxiety disorders having been shown to reduce quality of life and psychosocial functioning in sufferers (Mendlowicz & Stein, 2000). Furthermore, a longitudinal study of a birth cohort of 1,265 children born in Christchurch, New Zealand found significant results regarding the impact of anxiety on these children's lives up to the age of 21 years old (Woodward & Fergusson, 2001). Woodward and Fergusson (2001) found that as the number of anxiety disorders in adolescence rose, there was also an increase in the rates of later nicotine, alcohol and illicit drug dependence, as well as increases in suicidal behaviours, educational underachievement and early parenthood. However, Woodward and Fergusson (2001) concluded that anxiety was not directly responsible for these outcomes as when they controlled for a number of socio-familial and personal disadvantages faced by the individuals as young adults there was no longer a significant relationship between the number of anxiety disorders in adolescence and rates of later nicotine, alcohol and illicit drug dependence, suicidal behaviours, educational underachievement, and early parenthood.

Therefore, Woodward and Fergusson (2001) concluded that their study displayed evidence of negative life outcomes being a consequence of disadvantageous life factors associated with anxiety, rather than direct effects of early life anxiety. However, even after controlling for a number of possible confounding factors, including social, family and personal factors, there was still evidence found for significant relationships between the number of anxiety disorders reported in adolescence and early adulthood and later illicit drug dependence, further anxiety issues, major depression and failure to attend university (Woodward & Fergusson, 2001). As can be seen by this study, anxiety is a complex phenomenon with a range of direct and indirect outcomes.

Additionally, like depression, anxiety also has a huge economic burden that further highlights the need for good research in the area. In 1990 in the United States of America it was estimated that USD$46.6 billion was the total cost associated to anxiety disorders, which amounted to 31.5% of the total expenditure for mental illness (DuPont, Rice, Miller, Shiraki, Rowland & Harwood, 1996).

Comorbidity

Comorbidity can be defined as "having more than one physical or mental disorder at the same time" (Teeson, Slade & Mills, 2009). The impact of comorbidity on individuals is great, such that when a sample of 1,507 of older adolescents with either "pure" or comorbid forms of major psychiatric disorders were compared, the impact of comorbidity was the strongest indicator of increased negative outcome scores on measures of academic problems, increased mental health service utilization and greater numbers of past suicide attempts (Lewinsohn, Rohde & Seeley, 1995). In further support of the detrimental effects of comorbid mental disorders, a review of the findings of the 2007 Australian National Survey of Mental Health and Wellbeing (Teeson et al., 2009) found that comorbidity of mental health disorders was associated with greater impairment. More specifically, 54% of the individuals with comorbid mental disorders experienced severe levels of impairment as measured by each individuals experienced impact in the last 12 months as well as total days the individual was completely unable to perform usual activities for the last 30 days. In contrast, only 7.5% of individuals with only one mental health disorder experienced severe levels of impairment (Teeson et al., 2009). Teeson et al. (2009) suggest that prevention is crucial in comorbidity, however for this to be possible it is first imperative that we understand the pathways to comorbidity.

Comorbidity in Depression

Depression is most commonly found to be comorbid with anxiety disorders (Richards, 2011). The comorbidity of anxiety and depressive symptoms is so common, that 50-60% of people diagnosed with Major Depressive Disorder (MDD) are also diagnosed with a comorbid anxiety disorder in their lifetime (Kaufman & Charney, 2000). The comorbidity of anxiety and depression has also been shown to have detrimental effects on the individual concerned. For example, a longitudinal cohort study was completed in the south of Sweden and published in 2015 by Mattison, Bogren and Bradvik. This study identified 508 participants with mood disorders. Mattison et al. (2015) concluded that the comorbidity of mental disorders, including comorbidity with anxiety disorders, increased the risk of mortality. Another study, conducted by Young, Mufson and Davies (2006) investigated the treatment outcomes for 63 adolescents with depression. Those adolescents whom had comorbid anxiety had worse treatments outcomes and therefore were harder to treat than those individuals with uncomplicated depression (Young et al., 2006). Finally, it has also been found that comorbid depression and anxiety can lead to greater symptom severity (Ledley et al., 2005). Ledley et al. (2005) found that within their clinically socially anxious sample of 295 participants, those with higher depressive symptoms also showed greater anxiety symptoms.

It is also frequently reported that anxiety more often precedes depression (Brady & Kendall, 1992; Avenevoli, Stolar, Dierker, & Merikangas, 2001). The 2000 review by Kaufman and Charney noted that for cases of Generalised Anxiety Disorder (GAD) alone, 63% of cases reviewed noted the onset of GAD to precede MDD. Furthermore, as high as 78% of Post-traumatic Stress Disorder Cases had their onset prior to a MDD diagnosis (Kaufman & Charney, 2000).

It is not entirely clear as to why anxiety occurs primarily before depressive symptoms. There have been a number of models put forward in an attempt to describe the comorbidity of depression and anxiety. The following discussion will critically review some of the comorbidity models including the causal and correlated liabilities models, which have received a great deal of attention in the research (Mathew et al., 2011). The diathesis-anxiety model will also be introduced and compared to the other models. Furthermore, the utility of cognitive variables within diathesis-anxiety model to explain comorbid anxiety and depression will be reviewed in order to demonstrate the utility of the current study.

Causal Models

Causal models state that anxiety directly causes depressive symptoms (Starr, Hammen, Connolly & Brennan, 2014). This notion comes from the widely supported finding that anxiety precedes the onset of depression. In 2011 Mathew et al. completed a longitudinal study to test this model. Mathew et al. (2011) collected data from 941 randomly selected high school students from western Oregon, USA at four different times until they were 30 years of age. A measurement of lifetime anxiety, measured at time one, was examined as a predictor of depression at the final assessment at 30 years of age. Mathew et al. (2011) found that anxiety measured at adolescence significantly predicted the onset of depression in later life, even in those participants who had no prior history of depression. This remained true even when controlling for common risk factors of depression, such as gender and emotional reliance (Mathew et al., 2011). Mathew et al. (2011) describe emotional reliance as the "extent to which the individual desires excessive support and approval from others, is anxious about being alone or abandoned, and is interpersonally sensitive".

Despite the above findings, a number of common risk factors of depression also significantly contributed to the prediction of later life depression, implying that they may

play a role somehow in the comorbid relationship. Therefore, this finding suggests that a causal model is not complex enough to describe the whole relationship between comorbid anxiety and depression. It is important to note also that Mathew et al.'s (2011) study was based on a non-clinical population with a limited age range of up to 30 years old and low incidences of anxiety disorders within their sample meant low power to detect predictive effects of anxiety disorders. These limitations suggest that support found for a causal model of comorbidity in Mathew et al.'s (2011) study should be interpreted with caution in regards to its application in clinical samples and settings.

In further support of the idea that a causal model is too simple of an explanation is a longitudinal study conducted by Starr et al. (2014). They preferentially selected 705 infants from a birth cohort of 7,775 children on the basis of self-reported depression from their mother. The birth cohort was from Queensland, Australia and the children were followed from birth until age 23. As expected, Starr et al. (2014) found that anxiety greatly preceded depression in their sample. Starr et al. (2014) also concluded that interpersonal dysfunction may be one mechanism through which anxiety causes later depression as they hypothesized that anxiety may provoke interpersonally destructive behaviours, thereby creating strain on relationships, which may inturn trigger a depressive episode. The researchers came to this conclusion as their results showed that low sociability and interpersonal oversensitivity, which were described to be factors of interpersonal dysfunction, mediated the association between social anxiety measured at age 15 and later depression at ages 20 and 23 (Starr et al., 2014). This study has a number of limitations including being unable to explain the exact logistics of how interpersonal dysfunction plays a part in the comorbidity of anxiety and depression, the fact that interpersonal dysfunction was assessed by self-perceptions only and therefore could have been a cognitive symptom of depression, and lastly, depressed mothers were used to select participants creating a large confounding factor. Howeevr, one important

finding did come from this piece of research. The study by Starr et al. (2014) further showed that a causal model is too simplistic to explain comorbid anxiety and depression and that in fact a number of other variables are highly likely to be contributing to how anxiety leads to depression.

Correlated Liabilities Models

In Mathew et al.'s (2011) study, described above, the results alluded to the notion that anxiety and depression have common risk factors such as gender, social support, worry, loneliness, and emotional reliance which suggests that perhaps the comorbidity of anxiety and depression is more likely due to these shared risk factors. In much the same way, a correlated liabilities (shared etiology) model proposes that both depressive and anxiety symptoms have similar causes or etiological pathways (Rice, van den Bree & Thapar, 2004; Grant et al., 2014), such that the same set of cognitive vulnerabilities may interact with life events or stressors to cause both anxiety and depression.

The research on a common etiological mechanism of depression and anxiety, as well as unique mechanisms, has guided the way in which comorbid disorders are treated (Garber & Wersing, 2010). One such common etiological mechanism popularly researched has been negative affectivity (NA). This term comes from the tripartite model of anxiety and depression and can be described as the extent to which as in individual feels upset or unpleasantly engaged and includes a number of affective states such as angry, guilty, sad and worried (Anderson & Hope, 2008). Garber and Wersing (2010) describe NA as being high in both depression and anxiety. Furthermore, the tripartite model of depression and anxiety, as described by Clark and Watson (1991), states that when variance attributable to NA is removed, the correlation between anxiety and depression is reduced and therefore the remaining unique factors of both disorders are more discernable.

One longitudinal study looking at this idea of common etiological mechanisms of anxiety and depression as a way to explain the disorder's comorbidity was a 2004 study conducted by Rice et al. Their interest was in whether a shared genetic etiology causes the comorbidity of depression and anxiety, which fits with a correlated liabilities model. Within their study they also looked at whether a causal model, whereby anxiety as a phenotypic risk factor is described as causing depression, would be a better fit for their data. Rice et al. (2004) recruited their sample from a systematically ascertained, population-based register of all twin births in South Wales between 1980 and 1991. All families were invited to participate, and by time two of their study they had 338 pairs of twins in their sample with an average age of 12.64 years. It was required that twins participating is this study live in the same home and that participants be under 18 years of age at the time two follow-up (Rice et al., 2004). Information was gathered for the study via questionnaires sent to the families, which were completed by parents, as well as twins over the age of 11. The results of the Rice et al. (2004) study found that a causal model was a poor fit for their data. They also found that there was evidence of shared genetic effects between early anxiety and later depression, suggesting a common genetic etiology. However, Rice et al. (2004) do acknowledge that they found a significant individual genetic effect, specifically for later depression. This shows that anxiety and depression do not entirely manifest the same genetic risk, from which it can be concluded that whilst some of Rice et al. (2004) data do fit a correlated liabilities model, this model cannot account for the entire comorbid phenomena.

Further research on the correlated liabilities model of comorbid depression of anxiety and depression has also been conducted by Grant et al. (2014) and Hong and Cheung (2015). Grant et al. (2014) conducted a non-clinical study of 112 undergraduate students from a Midwestern university in the USA. Their sample was mainly female and was recruited via an online research recruitment system. Participants were asked to complete questionnaires three

times over a two-month period. The aim of the study conducted by Grant et al. (2014) was to discover if the comorbidity of social anxiety and depression could be explained by transdiagnostic mechanisms. These mechanisms were excessive reassurance seeking and brooding, which can be likened to rumination. Whilst Grant et al. (2014) found no evidence for excessive reassurance seeking as a mediator of social anxiety and later depression, there was evidence found that brooding mediated the relationship between social anxiety and later depression.

Although this study showed support for the correlated liabilities model of comorbid depression and anxiety through the transdiagnostic process of brooding, the study recruited a population that was non-clinical and so there is no indication as to whether Grant et al.'s (2014) findings would generalize to a clinical population. However, it can be concluded that rumination is an important variable to explore in relation to comorbid anxiety and depression.

More recently, Hong and Cheung (2015) also investigated the validity of a correlate liabilities model. Hong and Cheung (2015) conducted a meta-analysis of 73 articles from 1989-2013 in order to determine the extent to which six cognitive vulnerabilities associated with depression and anxiety were related to one another. In this way, Hong and Cheung (2015) were trying to determine if anxiety and depression had shared etiological pathways or risk factors. The cognitive vulnerabilities examined included pessimistic inferential style, dysfunctional attitudes, rumination, anxiety sensitivity, intolerance of uncertainty, and fear of negative evaluation. Results showed mean correlations between approximately .35 and .60, indicating a moderate to strong correlation between vulnerabilities (Hong & Cheung, 2015). Meta-analytic structural equation modeling was then applied to the underlying relationships among the vulnerabilities. It was found that a one-factor model provided the best fit to the meta-analytic data, which suggests that the cognitive vulnerabilities are not specific to anxiety or depression, but rather are common risk factors for both (Hong & Cheung, 2015).

Although this study also shows support for a correlated liabilities model of comorbid depression and anxiety, this study was conducted on largely non-clinical student samples. These types of samples are likely to create inflated mean correlation coefficients as non-clinical samples are unlikely to have restricted ranges on measures, unlike clinical samples who are more likely to endorse the higher ends of measures (Hong & Cheung, 2015). Also, due to the cross-sectional and correlational nature of the studies reviewed there is the possibility that the covariation amongst cognitive vulnerabilities could be accounted for by the comorbidity of anxiety and depression symptoms (Hong & Cheung, 2015).

Overall, the studies examined above show some support for a correlated liabilities models of comorbid depression and anxiety, however these studies were not without their flaws and were not conclusive as to the validity of the correlated liabilities model (Hong & Cheung, 2015; Grant et al., 2014; Rice et al., 2004). Furthermore, little support was shown for the causal model (Star et al., 2014; Mathew et al., 2011) and in one instance the causal model of depression and anxiety was dismissed (Rice et al., 2004). It is therefore imperative that we look beyond these two models to search for other mechanisms that are likely to explain the comorbidity of anxiety and depressive symptoms more clearly.

Diathesis-Anxiety Model

One such study that attempts to look beyond the causal and correlated liabilities models is a 2014 study by Cohen, Young, Gibb, Hankin and Abela. As some support has been shown for a correlated liabilities model and weak support for the causal model in past research, Cohen et al. (2014) attempt to clarify the research by creating a third model by combining both causal and correlated liabilities models. This combination forms a diathesis-anxiety model of comorbid depression and anxiety (see Fig. 1 for representations of all three models). Cohen et al. (2014) worked from within a cognitive-vulnerability framework and

therefore, the diathesis-anxiety model is described by Cohen et al. (2014) as the process of cognitive vulnerabilities interacting with anxiety symptoms resulting in depressive symptoms. The cognitive vulnerabilities examined by Cohen et al. (2014) included rumination and self-criticism.

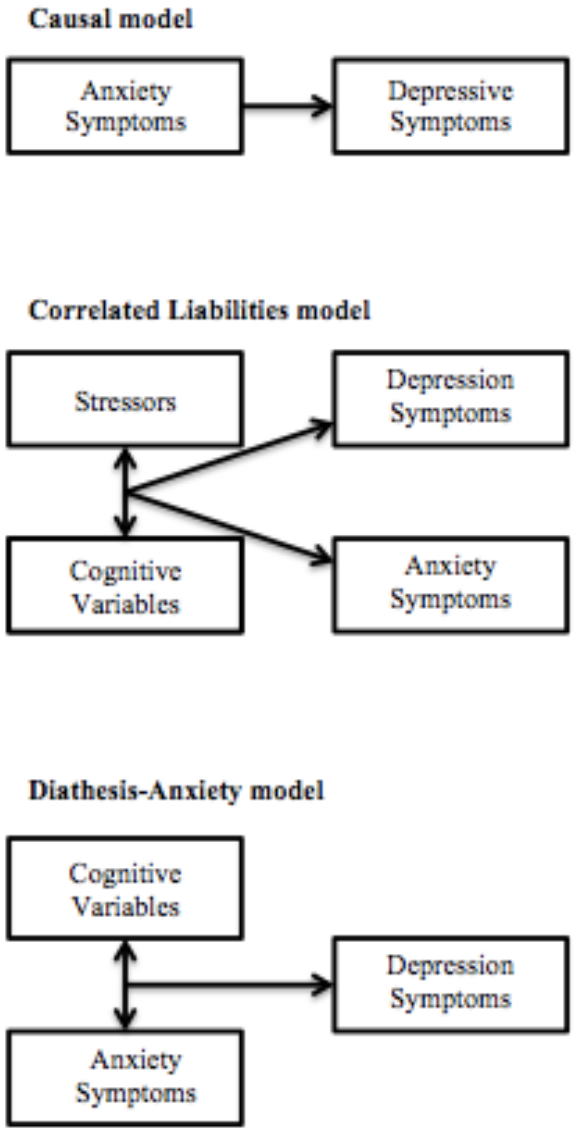

Figure 1. Causal, Correlated Liabilities and the Diathesis-Anxiety Model Adapted from Cohen et al. (2014).

It is important to note that Cohen et al. (2014) are not the first group of researchers to propose a diathesis-anxiety-type model of comorbid anxiety and depression. Three other

studies have tested a similar model to the one displayed above in Figure 1. In 2008 Feng, Shaw and Silk published a longitudinal study on 290 boys in the Pittsburgh metropolitan area. Their sample was aged 2-10 years of age and consisted of working class, low-income families. Data for their study was obtained through maternal ratings, laboratory observations and clinical interviews with both the mother and child (Feng et al., 2008). This study focused mainly on the trajectories of anxiety development from early childhood, some preliminary findings fitting to a diathesis-anxiety model. Feng et al. (2008) suggested that maternal control might interact with anxiety symptoms in early childhood to predict the development of depressive symptoms in later childhood.

Furthermore, Starr and Davila (2012), and Hankin (2008) also found support within their respective research of cognitive variables interacting with anxiety to predict depression symptoms. Starr and Davila (2012) proposed that the cognitive style in which one responds to anxiety might increase the risk they have of developing depression. Starr and Davila's (2012) study, labeled as study 3 within their research, contained a sample of 55 participants with generalized anxiety disorder (GAD) who were part of a larger study and were recruited through a variety of sources including advertisements on campus and online, through graduate training clinics in the Department of Psychology at Stony Brook University, from other research studies, and through undergraduate psychology courses. Their sample was 89% women and the average age of participants was 28.67 years. Star and Davila (2012) obtained their data through brief structured interviewing and self-report questionnaire, which were recorded at baseline and 4 weeks after baseline. Statistical modeling for interaction effects between cognitive styles and anxiety to predict depression followed.

The results of Star and Davila's (2012) study showed that anxious arousal, which was used to measure a unique aspect of anxiety that is not seen in depression due to the large overlap of symptoms often seen in depression and anxiety, interacted with the measure of

ruminative and hopelessness responses to anxiety to predict changes in depression measures (Star & Davila, 2012), supporting a diathesis-anxiety model containing cognitive variables interacting with anxiety to predict later depression. Whilst this study stands out from aforementioned studies as it is conducted with a clinical sample, the sample is small and needs to be studied further.

Hankin (2008) also looked at a diathesis-anxiety-type model in their multi-wave prospective study and further supported the results of Star and Davila (2012). Hankin (2008) studied a sample of 345 youth who were recruited from five Chicago area schools. The schools selected were chosen as to reflect the ethnic and socioeconomic diversity of the Chicago area. Their sample was 57% female and aged between 11 and 17 years. Data was collected via questionnaires at four time points over a five-month period (Hankin, 2008). Whilst Hankin (2008) had aimed to investigate the power of ruminative response styles to predict depression symptoms compared with anxiety and externalizing symptoms, they found evidence for baseline rumination interacting with prospective co-occurring fluctuations of anxious arousal over time. This interaction was shown to predict the highest levels of prospective depressive symptoms (Hankin, 2008). It is important to note however that this study was conducted on a non-clinical youth sample.

Cohen et al. (2014) tested the diathesis-anxiety model in more direct way than Star and Davila (2012) and Hankin (2008) by comparing it to the causal and correlated liabilities models in a multi-wave, longitudinal study. Cohen et al.'s (2014) participants were 208 third-, 245 sixth-, and 225 ninth-graders from a range of public schools; therefore this was not a clinical sample. Participants were recruited by letters sent home to parents of children who attended schools in participating districts of the USA. These districts included; Denver, Colorado, New Jersey and New Brunswick (Cohen et al., 2014). The participants completed evaluations every three months for 18 months. The measurements taken were self-report

questionnaires and assessed cognitive vulnerabilities (rumination and self-criticism), stressors, and depressive and anxiety symptoms. Cohen et al. (2014) tested all three models.

Cohen et al. (2014) discovered that anxiety symptoms at time one did not significantly predict depressive symptoms, neither did depressive symptoms at time one significantly predict anxiety symptoms at time two, therefore their data did not conclusively support a causal model of comorbidity. Cohen et al. (2014) also tested if cognitive vulnerabilities interacted with stressors to predict later depression and anxiety symptoms (correlated liabilities model). No significant support for this model was found either. Lastly, Cohen et al. (2014) tested whether self-criticism and rumination interacted with anxiety symptoms to predict later depressive symptoms. The results showed that both self-criticism and rumination significantly interacted with anxiety to predict depressive symptoms. Cohen et al. (2014) also found that those youth with higher levels of rumination or self-criticism who experienced high levels of anxiety symptoms were most at risk for developing higher levels of depressive symptoms in the future.

In order to provide a more stringent test, Cohen et al. (2014) also tested a reversed diathesis-depression model. It was found that depression did not significantly interact with cognitive vulnerabilities to predict later anxiety. Of these previous studies of the diathesis-anxiety model, only Star and Davila (2012) used a community adult sample. Therefore the applicability of the diathesis-anxiety model to adults is somewhat unclear. Even more relevant, is that no prior studies have examined the diathesis-anxiety model in a clinically comorbid sample.

Contribution of Cognitive Variables

The contribution of cognitive variables, such as ruminative response styles (Star & Davila, 2012; Hankin, 2008) and self-criticism (Cohen et al., 2014), to the comorbidity of

anxiety and depression has often been supported in psychological theories. The cognitive content-specificity hypothesis, which rose to popularity in the 1980's and has had a great impact on the way in which mental disorders are conceptualized and treated, states that depression and anxiety have their own cognitive vulnerabilities that are correlated such that, an increase in one risk factor for one condition is associated with an increase in risk factors for another condition (Beck & Perkins, 2001). This theory also posits cognitive content and profiles as highly important and defining of emotional disorders (Baranoff & Oei, 2014). However an in-depth meta-analysis by Beck & Perkins (2001) has found inconsistent support for this hypothesis and suggested that perhaps some cognitive variables are significant to depression, but perhaps also there are cognitive variables shared by anxiety and depression.

This idea that cognitive content is not specific to the individual disorders of anxiety and depression, but rather there is overlap has had mixed evidence in more recent years (Baranoff & Oei, 2014). More specifically, whilst there appears to be links between specific cognitive content and emotional responses, there is less momentum for the idea that cognitive-emotional relationships are unique, as a unique relationship would suggest limited or no cross-over of anxious and depressive cognitive content (Baranoff & Oei, 2014). Furthermore, recent research regarding the application of the cognitive content-specificity hypothesis to comorbid anxiety and depression has found unique and overlapping cognitive content (Brown et al., 2014) as well as suggestions that whilst the cognitive content of anxiety and depression have commonalities, they appear to be distinct profiles (Hendriks et al., 2014). Therefore, it can be suggested that cognitive vulnerabilities may be useful to examine in the context of comorbid anxiety and depression, and that not enough is known about their relationship to comorbid mental illnesses.

The current study aims to examine the diathesis-anxiety model proposed by Cohen et al. (2014) by testing the model in an adult clinical population. This sample has primary

diagnosis of depression with comorbid anxiety. The cognitive variables of rumination, dysfunctional attitudes, sociotropy and autonomy are to be used. These cognitive variables constitute characteristic patterns of encoding, interpreting, and recalling of information (Hong & Cheung, 2015) and have each received ranging support for their role in the comorbidity of depression and anxiety.

Rumination. Rumination has commonly been linked to both anxiety and depression (McLaughlin & Nolen-Hoeksema, 2011) and it has repetitively been shown that those who engage in rumination are more likely to have higher levels of depressive symptoms in the future (Nolen-Hoeksma, 2000). Not only this, but rumination is described as being a robust predictor of anxiety as well as depression (Michl, McLaughlin, Shepherd & Nolen-Hoeksema, 2013).

Rumination can be described as repetitive focus on one's own negative feelings and the implications (Lyubomirsky, Tucker, Caldwell, & Berg, 1999). Rumination often prevents problem solving (Nolen-Hoeksema, 1991; Lyumbomirsky et al., 1999), as the person is preoccupied cognitively with the problem, which can lead to feelings of hopelessness, a core feature of depressive disorders. Rumination has also been shown to positively correlate with anxiety and depression in students and patients (Segerstorm, Tsao, Alden, & Craske, 2000).

Sociotropy and Autonomy. Sociotropy and autonomy were originally linked to Beck's (1983, as cited in Clark et al., 1995) diathesis-stress model that proposes, in the context of depression, that life stressors interact with personal variables to produce depression. Someone who is highly sociotropic can be described as placing excessive value on interpersonal relationships and believes they must be loved and accepted by others (Clark et al., 1995). On the other hand, someone who is a highly autonomous individual greatly values his or her independence, mobility and freedom of choice (Clark et al., 1995).

Beck views the variables of sociotropy and autonomy as cognitive modes that can

dominate psychological functioning at any one time or another (Clark & Beck, 1991). Beck went on to theorize that highly sociotropic individuals would be likely to develop depression if life stressors were perceived as causing loss of social resources, whilst highly autonomous individuals would be likely to develop depression if life stressors were perceived as a threat to independence or goal-directed behaviours (Clark & Beck, 1991). It could be theorized then that anxiety could be seen as a stressor that causes loss of social resources and is a threat to independent behaviour. In more recent research, it has been shown that the constructs of sociotropy and autonomy also correlate highly with anxiety symptoms and are not specific to depression (Alford & Gerrity, 1995; Fresco, Sampson, Craighead, & Koons, 2001).

Dysfunctional Attitudes. Lastly, Beck (1978, as cited in Lee & Hankin, 2009) theorized that dysfunctional attitudes are the cognitive products of schemas that filter and negatively bias incoming information. Furthermore, these dysfunctional attitudes often have themes of inadequacy and failure, which lead to self-defeating thinking and therefore feelings of depression. Whilst the dysfunctional attitudes scale (DAS) was originally proposed to measure depressive symptoms (Weissman & Beck, 1978) it has been shown to not be a specific measure of depression (Hill, Oei, & Hill, 1989). This lack of specificity therefore suggests that dysfunctional attitudes may be a risk factor of later depression depending on other factors, such as anxiety symptoms. In further support of this, Kuiper, Olinger & Martin (1988) suggested that dysfunctional attitudes create irrational contingencies, which may then be viewed as a threat to positive self-evaluation and create anxiety. Dysfunctional attitudes in the form of Beck's cognitive triad of negative beliefs about the self, world and future (Weissman & Beck, 1978) are already heavily tied to the thinking of depressed individuals, and have also been shown to predict anxiety as they lower self-esteem (Lee & Hankin, 2009).

Aim of Current Study

The research discussed above demonstrates that whilst the comorbidity of depression and anxiety is a common phenomenon that has a large and varying effect on many individuals, there is not yet sufficient research to fully understand its mechanisms. This is somewhat impeded by methodological issues surrounding the definition and measurement of depression and anxiety, as well as the limitations of current models used to understand comorbidity. Furthermore, limitations are also seen in the range of populations used to study comorbid depression and anxiety, and whilst the diathesis-anxiety model shows promising preliminary results it too is hindered by it's currently limited application to adult clinical samples.

Therefore, the present study aims to investigate the utility of the anxiety-diathesis model to comorbid depression and anxiety in an adult clinical sample. In order to restrict limitations on the measurement of depression seen in previous research, the current study will measure both clinician-rated and self-reported depression.

Furthermore, a reversed diathesis-anxiety model will also be tested for each cognitive variable to discover whether the interaction of depressive symptoms with the cognitive variables to predict anxiety provides a better explanation of comorbid anxiety and depression. This reverse model was also investigated by Cohen et al. (2014), as discussed above.

Hypotheses for the Current Study

In the current study it is hypothesized that, based on current literature, the cognitive variables; rumination, sociotropy, autonomy, and dysfunctional attitudes will significantly interact with anxiety to predict later depression. There is also evidence above that a causal or correlated liabilities model is not sufficient to explain the comorbidity of anxiety and depression. Furthermore, the diathesis-anxiety model has been shown to be a possible valid

model by Cohen et al. (2014) in an adolescent non-clinical sample, and so these results are expected to be replicated in the current clinical adult sample. It is also expected that when the reverse diathesis-anxiety model is tested with the selected cognitive variables that the interaction of depression symptoms with the cognitive variables to predict anxiety will not provide a better explanation of comorbid anxiety and depression as research has consistently shown that anxiety precedes depression. Therefore, it is hypothesized that the diathesis-anxiety model will be a valid model of comorbid anxiety and depression.

Method

Participants

Participants were 177 adult outpatients (males *n* = 49; females *n* = 128) who had a principal major depressive episode diagnosis (DSM-IV). These participants were selected to be a part of the Christchurch Psychotherapy for Depression Study (CPDS), in which they were randomised to receive either Interpersonal Psychotherapy (IPT) or Cognitive Behavioural Therapy (CBT) for depression. In terms of severity of initial depression ratings, the participants had an average Montgomery-Asberg Depression Rating Scale (MADRS) score of 23.84 (± 6.33). Informed consent was obtained from all participants and approval for the study was obtained from the National New Zealand Ethics Committee.

Participants were referred to the study from a number of sources including general health practitioners, mental health services, family, and self-referrals. Participants were included in the study if they were over 18 years old, had a principal diagnosis of major depressive disorder (DSM-IV), and were medication free for at least two weeks (or 5 drug half lives for any centrally acting drugs) prior to entering the trial, except for the oral contraceptive pill and occasional hypnotic. Participants were excluded from the trial if they had a history of bipolar I disorder, schizophrenia, a major physical illness that was likely to interfere with assessment or treatment, current moderate to severe substance dependence as a principal diagnosis, severe anti-personality disorder, or had received as adequate trial of either CBT or IPT within the past year.

After screening for eligibility, participants underwent an initial psychiatric assessment, were provided with written information about the CPDS and provided written consent. Participants were then assessed using the clinician rated Structured Clinical Interview of the DSM-III-R-Patient version (SCID-P) and MADRS ratings, as well as a self-report booklet assessing demographic information and measures of psychological

functioning, cognitive style and personality. Participants were then randomized to a treatment. The current study used only baseline measurements from participants of the CPDS study. The details of variables measured are as follows.

Measurement of variables

Self-report depression. Self-report depression was assessed using the 21-item self-report Beck Depression Inventory – Second Edition (BDI-II; Beck, Steer, & Brown, 1996), which was administered at baseline to all participants. Each item in the BDI-II has four response items ranging from not present (0) to severe (3), which participant's rate based on their experiences in the previous two weeks. Scores are summed, with higher scores indicating a higher level of depressive symptoms (Beck et al., 1996). A comprehensive review by Wang and Gorenstein (2013) found the BDI-II to have good internal consistency with the average Cronbach's alpha of studies reviewed being .90. Retest reliability was also shown to be strong (Pearson's r = .73 – .96). Wang and Gorenstein (2013) also concluded that the BDI-II has high convergent validity with other depression measure, with Pearson's product-moment correlation coefficients (r) ranging from .82 to .94.

Clinician-rated depression. Clinician-rated depression was assessed with the Montgomery and Asberg Depression Rating Scale (Montgomery & Asberg, 1979;MADRS). The MADRS is a 10-item scale in which items are scored by a clinician based on more broadly phrased questions in a clinical interview. The 10 items include; apparent sadness, reported sadness, inner tension, reduced or increased sleep, reduces or increased appetite, concentration difficulties, lassitude, inability to feel, pessimistic thought and suicidal thoughts. The clinician must decide if the participant lays on the defined item steps (0,2,4,6) or in-between them (1,3,5). Scores are accumulative, ranging from 0 to 60, with higher scores indicating a higher level of depression (Montgomery & Asberg, 1979). An independent

clinician blind to treatment type conducted the MADRS. The MADRS was originally created to be sensitive to treatment-induced changes in depression symptoms and was created based on a sample of English and Swedish patients (Montgomery & Asberg, 1979). Montgomery and Asberg (1979) claimed to have inter-rater reliability coeffecients (r) ranging from .89 to .97. Davidson, Turnbull, Strickland, Miller and Graves (1986) found lower, yet still significant inter-rater reliability (r = .57 – .76). Davidson et al. (1986) also reported the MADRS to have good construct validity as 70% of the patients endorsed all symptoms represented by the MADRS. A further study examining the psychometric properties of the MADRS found internal consistency (Cronbach alpha) values ranging from .90 to .92 (Carmody et al., 2006).

Anxiety. Anxiety was measured using Anxiety subscale of the Hopkins Symptom Checklist (SCL-90). The SCL-90 is a 90 item self-report measure of psychopathology, which contains nine subscales. The Anxiety subscale consists of 10 items; participants rate each item from not at all (0) through to extremely (4). Items within each subscale are summed and dividing by the number of items to compute final scores (Derogatis, Lipman, & Covi, 1973). The SCL-90 was also developed primarily for use with outpatients (Derogatis et al., 1973). One study of 1002 psychiatric outpatients found that the SCL-90 subscale of Anxiety shows very good agreements between the hypothetical and empirical definitions of anxiety (Derogatis, Leonard, & Cleary, 1977). Another study identified good construct validity between the Anxiety subscale of the SCL-90 and anxiety scales of the Minnesota Multiphasic Personality Inventory (MMPI) with correlations (r) ranging from .54 to .69 (Dining & Evans, 1977). Derogatis, Rickels and Rock (1976) support these results as they also found that the dimensions of the SCL-90 correlated less strongly with non-analogous MMPI scales compared to analogous scales, which was interpreted as evidence of discriminant validity for the SCL-90. Hafkenscheid (1993) lends further support to the psychometric properties of the

SCL-90 by demonstrating good internal consistency for the Anxiety subscale (Cronbach's alpha = .88) and overall SCL-90 global scale (Cronbach's alpha = .97).

Cognitive Variables. ***Rumination*** was measured using the 22-item self-report Responses to Depression Rumination scale. This subscale measures the degree to which participants respond to their depressed moods in a ruminating manner (Nolen-Hoeksema, 1991). Participants were required to rate each item from almost never (1) to almost always (4). A psychometric evaluation of this scale on a large community sample uncovered high levels of internal consistency (Cronbach's alpha = .90) as well as high test-retest reliability (r = .67; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). This is supported by Kasch, Klein and Lara (2001) who also found a high Cronbach's alpha of .89 for the scale. This study also supported the construct validity of the Responses to Depression Rumination scale as the measure showed no significant relationship with problem-solving coping, positive temperament and disinhibition (r = .06, -.18, .17).

Dysfunctional attitudes were measured using the 40-item self-report Dysfunctional Attitudes Scale (DAS). The DAS was developed to measure pervasive negative attitudes of depressed individuals towards the self, world and future (Weissman & Beck, 1978). The DAS asks participants to rate items on a scale from 1 to 7, giving a single total score. Weissman and Beck (1978) provide a range of evidence for the psychometric properties of the DAS. Firstly, they found the DAS to be an internally consistent measure (Cronbach's alpha = .93) as well as a measure with good test-retest reliability (r = .71; Weissman & Beck, 1978). Similarly, Oliver and Baumgart (1985) support the test-retest reliability of the DAS (r = .73).

Sociotropy was measured using the 30-item Sociotropy scale of the Beck Sociotropy-Autonomy Scale (BSAS). Participants indicate the percentage of time each of the statements applies to them on a five-point scale from 0 (0%) to 4 (100%; Clark & Beck, 1991). Clark

and Beck (1991) reported high levels of internal consistency (Cronbach alpha = .87), which was replicated in a study by Sato and McCann (2000; Cronbach alpha = .90). Sato and McCann's (2000) study also supports the construct validity of the Sociotropy subscale as this measure was found to highly correlate (r = .76) with Personal Style Inventory (PSI) subscale of sociotropy.

Autonomy was also measured using the BSAS, however the 30-point Autonomy scale was used for this measurement. Scoring within this measure is the same as the Sociotropy scale above. However, this measure has three facets to it, those being; solitude, independence and individualistic achievement (Clark & Beck, 1991). The present study will utilize the total autonomy score. The facets of the Autonomy subscale have been shown to have good internal consistency in several studies (Beck & Clark, 1991; Sato & McCann, 2000) with Cronbach alphas ranging from .71 to .78.

Statistical Analyses

The Statistics Package for Social Science (SPSS, V.22, 2013) was used for the statistical analyses. Data was examined for normality, which showed Normal Q-Q plots for all variables that were approximately straight, the Kolmogorov-Smirnov test of normality showed the variables in these analyses to be of normal distribution except for the BDI-II and the Rumination measure. It should be noted however that this is common in large samples and is not expected to have affected the data. The data was also examined for missing values. The BDI-II measurement contained all cases, however measurements from the Anxiety subscale of the SCL-90, Rumination, DAS, and both Sociotropy and Autonomy subscale of the BSAS contained missing variables. Therefore, regression modelling that followed used listwise deletion. Although pairwise deletion allows the greatest use of the data, the sample is large enough to use listwise deletion. Secondly, pairwise deletion estimates the missing case

values and therefore correlations, means and standard deviations for each regression would be modelling on a slightly different data set (Howell, 2007).

Firstly, descriptive, demographic, and clinical variables of the sample were computed. Categorical variables were coded for as appropriate. A new variable was also created to identify alcohol and drug abuse in the past month. The variables for past month abuse of alcohol, sedatives/hypnotics/anxiolytics, cannabis, stimulants, opioids, cocaine, hallucinogens, and other drugs were combined such that presence on one or more of these variables would be coded for as the presence of alcohol or drug abuse in the past month

All cognitive variables were then transformed to create interaction variables with anxiety before multiple regression models were performed. Multiple regression analyses were performed to ascertain the independent contribution, or main effect, of anxiety and each cognitive variable (rumination, dysfunctional attitudes, sociotropy, autonomy) to depressive symptoms, as well as the interactive contribution, or interaction effect, of each cognitive variable with anxiety to predict depression symptoms. Regression models were run with self-report depression as the dependent variable and then re-run with clinician-rated depression as the dependent variable. Assumptions of multicollinearity, normality, homoscedasticity and independence of residuals were all met. In all regression models the Mahalanobis distance values exceeded that recommended by Pearson & Hartley (as cited in Pallant, 2010), indicating outliers. However, the Cook's distance value for each model was below 1 and therefore the outlying cases can be concluded as not having an undue influence on the model as a whole (Pallant, 2010). A p-value of <0.05 was taken to indicate statistical significance for all models.

The variables producing significant main effects for self-report depression were placed in a hierarchical regression model beginning with anxiety followed by, in order, the variables with the greatest to the least greatest variance accounted for. The variables

producing significant main effects for the regression models of clinician-rated depression followed the same procedure.

A reversed diathesis-anxiety model was also tested for each cognitive variable to determine whether the interaction of depressive symptoms with the cognitive variables to predict anxiety provided a better explanation of comorbid anxiety and depression. In order to do this, all cognitive variables were transformed to create interaction variables with self-reported depression and clinician-rated depression. Multiple regression analyses were performed to ascertain the independent contribution, or main effect, of self-report depression and each cognitive variable (rumination, dysfunctional attitudes, sociotropy, autonomy) to anxiety symptoms, as well as the interactive contribution, or interaction effect, of each cognitive variable with self-report depression to predict anxiety symptoms. The models were then re-run with clinician-rated depression, rather than self-report.

Assumptions of multicollinearity, normality, homoscedasticity and independence of residuals were all met. In all regression models the Mahalanobis distance values exceeded that recommended by Pearson & Hartley (as cited in Pallant, 2010), indicating outliers. However, the Cook's distance value for all but one model was below 1 and therefore the outlying cases can be concluded as not having an undue influence on the model as a whole (Pallant, 2010). The model in which clinician-rated depression was the independent variable, and sociotropy, anxiety and the interaction of sociotropy and anxiety were the dependent variables had one significant outlier in which the Cook's distance value was above 1. This variable was not excluded from regression modelling as on closer investigation this case was found to have the highest MADRS and lowest Sociotropy, therefore it was not considered to be significantly impacting on the model. A p-value of <0.05 was taken to indicate statistical significance for all models.

The regression models of anxiety in which self-report depression was used as the depression measurement produced a significant main effect of self-report depression. Therefore this variable was the only placed in a final regression model. The regression models of anxiety in which clinician-rated depression was used as the depression measurement also produced a significant main effect of depression for predicting anxiety, as well as interaction effect of clinician-rated depression and sociotropy. Therefore, these variables were placed in a final hierarchical regression model in the order of greatest to the least variance accounted for.

Results

Descriptive statistics

Table 1 shows the descriptive characteristics of the sample, which were predominantly female (72.30%) and had a mean age of 35.19 years (± 10.22). The majority of participants were European/Pakeha (88.70%) and referrals to the study were wide ranging. Comorbidity characteristics collected from participants showed 35.6% with an anxiety disorder in the last month. Fewer participants reported having a comorbid alcohol and drug disorder (9.60%), bipolar disorder (excluding bipolar I disorder; 3.4%), or eating disorder (1.20%) in the past month. The average global assessment of functioning rating was 56.46 (± 5.59), indicating a moderate to serious impairment in social and occupational functioning due to psychiatric symptoms (Spitzer, First, Gibbon, & Williams, 1989).

Table 1

Demographic Characteristics for Participants, N = 177.

Demographic	*N*	% or *M* (*SD*±)
Age	177	35.19 (± 10.22)
Gender		
Male	49	27.70
Female	128	72.30
Marital status		
Married or living together (1+yrs)	78	44.10
Separated	12	6.80
Divorces	14	7.90
Widowed	1	0.60

Never married	72	40.70
Ethnicity		
European/Pakeha	157	88.70
Maori	8	4.50
Pacific Islander	1	0.60
Asian	8	4.50
Referral source		
Self	64	36.20
Friends/family	16	9.00
General practitioner	37	20.90
Mental health service	60	33.90

Table 2 displays the descriptive statistics for the variables to be examined in the proposed diathesis-anxiety model. Participants had an average MADRS score of 23.84 (SD = 6.33) indicating the participants were, on average, moderately depressed. This rating is according to cut-off scores for graded depression ratings described by Snaith et al. (1986). Cut-offs described by Müller et al. (2000) support this classification of moderate depression also. The average score on the self-report BDI-II was 28.18 (SD = 9.86), indicating participants were, on average, moderately depressed based on the original cut-off scores ruled by Beck et al. (1996) from 500 outpatients. A follow-up meta-analytic study of 118 articles published between 1996 and 2012 by Wang and Gorenstein (2000) also concluded that, within their sample, those participants who had mood disorders scored an average BDI-II score of 26.60. This evidence indicates that the current sample can be considered depressed. The mean anxiety score, as rated by the SCL-90, was 1.29 (SD = .07) for our sample. Based on the norms for psychiatric outpatients, a score of above .73 is considered to indicate anxiety

(Derogatis, 1977). The mean scores for the cognitive variables were as follows: The average score on for Rumination was 55.44 (SD = 9.88). Whilst the Responses to Depression subscale of Rumination used does not provide specific cut offs to measure the level of rumination, the scale has been shown to consistently show higher levels of rumination in those diagnosed with depressive or anxiety disorders than those without (Nolen-Hoeksema, 2000). Previous research which has utilised the Responses to Depression subscale of Rumination show depressed samples scores to range between 33.50 to 59.43 (Watkins & Brown, 2002; Joormann, Dkane, & Gotlib, 2006; Watkins et al., 2007). Our sample provided a Dysfunctional Attitudes Scale (DAS) score of 150.33 (SD = 37.10). Scores for this measure can range for 40 – 280, and in a 2003 review of studies using the DAS as a cognitive measure related to depression the average DAS score based on 35 normative samples was 119.01 (SD = 26.89; Dozois, Covin, & Brinker, 2003). This normative data is somewhat lower than our current sample's DAS scoring and so our sample can be considered to have higher levels of dysfunctional attitudes than is considered normal. The current study's participants also displayed Sociotropy and Autonomy scores somewhat consistent with other clinical adult samples. In the current study participants had a mean Sociotropy score of 69.18 (SD = 10.22), and a score of 65.65 (SD = 14.00) for the Autonomy scale. This is consistent with a large 1996 study of 119 unipolar depressed patients prior to undergoing treatment who scored average scored of 77.9 (SD = 17.10) and 69.30 (SD = 16.00) on the Autonomy and Sociotropy scales respectively (Moore & Blackburn, 1996).

Table 2

Descriptive Statistics for Diathesis-Anxiety Model Variables.

Variable	*N*	*M* (± *SD*)
MADRS	177	23.84 (± 6.33)

BDI-II	177	28.18 (± 9.86)
Anxiety	176	1.29 (± 0.70)
Rumination	168	55.44 (± 9.88)
Dysfunctional attitudes	174	150.33 (± 37.10)
Sociotropy	169	69.18 (± 10.22)
Autonomy	169	65.65 (± 14.00)

Table 3 presents a correlation matrix of all independent and dependent variables to be used in the linear regression modelling. The matrix shows that all variables are independently correlated with each other, except for the cognitive variable of Autonomy, which was not correlated with any other variables.

Table 3

Correlation Matrix of the Independent and Dependent Variables for Linear Regression Modelling.

	1	2	3	4	5	6	7
1. BDI-II							
2. MADRS	.60**						
3. Anxiety	.62**	.55**					
4. Rumination	.47**	.27**	.38**				
5. Dysfunctional Attitudes	.46**	.41**	.31**	.36**			
6. Sociotropy	.36**	.24**	.35**	.33**	.60**		
7. Autonomy	.05	.02	.05	.12	-.07	-.08	

** 0.01

Regression Modelling with Self-Report Depression as the Dependent Variable

Table 4 displays the multiple linear regression models in which the independent contribution of anxiety and cognitive variables to self-report depression is tested, as well the as the interaction of anxiety and cognitive variables to predict self-report depression. Each model is summarized using the statistical measure of R^2. It can be seen that the overall models for rumination (R^2 = .44, F (3,164) = 43.40, p < .01), dysfunctional attitudes (R^2 = .49, F (3,170) = 54.10, p < .01), sociotropy (R^2 = .40, F (3,165) = 37.12, p < .01) and autonomy (R^2 = .37, F (3,165) = 33.65, p < .01) are all significant.

Main effects were found for anxiety in all four multiple linear regression models (p < .05). A main effect was also found for rumination (Standardized β = .32, t = 2.71, p < .01). A main effect was also found for the independent contribution of dysfunctional attitudes (Standardized β = .42, t = 3.69, p < .01). However, no main effects were found for sociotropy (Standardized β = .12, t = 1.02, p = .308) or autonomy (Standardized β = .04, t = 0.32, p = .750). No interactions between cognitive variables and anxiety for predicting self-report depression were significant.

Table 4

Regression Models for the Main Effects of Anxiety and Cognitive Variables and, the Interaction Effects of Anxiety and Cognitive Variables for Predicting Self-Report Depression.

	Self-report depression		
	Standardized β	t	P
1.			
Anxiety	.65*	2.03	.044
Rumination	.32**	2.71	.008

Anxiety_Rumination	-.16	-0.44	.663
Model summary, R^2 = .44**			
2.			
Anxiety	.82**	3.69	.000
Dysfunctional Attitudes	.42**	3.69	.000
Anxiety_Dysfunctional Attitudes	-.34	-1.27	.205
Model summary, R^2 = .49**			
3.			
Anxiety	.46*	2.16	.032
Sociotropy	.12	1.02	.308
Anxiety_Sociotropy	.12	0.47	.641
Model summary, R^2 = .40**			
4.			
Anxiety	.67*	2.064	.041
Autonomy	.04	0.32	.750
Anxiety_Autonomy	-.07	-0.19	.854
Model summary, R^2 = .38**			

** 0.01, * 0.05

Final Hierarchical Regression Model for Predicting Self-Report Depression

Table 5 displays the final regression model for predicting self-report depression. This model only included the significant effects from the multiple linear regression modelling. The first step in the model included only anxiety and significantly explained 41% of the variance

in self-report depression (F (1,165) = 112.34, $p < .01$). After entry of dysfunctional attitudes at step two the total variance explained by the model as a whole is 47% (F (2,164) = 73.94, $p < .01$). The addition of dysfunctional attitudes caused a significant increase of variance accounted for by 7% (F change (1,164) = 21.55, $p < .01$). After the addition of rumination in step three the model then accounted for 52% of the total variance of self-report depression (F (3,163) = 58.79, $p < .01$). The addition of rumination significantly increased the variance accounted for by 5% (F change (1,163) = 15.45, $p < .01$). Therefore the final hierarchical regression model that best predicts self-report depression includes individual contributions of anxiety, dysfunctional attitudes and rumination.

Table 5

Final Hierarchical Regression Model of Significant Effects for Predicting Self-Report Depression.

	Self-report depression			
	R^2	R^2 change	Standardized β	t
Step 1	.41**	.41**		
Anxiety			.64**	10.60
Step 2	.47**	.07**		
Anxiety			.55**	9.29
Dysfunctional attitudes			.28**	4.64
Step 3	.52**	.05**		

Anxiety	.48**	8.11
Dysfunctional attitudes	.21**	3.56
Rumination	.24**	3.93

** 0.01

Regression Modelling with Clinician-Rated Depression as the Dependent Variable

Table 6 displays the multiple linear regression modelling the abilities of anxiety and cognitive variables to independently predict clinician-rated depression, as well the as the ability of the interaction of anxiety and cognitive to predict clinician-rated depression. Overall, the models for rumination (R^2 = .31, F (3,164) = 24.83, p < .01), dysfunctional attitudes (R^2 = .37, F (3,170) = 33.74, p < .01), sociotropy (R^2 = .31, F (3,165) = 24.56, p < .01), and autonomy (R^2 = .32, F (3,165) = 25.47, p < .01) are significant.

Although anxiety showed significant main effects when predicting self-report depression across all models, anxiety only shows a significant independent contribution in the multiple linear regression models for predicting clinician-rated depression in which dysfunctional attitudes (Standardized β = .60, t = 2.44, p < .05) and sociotropy (Standardized β = .46, t = 2.00, p < .05), plus their interactions with anxiety, are also predictors. However, in the regression models for rumination (Standardized β = .23, t = 0.65, p = .518) and autonomy (Standardized β = .03, t = 0.09, p = .927) there was not a significant main effect of anxiety. Main effects were also found for dysfunctional attitudes (Standardized β = .32, t = 2.48, p < .05), however no main effects were found for rumination (Standardized β = -.02, t = -0.17, p = .867), sociotropy (Standardized β = .01, t = 0.07, p = .944) or autonomy (Standardized β = -.21, t = -1.46, p = .146).

Again, no interaction effects were found for any of the cognitive variables with anxiety for predicting clinician-rated depression.

Table 6

Regression Model for the Main Effects of Anxiety and Rumination and, the Interaction Effects of Anxiety and Rumination for Predicting Clinician-Rated Depression.

	Clinician-rated depression		
	Standardized β	*t*	*P*
1.			
Anxiety	.23	0.65	.518
Rumination	-.02	-0.17	.867
Anxiety_Rumination	.35	0.85	.398
Model summary, R^2 = .31**			
2.			
Anxiety	.60*	2.44	.016
Dysfunctional attitudes	.32*	2.48	.014
Anxiety_Dysfunctional attitudes	-.15	-0.51	.614
Model summary, R^2 = .37**			
3.			
Anxiety	.46*	2.00	.047
Sociotropy	.01	0.07	.944
Anxiety_Sociotropy	.01	0.35	.725
Model summary, R^2 = .31**			
4.			

Anxiety	.03	0.09	.927
Autonomy	-.21	-1.46	.146
Anxiety_Autonomy	.58	1.55	.122
Model summary, R^2 = .32**			

** 0.01, * 0.05

Final Hierarchical Regression Model for Predicting Clinician-Rated Depression

Table 7 displays the final regression model for predicting clinician-rated depression. Again, this model only includes the significant effects from the multiple linear regression modelling. The first step in the model only includes anxiety and is able to significantly explain 31% of the variance in clinician-rated depression (F (1,172) = 77.70, p < .01). The second step added dysfunctional attitudes to the model and significantly increased the variance accounted for by the model by 6% (F change (1,171) = 16.64, p < .01). The final model, after the addition of dysfunctional attitudes, now accounts for 37% of the total variance of clinician-rated depression (F (2,171) = 50.70, p < .01) and includes anxiety and dysfunctional attitudes each contributing independently to clinician-rated depression.

Table 7

Final Hierarchical Regression Model of Significant Effects for Predicting Clinician-Rated Depression.

	Clinician-rated depression			
	R^2	R^2 change	Standardized β	t
Step 1	.31**	.31**		
Anxiety			.56**	8.81

Step 2	.37**	.06**		
Anxiety			.48**	7.52
Dysfunctional attitudes			.26**	4.08

** 0.01

Reverse Regression Modelling with Anxiety as the Dependent Variable, and the Depression Variable Measured via Self-Report.

Table 8 displays the multiple linear regression modelling of self-report depression and cognitive variables to independently predict anxiety, as well the as the ability of the interaction of self-report depression and cognitive variables to predict anxiety. This is the reverse of the initially proposed diathesis-anxiety model. Overall, the models for rumination (R^2 = .39, F (3,164) = 34.62, p < .01), dysfunctional attitudes (R^2 = .40, F (3,170) = 38.44, p < .01), sociotropy (R^2 = .39, F (3,165) = 36.99, p < .01), and autonomy (R^2 = .38, F (3,165) = 33.86, p < .01) are significant.

Self-report depression only shows a significant independent contribution in the multiple linear regression models for predicting anxiety in which dysfunctional attitudes (Standardized β = .55, t = 2.55, p < .05) and autonomy (Standardized β = .83, t = 2.637, p < .01), plus their interactions with self-report depression, are also predictors in the model. No other variables showed significant independent contributions and no significant interactions were found.

Table 8

Reverse Regression Model for the Main Effects of Self-Report Depression and Rumination and, the Interaction Effects of Self-Report Depression and Rumination for Predicting Anxiety.

	Anxiety		
	Standardized β	*t*	*P*
1.			
Self-report depression	.52	1.73	.086
Rumination	.09	0.52	.604
Depression_Rumination	.06	0.15	.884
Model summary, R^2 = .388**			
2.			
Self-report depression	.55*	2.55	.012
Dysfunctional Attitudes	-.05	-0.30	.763
Depression_Dysfunctional Attitudes	.13	0.41	.680
Model summary, R^2 = .40**			
3.			
Self-report depression	.38	1.78	.077
Sociotropy	.01	0.04	.967
Depression_Sociotropy	.28	0.89	.375
Model summary, R^2 = .40**			
4.			
Self-report depression	.83**	2.64	.009

Autonomy	.14	0.73	.464
Depression_Autonomy	-.25	-0.69	.494
Model summary, R^2 = .38**			

** 0.01, * 0.05

Table 9 displays the final hierarchical regression model for predicting anxiety. As only self-report depression was shown to significantly predict depression, this was the only variable in the final model. The final model accounts for 38% of the total variance of anxiety (F (1,174) = 107.27, p < .01).

Table 9

Final Hierarchical Regression Model of Significant Effects for Predicting Anxiety.

	Anxiety			
	R^2	R^2 change	Standardized β	t
Step 1	.38**			
Self-report depression			.62**	10.36

** 0.01

Reverse Regression Modelling with Anxiety as the Dependent Variable, and the Depression Variable Measured via Clinician-Rating.

Table 10 displays the multiple linear regression modelling of the ability for clinician-rated depression and cognitive variables to independently predict anxiety, as well the as the ability of the interaction of clinician-rated depression and cognitive variables to predict

anxiety. This is again the reverse of the initially proposed diathesis-anxiety model, however this analysis uses clinician-rated depression instead of self-report. Overall, the models for rumination (R^2 = .36, F (3,164) = 31.22, p < .01), dysfunctional attitudes (R^2 = .32, F (3,170) = 26.50, p < .01), sociotropy (R^2 = .39, F (3,165) = 34.28, p < .01), and autonomy (R^2 = .31, F (3,165) = 24.68, p < .01) are significant.

Clinician-rated depression shows a significant independent contribution in the multiple linear regression models for predicting anxiety in which dysfunctional attitudes (Standardized β = .52, t = 2.16, p < .05) it's interaction with self-report depression, is also a predictor. Whilst no other main effects were found, one interaction effect was found after controlling for a significant outlier. The Cook's distance value for the model in which Clinician-rated depression was the independent variable, and sociotropy, anxiety and the interaction of sociotropy and anxiety were the dependent variables had one significant outlier in which the Cook's distance value was above 1. Therefore the outlying case could be concluded as having an undue influence on the model as a whole (Pallant, 2010). However, this variable was not excluded from the model as when the case was examined it was found to be the case with the highest MADRS and lowest Sociotropy score and therefore it can be concluded that this outlier is non-significant to the model.

Table 10

Reverse Regression Model for the Main Effects of Clinician-Rated Depression and Rumination and, the Interaction Effects of Clinician-Rated Depression and Rumination for Predicting Anxiety.

	Anxiety		
	Standardized β	t	P
1.			

Clinician-rated depression	.22	0.68	.496
Rumination	.06	0.25	.804
Depression_Rumination	.37	0.85	.396
Model summary, R^2 = .36**			
2.			
Clinician-rated depression	.52*	2.16	.032
Dysfunctional Attitudes	.09	0.41	.682
Depression_Dysfunctional Attitudes	.00	0.00	1.00
Model summary, R^2 = .32**			
3.			
Clinician-rated depression	.20	1.03	.303
Sociotropy	-.07	-.36	.718
Depression_Sociotropy	.49	1.60	.111
Model summary, R^2 = .37**			
4.			
Clinician-rated depression	.35	1.14	.255
Autonomy	-.12	-0.48	.631
Depression_Autonomy	.26	0.66	.513
Model summary, R^2 = .31**			

** 0.01, * 0.05

Table 11 displays the final hierarchical regression model for predicting anxiety. This model only includes the significant main effects from the multiple linear regression modelling of the reverse diathesis-anxiety model with clinician-rated depression. This model contains only one step with clinician-rated depression being able to significantly explain 31% of the variance in anxiety (F (1,167) = 73.78, $p < .05$).

Table 11

Final Hierarchical Regression Model of Significant Effects for Predicting Anxiety.

	Anxiety			
	R^2	R^2 change	Standardized β	t
Step 1	.31**	.31**		
Clinician-rated depression			.55**	8.59

** 0.01

Discussion

Summary of Findings

The current study looked at the ability of the cognitive variables of rumination, sociotropy, autonomy, and dysfunctional attitudes to interact with anxiety to predict later depression in a clinical adult sample. Importantly, this study also examined the diathesis-anxiety model with both clinician-rated and self-reported measures depression in order to determine any discrepancies based on the reporting of depression symptoms by different informants, as this is often overlooked in research. A reverse diathesis-anxiety model was also performed to determine whether the interaction of depressive symptoms with cognitive variables to predict anxiety provided a better temporal and interactive explanation of comorbid anxiety and depression. The diathesis-anxiety model examined was based on that described in Cohen et al.'s (2014) study whereby they discovered that both self-criticism and rumination significantly interacted with anxiety to predict depressive symptoms in a non-clinical youth sample. However, in the current study none of the cognitive variables selected significantly interacted with anxiety to predict clinician-rated or self-reported depression. Interaction effects were also not found in the reverse model. However, there were significant direct relationships, or main effects, between certain variables tested for both models.

Firstly, it was found that anxiety did predict depression as rated by both participants and clinicians. The contribution of anxiety remained significant for self-reported depression even after controlling for the effects of the cognitive and interaction variables. However, anxiety was found to no longer be able to predict clinician-rated depression scores after statistically controlling for the effects of rumination and sociotropy, and their interaction variables.

Furthermore, dysfunctional attitudes were found to significantly predict both self-reported and clinician-rated depression, when controlling for the significant contribution of anxiety. However, again no interaction effects were found suggesting that dysfunctional attitudes contribute to depression in an independent manner to anxiety.

On inspection of the reverse diathesis-depression model the results also showed that both self-reported and clinician-rated depression significantly predicted anxiety. Furthermore, this relationship was no longer significant when rumination or sociotropy and their interaction variables were present in the model. Additionally, clinician-rated depression no longer made a significant contribution to the prediction of anxiety when the effects of autonomy and it's interaction variable were accounted for.

Discussion of Findings

The current study did not support the model proposed by Cohen et al. (2014) as no evidence was found for interaction effects between anxiety and the selected cognitive variables to predict depression of either the self-reported or clinician-rated kind. Therefore, the main hypothesis of this study was not confirmed. Although the current sample studied did not show support for the diathesis-anxiety model with the selected cognitive variables in an adult clinical population, past research (Feng et al., 2008; Hankin, 2008; Starr & Davila, 2012; Cohen et al., 2014) and explanatory theory such as the helplessness-hopelessness theory (Alloy et al., 1990) give support for the diathesis-anxiety model of comorbid anxiety and depression. Therefore, whilst the diathesis-anxiety model was not applicable to the current sample it may still be a useful hypothesis that, due to a number of studies confirming it's validity, needs to be further investigated.

The current study was able to further confirm the well-supported finding that anxiety symptoms largely precede depression symptoms (Brady & Kendall, 1992; Kaufman &

Charney, 2000; Avenevoli et al., 2001; Cohen et al., 2014). One theory that attempts to explain why this temporal association is so commonly found is Alloy, Kelly, Mineka & Clements' 1990 helplessness-hopeless theory of anxiety and depression. This theory describes helplessness as a component of both anxiety and depression that is characterised by "the expectation that future negative outcomes, should they happen, would be uncontrollable"(Swendsen, 1997). Whilst hopelessness is described as a component of depression characterised by "an expectation that future negative events will occur due to attributions that the causes of negative events are stable and global" (Swendsen, 1997). In regards to the temporal association of anxiety and depression's comorbidity, the helplessness-hopelessness theory proposes that anxiety and helplessness, are the initial responses to negative events, this can then evolve into hopelessness, and therefore depression, as the individual characterises the causes of negative events as stable and global (Swendsen, 1997). This theory also links with the current study as it suggests cognitive variables such as attributional styles of helplessness and hopelessness, may interact with anxiety to cause depression and therefore form the basis of the anxiety-diathesis model investigated in the current study. Furthermore, recent research has shown that cognitive attributional styles of hopelessness and rumination are important for explaining the temporal association between anxiety and depression (Starr & Davila, 2012).

The current study did not explicitly examine any other possible models of comorbidity, however the commonly proposed causal (Star et al., 2014; Mathew et al., 2011) and correlated liabilities models (Hong & Cheung, 2015; Grant et al., 2014; Rice et al., 2004) can be hypothetically explored given the results from the current study.

Firstly, the findings of the current study suggest that anxiety predicts depression, which appears to support the premise of the causal model, which proposes that anxiety directly causes depression. However, further statistical examination uncovered that this result

became insignificant when controlling for rumination and sociotropy. Furthermore, the reverse causality relationship of depression's ability to predict anxiety was also found to be insignificant when controlling for rumination, sociotropy and autonomy. This then suggests that these cognitive variables somehow significantly contribute to the relationship of anxiety and depression, perhaps through mediation, moderation or transdiagnostic factors, as has been the suggested in the case of rumination (McLaughlin & Nolen-Hoeksema, 2011). Therefore, the current findings do not support a directly causal relationship of comorbid anxiety and depression as regression analysis suggests the relationship is influenced by other variables, and is more complex than a simple causal link.

The second model that is often explored to try to understand the comorbidity of anxiety and depression is the correlated liabilities model. Whilst, again, this model was not directly tested in the present study the statistical analyses can be hypothetically explored in order to discover if there is support in our sample for the correlated liabilities model. As explained above, the direct relationship of depression and anxiety was bidirectionally affected by the cognitive variables of rumination and sociotropy, such that depression and anxiety's effects on each other were no longer statistically significant when rumination and sociotropy were controlled for. This is important as it suggests that rumination and sociotropy may be common etiological variables in the development of anxiety and depression.

There has been previous research to support rumination as a common etiological factor for anxiety and depression. In particular, McLaughlin and Nolen-Hoeksema (2011) found evidence as a transdiagnostic risk factor for anxiety and depression in a community sample of adults and adolescents. They suggested that rumination may create an increased risk for depressive and anxious symptoms through the direct effects of rumination on affect or possibly due to rumination leading to a number of negative consequences including poor

problem-solving, and decreased interpersonal functioning (McLaughlin & Nolen-Hoeksema, 2011).

Past research has also indicated that sociotropy may be related to both depression and anxiety (Alford & Gerrity, 1995). However, more recent research has not replicated these findings. Fresco et al.'s (2001) study on a group of undergraduate women found support for the relationship of sociotropy and anxiety across two time points, however they were unable to find support for the relationship of sociotropy to depression. Instead, they found depression to relate to autonomy (Fresco et al., 2001). Furthermore, research has suggested that the relationship between sociotropy and risk for depression and anxiety may be moderated by the coping style used in response to life stress (Connor-Smith & Compas, 2002). Those individuals who chose disengagement or avoidance coping strategies, such as those strategies characterised by low levels of acceptance and attempts to problem solve and high levels of avoidance and denial, had an amplified relationship between sociotropy and, depression and anxiety (Connor-Smith & Compas, 2002). Therefore, the current research on sociotropy's relationship to depression and anxiety is not clear and unfortunately the current study could not clarify this relationship.

It is important to note that the ability for anxiety to predict depression was only affected by the controlling of rumination and sociotropy for the clinician-rated depression measure, the MADRS. This could be for a number of reasons and therefore the interpretation of results as evidence of support for the correlated liabilities model should be done so with caution. The effect of controlling for rumination seen in the case of the clinician-rated MADRS but not the self-report measure of depression, the BDI-II, may be somewhat explained through the higher focus on psychological symptoms, such as rumination, of the MADRS compared to the BDI-II, which has a greater focus on physical symptoms (Cusin, Yang, Yeung, & Fava, 2009). Hence, the rumination scale used by this study to measure

rumination in participants may overlap a large portion of the MADRS measure in comparison to the BDI-II, which focuses less on psychological symptoms. Therefore, by controlling for the effects of rumination on the anxiety and clinician-rated depression relationship this may have controlled for a large proportion of the MADRS's effect. Furthermore, the current literature does not offer explanations as to why the effect of controlling for sociotropy differed for the two depression measures.

To further the discussion regarding differences in outcomes for self-report and clinician-rated depression, there has been research in the past into the differing accounts of depression given for the BDI-II and the MADRS. Firstly, it has been found that a number of different factors can effect differences in self-reported versus clinician-rated depression severity and symptoms (Domken et al., 1994). These can include demographic, clinical and personality variables (Enns et al., 2000).

In particular, gender has been found to play a role in the differences between self-report and clinician-rated depression. As this was not controlled for in the current study's regression analyses it may have affected differences in outcomes for self-reported and clinician-rated measures of depression. In particular, it has been found that males are observed by clinician's as exhibiting more sadness than females, but self-report less emotionally depressive symptoms (Carter, Joyce, Mulder, Luty, & McKenzie, 2000). Gender role theory offers a possible explanation for this occurrence such that the masculine gender role is inconsistent with emotionally depressive symptoms, whilst the feminine gender role permits the expression of emotions (Oliver & Toner, 1990). Therefore, this leads men to endorse higher levels of somatic and withdrawal symptoms whilst women endorse more emotional symptoms (Oliver & Toner, 1990). In further support of the role of gender roles, Oliver and Toner (1990) were also able to show that more feminine individuals reported more self-deprecating and emotional depressive symptoms compared to masculine

individuals. This further supports the idea that the way in which society and the individual views gender roles is important to consider when discrepancies are observed in self-reported versus clinician-rated depressive symptoms.

Furthermore, age may play a role in the discrepancies between self-reported and clinician-rated depression (Enns et al., 2000; Sayer et al., 1993). This was another variable that was not explicitly controlled for in the statistical analysis of the current sample. Although our sample contained only adults it has been shown that even amongst adult samples age can play a significant role in discrepancies of self-reported depression (Enns et al., 2000; Sayer et al., 1993). Enns et al. (2000) reported that whilst clinician-rated depression was consistent across the age range of their clinical adult sample, self-reported depression ratings were given more severe ratings by younger individuals on the psychological and cognitive symptom indices, however this was not found for somatic symptoms of depression. It is proposed that this may be due to a maturational effect such that individuals either report or experience less depressive symptoms as they mature (Enns et al., 2000). This was also inline with earlier findings by Domken et al. (1994).

An important clinical variable that has also been reported to play a role in the discrepancies between self-reported and clinician-rated depression is depressive subtype (Enns et al., 2000). Enns et al. (2000) distinguished those who had melancholic and atypical depressive subtypes in their research sample, as according to the DSM-IV, however our sample was not examined for subtypes of depressive symptoms. Enns et al. (2000) concluded that those participants with melancholic or atypical subtypes of depression, in comparison to those with depression without melancholic or atypical features, self-reported relatively lower depressive symptoms in comparison with observer ratings. On further examination Enns et al. (2000) discovered this was particularly prevalent with ratings of somatic symptoms for those with melancholic depression and with ratings of psychological distress for those with atypical

depression. Therefore it is possible that undetected subtypes of depressive symptoms an effect on self-report and clinician-rated depression variations in the current sample.

One other reason why self-report and clinician-rated depressive symptoms may vary pertains to personality variables of the individual. Enns et al. (2000) were able to show that a high neuroticism, low extraversion and low agreeableness was related to higher self-reported ratings of depressive symptoms relative to observer ratings. Furthermore, Domken et al. (1994) also reported finding in their study that self-esteem accounted for almost half of the variance between self-reported and clinician-rated measures of depression.

Another important finding to discuss from the current study is that dysfunctional attitudes were found to significantly predict both self-reported and clinician-rated depression, when controlling for the significant contribution of anxiety. Beck (1978, as cited in Lee & Hankin, 2009) originally theorized that dysfunctional attitudes commonly have themes of inadequacy and failure, leading to self-defeating thinking, which was then thought to lead to depression. Furthermore, there is a sizeable amount of literature surrounding dysfunctional attitudes in the form of Beck's cognitive triad of negative beliefs (Weissman & Beck, 1978) which continues to be linked to the thinking of depressed individuals today and is often considered when conceptualising an individuals depressive episode. Therefore, the finding that dysfunctional attitudes significantly predict self-reported and clinician-rated depression even when controlling for anxiety symptoms is not surprising.

Although dysfunctional attitudes significantly predicted both self-reported and clinician-rated depression, again no interaction effects were found suggesting that dysfunctional attitudes contribute to depression in an independent fashion to anxiety. It may not be that dysfunctional attitudes predict depression but rather that the measure of dysfunctional attitudes is mood-state dependent. There are a number of studies that suggest this (Miranda & Persons, 1988; Zuroff, Igreja & Mongrain, 1990; Farmer et al., 2001).

Miranda & Persons (1988) describe the mood-state dependence of dysfunctional attitude measures as due to the ability to access and report dysfunctional attitudes as being dependent on the individual's mood. Therefore, a person who is currently severely depressed is likely to report higher levels of dysfunctional attitudes compared to someone who is currently mildly depressed. This effect may also operate in the opposite fashion also, such that a person who experiences an increase in dysfunctional attitudes reports increased symptoms of depression. As our sample was administered the depression and dysfunctional attitude ratings at the same time point it is no surprise, given the research on the mood-state dependence of dysfunctional attitudes, that the dysfunctional attitudes measures is closely related to the depression measure.

Limitations

The current study contained a number of limitations. Firstly, this study was not a longitudinal study and therefore was not able to measure the development of comorbidity over time. Instead, this study took data from one time point and statistically modelled outcomes. Whilst this method is still valid, a longitudinal study would be preferential to garner the clearest understanding of the comorbid relationship of anxiety and depression within a diathesis-anxiety model. However, longitudinal research also has a number of downfalls, including the cost and time required to complete this standard of research.

Whilst this study has done what many others have not by collecting both self-report and clinician-rated measures of depression, it is limited by the fact that other measures used were entirely self-report. Therefore, whilst the ratings of depression controlled for confounding factors related to self-report biases, the measures of anxiety and cognitive variables did not. This may influence results as it was able to be demonstrated that the current sample showed differences between self-reported and clinician-rated measures of depression,

therefore it is likely that there would be differences between self-report and clinician-ratings on other measures. This should therefore be considered when interpreting results.

Although the sample used by this study extended the research to other populations than previously used to examine the diathesis-anxiety model, there are still some limitations within the sample used. Firstly, 88.7% of the sample was NZ/European in ethnicity, indicating that the population used was not very ethnically diverse. Therefore, the data obtained from this sample is limited by cultural differences between ethnicities. Secondly, the sample was 72.3% female; therefore the ability for results to be extended to males is limited by the largely female sample on which results were obtained, furthermore gender theory has shown that males and females exhibit differing symptoms of depression (Oliver & Toner, 1990) which further indicates that research based on largely female samples may not be able to be translated fully to males. However, it is important to note that depression and anxiety are more common in females, including comorbidity of the two (McLean, Asnaani, Litz, & Hofmann, 2011), and therefore this sample is perhaps more representative of a clinical population than one of equal number of female and male participants.

The final limitation of the current study is based upon the statistical methods used. Due to reasons described in the methods section above, listwise deletion was used in the statistical analyses. Whilst this was the better option for analysing the data at hand, this approach does have its limitations. The listwise approach drops from analysis any cases with missing values. If the cases are missing at random, of which they are in the studied sample, then listwise deletion does not bias results (Howell, 2007). The only limitation for the current study using listwise is that the statistical power is likely to have been lowered. Given the large sample however, this is of minimal effect.

Strengths

Despite not replicating the results found by Cohen et al. (2014) this study did have a number of strengths. Firstly, unlike the Cohen et al. (2014) study it used a clinical sample with a primary diagnosis of MDD. Therefore, the results found can be applied in clinical practise to those who are primarily clinically depressed. Previous research of the diathesis-anxiety model also shows that no prior studies have tested the model on a clinically comorbid sample. The sample used in the current study was also an adult population. The original sample used by Cohen et al. (2014) was an adolescent sample and therefore the current study gives important information about the use of the diathesis-anxiety model on an adult population. Past research shows only one study previous using an adult sample (Star & Davila, 2012).

The current study was also able to examine the possible differences between self-reported and clinician-rated depression within the diathesis-anxiety model. Many studies do not examine this and therefore it often is not clear what the impact of assessment methods have on outcomes. This is further demonstrated in the current study with the discrepancies found between clinician-rated and self-reported depression.

Another methodological strength of the current study is that all measures used were taken at the same time for each participant. This meant that measures were more likely to be consistent and be able to better show relationships across measures compared to measures taken at different time intervals. Furthermore, participants in the current study were free of antidepressant medication meaning that the effects or side effects of medication use did not affect the results shown. Participants were also only included if they had not had an adequate trial of CBT or IPT in the last year. Therefore, the effects of therapy did not influence measures taken from participants. These are all important methodological strengths as they work to control for confounding factors that may otherwise influence results.

Although the Cohen et al. (2014) results were not replicated on an adult clinical sample, the results still give important information about the diathesis-anxiety model proposed by Cohen et al. (2014). Most importantly, the current study indicates that there are likely differences in the relationship between comorbid anxiety and depression for non-clinically depressed adolescents and clinically depressed adults due to difference in findings between the current sample and that of Cohen et al.'s (2014) research. This is likely to occur for a number of reasons including cognitive, emotional and social maturational differences. The role of cognitive variables in this comorbid relationship for these two groups is also likely to differ. The results from the current study therefore also highlight the importance of using clinically matched data to inform clinical practice due to the number of differences that can occur between populations.

Implications for Clinical Practice

The current study contributes valuable information to the current literature on the comorbidity of anxiety and depression such as the disconfirmation of sociotropy, autonomy, rumination and dysfunctional attitudes as contributing cognitive variables in the diathesis-anxiety model of comorbid depression and anxiety. However, the true nature of the relationship between comorbid anxiety and depression continues to not be well understood. It is highly important that we continue to examine and test models for explaining how comorbid anxiety and depression develop as this phenomenon is highly prevalent amongst clinical populations (Kaufman & Charney, 2000) and leads to greater disability compared to those with 'pure' forms of psychiatric illness (Teeson et al., 2009).

The current study showed discrepancies in clinician-rated versus self-reported measures of depression. These results highlight the importance of using multiple informants and measures in research and clinical practice. In the research setting the use of multiple

measures is likely to reduce biases that could be influencing results including possible influences of age, subtypes of depressive presentations, severity of symptoms, education levels and personality variables such as neuroticism, extraversion and agreeableness (Enns, Larsen & Cox, 2000). Therefore, research conducted can display a better understanding of the phenomena examined and better apply this knowledge to clinical practice by identifying biases or discrepancies in psychometrics used that may need to be explored and clarified further with clients in order to reduce errors in understanding a client's issues. Furthermore, within clinical practise emphasizing the importance of using clinician-rated and self-reported measures of symptoms is essential as they are likely to highlight differences in perceptions of the problem between the individual and the clinician which, if undetected, may undermine treatment, collaboration and compliance (Domken et al., 1994).

Results from this study supported the commonly held theory that anxiety precedes depression, however evidence was also found for depression preceding anxiety. In regards to clinical practice, this is important as those with depression and anxiety should both be monitored for possible comorbid anxiety and depression respectively. This also means that clinical assessment should always investigate the possible presence of both anxiety and depressive symptoms, never one alone. Furthermore, those individuals seeking treatment for either anxiety or depression should have included in their treatment preventative measures to reduce the risk of developing comorbid depression or anxiety, respectively.

Cohen et al.'s (2014) results suggested that those cognitive variables found to interact with anxiety to cause depression should be specifically targeted in treatment for adolescent individuals with comorbid anxiety and depression. However, as the current study did not find any interaction effects for the cognitive variables examined this suggests that interventions targeting the cognitive variables examined in treatment with adults who have comorbid anxiety and depression should be reduced.

Although, the current study did not give support to the Cohen et al. (2014) model, it did show that research on a community adolescent sample could not be translated to a clinical adult sample. Therefore, those working with clinically depressed and anxious adults should take caution when interpreting research that was not conducted on a sample homogenous to their client population. This is important for both clinical assessment and treatment in order to maximise the benefit and reduce the harm to those with mental illness, as well as maximise the use of resources.

Future Directions of Research

The current study contributes towards the literature that attempts to explain comorbid anxiety and depression. This study indicates that there is unlikely an interactive relationship between anxiety and the cognitive variables studied that predicts depression symptoms. Therefore, this research has implications for future research such that it suggests exploring further cognitive variables, other than those studied in the present study, within a diathesis-anxiety model. Such cognitive variables could include those related to cognitive biases. As one comprehensive review found, those individuals with emotional disorders have been shown to selectively attend to emotionally congruent clues, recall unpleasant memories more often and have a bias towards negative interpretation of ambiguous stimuli compared to more emotionally stable individuals (Mathews & MacLeod, 2005). The cognitive variables pertaining to cognitive biases would be important to examine and are more commonly labelled as negative ideation, worry and selective attention.

The previous research conducted suggests that the diathesis-anxiety model is likely to be a valid model of comorbidity and therefore exploring the limitations and applicability of this model to other samples and variables is important. In particular, further exploration of the model in a clinical population is important to complete due to the lack of clinical samples

on which the model has been tested. Furthermore, there is a range of other cognitive variables that could be tested in the model also.

In order to further extended the research on the diathesis-anxiety model for understanding the comorbidity of anxiety and depression it would be imperative that a longitudinal study that consisted of measures of a wide range of cognitive variables be completed on a clinical population. This study would not only be able to test the validity of the diathesis-anxiety model, but it would also be important to place the data within other models, including the causal and correlated liabilities models, to test their significance. Whilst a longitudinal study of this magnitude would be costly and time consuming, the knowledge gained from a study such as this would far out weigh the financial cost given the current psychological and financial burden comorbid mental illness current have on our mental health system and those who suffer from them.

In summary, this study has provided valuable information regarding the utility of Cohen et al.'s (2014) proposed diathesis-anxiety model for explaining comorbid anxiety and depression on a clinical adult population. This research had not previously been conducted and therefore indicates a number of future directions that would be useful for expanding knowledge of comorbid depression and anxiety, as well as how to prevent it.

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