
Electronic Thesis and Dissertation Repository

8-24-2016 12:00 AM

Cognitive Schemas as Longitudinal Predictors of Depressive Relapse/Recurrence in an Undergraduate Population

Daniel Machado, *The University of Western Ontario*

Supervisor: Dr. David Dozois, *The University of Western Ontario*

A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Psychology

© Daniel Machado 2016

Follow this and additional works at: <https://ir.lib.uwo.ca/etd>



Part of the [Clinical Psychology Commons](#)

Recommended Citation

Machado, Daniel, "Cognitive Schemas as Longitudinal Predictors of Depressive Relapse/Recurrence in an Undergraduate Population" (2016). *Electronic Thesis and Dissertation Repository*. 4046.
<https://ir.lib.uwo.ca/etd/4046>

This Dissertation/Thesis is brought to you for free and open access by Scholarship@Western. It has been accepted for inclusion in Electronic Thesis and Dissertation Repository by an authorized administrator of Scholarship@Western. For more information, please contact wlsadmin@uwo.ca.

Abstract

This study assessed longitudinally the predictors of relapse/recurrence in major depressive disorder, as well as future depressive symptomatology, by examining how people organize information and beliefs about the self and how this changes over time. A secondary objective was to assess the long-term stability of self-schema structures. A sample of undergraduate students completed a computer-based task assessing schema structure, as well as two measures of schema content at baseline, and three and six-month intervals. Analyses for relapse/recurrence yielded insignificant results. However, as predicted, social cognitive distortions at Time 1 and schema structure for negative interpersonal content at Time 1 predicted depressive symptoms at Time 2. These predictors at Time 2 were not significant when assessing depressive symptoms at Time 3. Additionally, negative structure became less stable over time, whereas positive structure showed the opposite pattern. The implications of these findings are discussed and directions for future research outlined.

Keywords: depression; relapse; recurrence; self-schemas; cognitive vulnerability

Acknowledgements

First of all, I would like to express my heartfelt gratitude to my supervisor, Dr. David Dozois. You gave so generously with your time, knowledge, support, and patience. I have been shaped immensely by your expertise and guidance, and I am excited to continue working with you in the years to come.

Special thanks to Erin Shumlich, Katerina Rnic, Nadia Maiolino – taking time out of your busy schedules to help administer the SCID interviews was immeasurably appreciated. Sincere thanks to my research team: Teigan Chorny, Rachel Dozois, Victor Foo, Callie Forchuk, Sam Kingissepp, Chelsea Sinclair, Scott Squires, and James Welsman. Your dedication and work ethic day in and day out over nearly a year of data collection was admirable.

I would also like to thank my committee members, Dr. Nick Kuiper, Dr. Leora Swartzman, and Dr. Alan Leschied for your time and for your valuable feedback. Special thanks to Dr. Rod Martin who programmed and organized the online questionnaires for the study; your time and effort was very much appreciated.

Thank you to my lab mates, Katerina Rnic, Lindsay Szota, and Jesse Wilde, for your support and of course all of the laughs. You helped make my MSc years a great experience. Finally, I want to thank my family and friends for all the love and encouragement over the years. You have always believed in me, and supported me in my ventures non-judgmentally. This thesis is dedicated to you.

Table of Contents

| | |
|----------------------------|-----|
| Abstract and Keywords..... | ii |
| Acknowledgements..... | iii |
| Table of Contents..... | iv |
| List of Tables..... | v |
| Introduction..... | 1 |
| Method..... | 20 |
| Participants..... | 20 |
| Materials..... | 21 |
| Procedure | 25 |
| Results | 27 |
| Discussion..... | 39 |
| References | 58 |
| Appendices..... | 81 |
| Curriculum Vitae..... | 94 |

List of Tables

| <i>Table</i> | | <i>Page</i> |
|--------------|--|-------------|
| Table 1 | Logistic Regression: Predictors of Relapse/Recurrence at Time 2 | 29 |
| Table 2 | Logistic Regression: Predictors of Relapse/Recurrence at Time 3 | 30 |
| Table 3 | Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 2 | 33 |
| Table 4 | Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 3 (Time 1 Predictors) | 34 |
| Table 5 | Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 3 (Time 2 Predictors) | 36 |
| Table 6 | Self-Schema Stability Over Time | 38 |

List of Appendices

| <i>Appendix</i> | | <i>Page</i> |
|-----------------|---|-------------|
| Appendix A | Advertisement Poster: Western University Campus | 81 |
| Appendix B | Advertisement: Western University Facebook Groups | 82 |
| Appendix C | Participant Initial Contact Script: Email | 83 |
| Appendix D | Participant Initial Contact Script: Phone | 84 |
| Appendix E | Participant Information Letter and Consent Form | 85 |
| Appendix F | Participant Debriefing Letter | 89 |
| Appendix G | Ethics Board Approval | 93 |

Cognitive Schemas as Longitudinal Predictors of Depressive Relapse/Recurrence in an Undergraduate Population

Introduction

Major Depressive Disorder (MDD) is among the most prevalent of mental disorders, affecting an estimated 350 million people worldwide (World Health Organization [WHO], 2015). Roughly one in six men, and one in four women, will experience at least one episode of major depression in their lifetime (Kessler et al., 1994). Depression is not only prolific, its effects are frequently devastating; in Canada, depression is responsible for more years of life lived at less than full function, and years lost to early death, than all other mental disorders. Depression has more than twice the impact of the next most impairing mental disorder (Bipolar Disorder), and carries more than the combined burden of lung, colorectal, breast, and prostate cancers (Ratnasingham, Cairney, Manson, Rehm, & Lin, 2013). Moreover, depression is expected to be the second leading cause of disability worldwide by the year 2020, trailing only heart disease (WHO, 2015). The reach and impact of depression are widespread, with debilitating effects in multiple life domains.

Achieving a greater understanding of the factors that precipitate and maintain the disorder is of paramount importance to our national mental health strategy, as depression is a highly recurrent disorder across the lifespan. Individuals who experience a first episode of depression have a 50% chance of recurrence at some point in their lifetime, whereas those with a history of two or more episodes have an 80% chance of recurrence (Kupfer, Frank, & Wamhoff, 1996). However, the clinical focus on depression has

traditionally been one of symptom relief for a temporary disorder, and consequently research regarding depression's chronicity has been lacking (Segal et al., 2006). With such a large proportion of individuals suffering more than one episode, it is important to develop research that elucidates the risk factors for depressive relapse/recurrence.

Increasing our understanding of the factors that underlie depressive relapse/recurrence could aid prevention efforts, as well as improve the efficiency of service delivery and the distribution of mental health resources. Wilson, Joffe, & Wilkerson (2000) reported that major depression costs the North American economy upwards of \$60 billion per year; another estimate puts the costs of treatment alone in the United States at \$26.1 billion (Greenberg et al., 2010). Given the statistics regarding the risk of relapse/recurrence, it is probable that treating repeat sufferers has inflated both of these figures.

In addition to this growing financial burden, the incidence rates of depression remain staggering. So if we cannot prevent the train from leaving the station, perhaps we can prevent it from repeatedly looping around the tracks. Bolstering prevention efforts by increasing research that identifies precipitating factors behind the cyclic nature of the disorder could raise the figurative trip arm on the railway track and help bring this runaway train to a halt. Post (1992) adapted the kindling model of behavioural activation from neuropsychology as a possible explanation for the multiplication of risk that occurs with repeated episodes of depression. Post suggests that with subsequent occurrences, episodes of depression become more "autonomous"—or less predictable—and therefore less traceable to established precipitants such as stressful life events. Essentially, the tinder that sparks an episode of depression becomes more flammable with the occurrence

of each episode. Alternatively, the “scar” hypothesis asserts that the experience of depressive episodes leads to the development of enduring psychological traits—or scars—that elevate one’s risk for developing future episodes, in comparison to those with no previous episodes of depression (Lewinsohn, Steinmetz, Larson, & Franklin, 1981). However, at this time, the exact mechanisms surrounding recurrence remain beyond our grasp (Dozois & Dobson, 2004).

Definitional Considerations

The literature base on depressive relapse/recurrence has been characterized by inconsistent application from study to study of clear definitions for what constitutes relapse and recurrence (Frank et al., 1991). This variance creates difficulties in categorizing and interpreting studies, and can call into question the validity of conclusions reached. In 1988, the MacArthur Foundation Research Network on the Psychobiology of Depression assembled a task force with the mandate to develop clear operational criteria for describing and studying the course of major depression; specifically, the terms *remission*, *recovery*, *relapse*, and *recurrence* (Frank et al., 1991). According to Frank et al. (1991), the task force defines *remission* as a brief period during which an individual no longer meets diagnostic criteria for major depression (i.e., has no more than minimal symptoms), and *recovery* as remission that lasts for a certain number of days or longer (usually 8 weeks; Burcusa & Iacono, 2007). *Relapse* occurs when symptoms return—and meet full diagnostic criteria for an episode of major depression—during a period of remission, but before an individual meets definitional criteria for

recovery (i.e., within 8 weeks). Lastly, *recurrence* is when an individual meets diagnostic criteria for a new episode of major depression during the recovery period.

Risk Factors for Relapse/Recurrence

The overall research base on depressive relapse/recurrence is relatively sparse. Hardeveld, Spijker, De Graaf, Nolen, & Beekman (2010) conducted a meta-analysis of research performed within the last 30 years on risk factors of recurrence. The strongest predictors were the number of previous depressive episodes, residual (sub-clinical) symptoms after recovery from the last depressive episode, and childhood maltreatment. As a further illustration of how uncultivated investigation into the chronicity of depression is, only two studies to date—both conducted within the last two years—have sought to develop a prediction algorithm for depressive relapse/recurrence. Wang et al. (2014) examined sample data of 1518 individuals from the U.S. National Epidemiological Survey on Alcohol and Related Condition who had a diagnosis of major depression at baseline. Number of previous episodes, residual symptoms, co-occurring physical health problems, co-morbid mental health disorders, and psychosocial difficulties were all found to be important prognostic factors. van Loo, Aggen, Gardner, and Kendler (2015) used prospective data from a sample of female twins who had experienced an episode of depression within the past year. Significant predictors of relapse identified included: depression and anxiety symptoms during the index episode, family history, and early and recent stressful life events.

The cross-sectional literature base contains additional evidence for risk factors of relapse/recurrence. Commonly-cited findings are: family history of depression (e.g.,

Daley, Hammen, & Rao, 2000; Eccleston & Scott, 1991; Gonzales, Lewinsohn, & Clarke, 1985), earlier age of onset (e.g., Eccleston & Scott, 1991; Giles, Jarrett, Biggs, Guzick, & Rush, 1989; O’Leary, 1989), and being unmarried (Johansson, Lundh, & Bjärehed, 2015; Mueller, Leon, Keller, Solomon, & Endicott, 1999; van Loo et al., 2015). Other risk factors that have been reported include severity of symptoms during the index episode (Barkow et al., 2003; O’Leary, Costello, Gormley, & Webb, 2000; van Loo et al., 2015); suicidal ideation during the index episode (Barkow et al., 2003); female gender (Mueller et al., 1999; Gonzales et al., 1985)—but only as a risk factor for relapse, and not recurrence (see Burcusa & Iacono, 2007, for a review); high neuroticism (Eccleston & Scott, 1991; Berlanga, 1999); poor overall health, and life role dissatisfaction (Gonzales et al., 1985); higher levels of marital distress and perceived criticism (Hooley & Teasdale, 1989); more years of education (O’Leary, 1989); multiple stressful life events before and after onset of the first episode (Eccleston & Scott, 1991; Gonzales et al., 1985); and chronic stress (Bockting, Spinhoven, Koeter, Wouters, & Schene, 2006; Daley, Hammen, & Rao, 2000; Sheets & Craighead, 2014). Overall, empirical research has not consistently identified risk factors in the cross-sectional database (Hardeveld et al., 2010; Nanni, Uher, & Danese, 2012). Potential reasons for this disparity include the use of fairly homogeneous samples (e.g., women, twins) in many studies and the evaluation of groups in the context of treatment programs (e.g., participants who receive concurrent Cognitive Behavioural Therapy rather) than a more naturalistic study designs.

Etiology

Major depression is a complex disorder characterized by a multitude of etiological factors, with biological, developmental, interpersonal, affective, and behavioural inputs postulated. In the cognitive school, a wide array of research evidence has converged to support a diathesis-stress perspective of vulnerability to depression. In this paradigm, cognitive variables are the diatheses that interact with life stressors, such that differences in these variables between vulnerable and non-vulnerable individuals determine whether or not a given stressor will lead to the onset of a depressive episode (Abramson, Metalsky, & Alloy, 1988; Luxton, Ingram, & Wenzlaff, 2006; Monroe & Simons, 1991). According to Aaron Beck's (1967) cognitive model, depressive symptoms are largely caused by maladaptive cognitive styles. Beck's theory of depression has a three-fold focus: the cognitive triad (cognitions about oneself, the world, and one's future), information-processing errors (e.g., cognitive distortions, attention and memories biases), and self-schemas. Schemas are a key component of Beck's model of depression, as they impose order and organization on one's thoughts (including the cognitive triad), and facilitate information-processing. Schemas have been defined as "the basic structural components of cognitive organization through which humans come to identify, interpret, categorize, and evaluate their experiences" (Schmidt, Schmidt, & Young, 1999, p. 129). Schemas are believed to originate in childhood, and are expanded on throughout development; they are used to efficiently organize aspects of an individual's experience, based on input from internal and external sources, and provide a lens through which future experiences are filtered (Beck, 1967; Segal, 1988). Our individual experiences are varied, and the development of our internal world is shaped—adaptively and maladaptively—by our subjective interpretations of these events (Riskind & Alloy,

2006). Schemas also have influence at an emotional level, in that the organization of information within these structures affects the generation of mood states such as depression, by facilitating more fluid processing of negative information (Clark, Beck, & Alford, 1999).

Beck (1967) theorized that depressive schemas originate early in life in response to adverse experiences, and remain latent until they are kindled by the spark of a stressful experience later in life. Specifically, individuals who have a predisposition to depression are prone to encoding self-relevant information in a negative fashion; so when they experience a stressor similar to one that was originally experienced in childhood (e.g., interpersonal rejection, loss of a close relationship), the maladaptive schema is activated. This latent activation triggers a cascade of further activity that leads an individual to generalize feelings of negativity to other situations or environments, entrapping them in a pattern of inaccurate perceptions that promotes further negative affect.

In a revision of the schema concept, Young (1995) identified 15 early maladaptive schemas (EMSs)—comprising five schema domains—that he proposed develop through unmet developmental needs in childhood. Young's (1995) schemas are dimensional, varying in severity and thus the degree of risk they confer to the development of psychopathology. Schemas within the domains of Connection and Rejection (which pertain to security and acceptance needs) and Impaired Autonomy (expectations about the self and environment that impede one's perceived ability to function in the world, or achieve success independently) have been linked in several studies to the development of depressive symptomatology (Calvete, Estévez, López de

Arroyabe, & Ruiz, 2005; Lumley & Harkness, 2007; Eberhart, Auerbach, Bigda-Peyton, & Abela, 2011).

Schema content and structure

Cognitive schemas can be delineated into two separate, yet interrelated constructs: content and structure. Schema content refers to one's fundamental beliefs, which are largely formed through previous learning experiences (Clark et al., 1999). Beck (1976) conceptualized depressive schema content as operating at different "levels" of thought, and consisting of negative automatic thoughts, dysfunctional attitudes, and core beliefs. Negative automatic thoughts are the most surface-level of these constructs, and refer to the stream of consciousness-like thoughts that go through one's mind at any given time (Beck & Dozois, 2008). These thoughts are spontaneous—rather than deliberate—and believed to be related to one's core belief system, in that they seem to arise as by-products of schematic activation from environmental stimuli (Dobson & Dozois, 2008). Dysfunctional attitudes follow in the levels of processing hierarchy (Beck, 1976), and are characterized by negative assumptions pertaining to the cognitive triad, which is strongly associated with vulnerability to depression (Beck, 1979). These negative assumptions are conditional in nature, taking the form of "if-then" statements, such as "*If* everyone does not like me, *then* I am not a worthwhile individual" (Dobson & Dozois, 2008). Core beliefs exist at the deepest level of consciousness, and are absolute and axiomatic holdings that influence one's perceptions of a given situation (Leahy & Dowd, 2002). Core beliefs can be either negative in valence (e.g., "I am unlovable"; Beck, 1967), or positive (e.g., "I'm a good person"; Beck, 1967). Cognitive theorists assert that negative

beliefs in either interpersonal or achievement domains (Beck referred to similar constructs as *sociotropy* and *autonomy*) can confer vulnerability to depression when matched with congruent stressful life events (Coyne & Whiffen, 1995), and measures of schema content have factor structures that allow the instruments to be parsed into scales that allow the two domains to be analyzed individually.

Self-schema structure refers to the organizational properties of self-relevant information; specifically, how the information within this framework is arranged, integrated, and stored (Ingram, Miranda, & Segal, 1998). Self-schemas vary structurally in the degree of how tightly-packed—or interconnected—they are, which influences information-processing (Clark et al., 1999). That is, information becomes more readily accessible and encoded as one's existing self-structure becomes more closely-organized (Segal, 1988). The majority of schema research to date has focused on content, as structure is inherently less accessible, and thus more difficult to measure in a research paradigm. Traditionally, semantic priming or self-referent encoding tasks—such as modified Stroop designs—have been used to investigate these cognitive processes (Segal & Vella, 1990, Dozois & Dobson, 2001). In a classic Stroop design, individual words are presented in contrasting colours (e.g., the word “blue” is shown in yellow text). Participants are tasked with naming the colour of the stimulus (yellow), which will ostensibly require them to first suppress the meaning of the word (blue). This pairing of incongruent stimuli been shown to result in longer response times—compared to pairing congruent stimuli, such as the word “blue” in blue text—from the interference that comes from suppressing the word meaning in order to name the text colour, which is referred to as the Stroop Effect (Epp, Dobson, Dozois, & Frewen, 2012; MacLeod, 1991). The

Stroop paradigm has been altered to include various disorder-specific stimuli, in what is called an “Emotional” or a “Modified” Stroop design (Epp et al., 2012). In a modified Stroop design, participants are shown a series of word pairs on a screen. However, instead of colours, the content of the stimuli shown on the screen are related to the specific disorder that is being investigated. Stroop designs for depression often use mood-specific words such as sad, and upset. Longer response times for naming these depression-related words—compared to neutral or positive words—are theoretically an indication of a negative attentional bias (Epp et al., 2012; Gotlib, et al., 1996). These tasks can also reveal information about the structure of the underlying self-system. When presented with sequential words related to a negative self-view, it is expected that priming one word will lead to activation of the other, since the constructs exist within a closely interrelated neural system (Segal & Gemar, 1997).

When assessing depressive schemas, self-referent adjectives are typically used as stimuli, which can be used to tap into and make inferences about self-structure. In one such task, Segal and Vella (1990) asked depressed and non-depressed participants to colour-name self-descriptive adjectives. Participants were presented with a prime word, followed by the target self-referent adjective that appeared in coloured text. Segal and Vella (1990) found that longer response times (latencies) were observed in both groups (when the non-depressed participants were placed in a heightened state of self-awareness) when the prime and target words were both adjectives that were rated as personally meaningful to the participant, compared to when only the target word was rated as personally meaningful, or the prime word was neutral. Conversely, without the self-awareness induction, non-depressed participants displayed a much weaker prime-target

relatedness effect. These results provide an example of how self-referent adjectives can be used to theoretically provide information on the underlying structure of an individual's self-schema. Individuals with a highly-consolidated negative self-schema are assumed to be slower to colour-name an adjective that is negatively self-referent when it is preceded by one that is also negatively self-referent—compared to a neutral adjective—because spreading activation from processing the related meaning of the words ostensibly interferes with the colour-naming task (Segal & Vella, 1990). Individuals with highly consolidated negative schemas also tend to endorse more negative traits as being self-descriptive than do controls (Segal, 1988). These results are believed to illuminate underlying processes of depressogenic thinking patterns, in that negative self-traits are closely linked structurally, and being exposed to negative information about the self renders it more likely that nearby elements in the negative self-schema structure will also be activated (Segal & Vella, 1990).

More recently, schema structure has been assessed using the Psychological Distance Scaling Task (PDST; Dozois & Dobson, 2001b), which asks individuals to position adjectives on a grid based on each adjective's level of self-descriptiveness and valence. Research with this measure has shown that the degree of organization of one's schema may be a vulnerability factor for depression (Dozois, 2007; Seeds & Dozois, 2010). For instance, individuals with depression have more tightly- interconnected negative schema structure and content, and more loosely-interconnected positive structure and content than do healthy controls (Dozois & Dobson, 2001b; Dozois & Frewen, 2006). Moreover, whereas negative content tends to disperse in depressed

individuals following remission of the depressive episode, negative cognitive structure tends to remain well-organized (Dozois, 2007; Dozois and Dobson, 2001a).

Cognitive reactivity and depressive relapse/recurrence

The link between dysphoric mood and negative information-processing in currently-depressed (Gotlib & MacLeod, 1997; Williams, Watts, MacLeod, & Mathews, 1988) and non-depressed individuals (Ingram, Bernet, & McLaughlin, 1994; Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990), and the resulting relationship to vulnerability to depression has been well-established. When in a negative mood state, individuals exhibit biases toward attending to and processing negative information; in individuals at risk for depression, this appears to constitute a vulnerability factor. When assessing risk among remitted/recovered individuals, the degree of depressogenic information-processing exhibited may be an especially important marker. Research has shown that the link between negative information-processing and dysphoria is stronger among previously-depressed individuals than it is with those experiencing first onset or with non-depressed controls, using measures of dysfunctional attitudes (Lewinsohn, Allen, Seeley, & Gotlib, 1999; Miranda & Persons, 1988; Miranda et al., 1990) and life stress (Lewinsohn et al., 1999).

Remitted/recovered individuals appear to be especially prone to these cognitive errors, and a possible explanation can be found in an enduring form of cognitive reactivity that has been proposed as a vulnerability factor in this population (Hollon et al., 2006; Kovacs & Beck, 1978). This theory postulates that residual depressogenic thinking styles that were present during the previous episode of depression and either became

latent, or persisted to a certain extent following remission/recovery, can be re-activated under certain stressful conditions (Scher, Ingram, & Segal, 2005). In one study, Timbremont and Braet (2004) induced dysphoric mood in three groups of adolescents: currently depressed, recovered, and never depressed. The authors then asked participants to rate a series of adjectives on their degree of valence and self-descriptiveness. Results showed that both the currently depressed and recovered groups rated more negative words as being self-descriptive than did the never depressed group.

Segal and Gemar (1997) found similar results with an adult sample using a modified Stroop design. Segal et al. (2006) followed a sample of remitted individuals over a period of 18 months who had undergone a sad mood induction, and found that relapse was significantly predicted by the magnitude of mood-linked cognitive reactivity. These findings all suggest that depressogenic thinking styles persist after recovery and are accessible, provided appropriately fertile conditions are present to activate the vulnerability (Segal et al., 2006). Therefore, negative schemas and the conditions in which they become predictive of relapse/recurrence are potentially important risk factors that require further study outside of a sad mood induction.

Relapse/Recurrence Risk and Interpersonal Schemas

Relapse/recurrence becomes increasingly likely with repeated episodes of depression, but there remains a subset of individuals that remains remitted/recovered. After experiencing a first episode of depression, 50% will not relapse/recur, and after two or more episodes, 20% will not relapse/recur (Kessler et al., 1994). It is important to investigate what differentiates these two groups, and the factors that leads an individual

down a pathway of relapse/recurrence versus a path of recovery. To this end, looking for differences in cognitive organization may be a fruitful endeavor. Negative cognitive structure tends to remain densely interconnected even after a current episode of depression has remitted—this is especially true for interpersonal content—and therefore may be a stable vulnerability factor for relapse/recurrence (Dozois, 2007). This finding is consistent with previous research that has identified relational schemas as potentially important factors in the development and maintenance of depression (Ingram et al., 1998; Schmidt et al., 1999). Moreover, depressed individuals become more susceptible to sustaining additional episodes of depression as negative self-schemas become more tightly-interconnected (Segal, Williams, Teasdale, & Gemar, 1996).

Additionally, interpersonal stress and conditions strongly predict recurrence for individuals with past depression (Hammen, 2009; Gotlib & Hammen, 2014), whereas non-interpersonal stress is not associated with recurrence (Sheets & Craighead, 2014). It is possible that at-risk individuals who experience greater interpersonal stress will exhibit tighter negative structure and interpersonal content, therefore exposing them to a higher risk of relapse/recurrence. A diathesis-stress perspective posits that a cognitive vulnerability will interact with a stressor of a sufficient magnitude, which then potentiates the pathology in vulnerable individuals. It is possible that differing levels of schemas specific to interpersonal—or social—content may help explain the differential rates of relapse/recurrence among remitted and recovered individuals.

One's interactions and relationships with others greatly inform one's identity and sense of self, as humans are inherently social beings. Accordingly, interpersonal elements have been incorporated into models of depression (e.g., Joiner & Timmons, 2009), and

interpersonal risk factors for the development and maintenance of the disorder are also well-established in the research base (Gotlib, Lewinsohn, & Seeley, 1998; Hames, Hagan, & Joiner, 2013; Hammen, 1991). Beck (1983) originally declared the trait sociotropy, along with interpersonal stress, to be the greatest risk factors for depression, and Calvete (2011) found that sociotropy predicted the development of depressive symptoms in a sample of adolescents.

Depression is also associated with difficulties in multiple social realms, such as marriage (Gotlib, Lewinsohn, & Seeley, 1998; Gotlib & Whiffen, 1991), parenting (Gunlicks & Weissman, 2008; Wilson & Durbin, 2009;), and peer relationships (Joiner, 2000; Puig-Antich et al., 1985). Social and communication skills are also often impaired in depression (Segrin & Abramson, 1994), and expressions of depression may elicit negative attitudes and rejection behaviours in close others (see Hammen & Watkins, 2008, for a review). Additionally, rejection sensitivity, excessive-reassurance-seeking, and dependency are all stable traits and behaviours that are not necessarily confined to depressive episodes and can contribute to interpersonal stress and depression (Evraire & Dozois, 2011; Starr & Davila, 2008). University is also a time when many individuals are cultivating a new sense of personal identity, and establishing new social ties, and preoccupation with interpersonal concerns may be especially salient in this population.

While previous studies have investigated negative schema content as a marker for relapse/recurrence in previously-depressed individuals, there is a lack of investigation of schema structure in this context. There is also a need to study how both schema content and structure solidify over time. Most cognitive vulnerability research to date has either been conducted cross-sectionally, or has not formally involved a sample of individuals

with past depression (Scher, Ingram, & Segal, 2005). Examining maladaptive cognitions among remitted/recovered individuals in a longitudinal design allows inferences to be made about whether these cognitions precipitate depressive relapse/recurrence, approximately when in time they begin to appear or intensify, and how they may relate to organizational properties of schemas. Individuals with past depression are believed to be at risk for additional episodes in part because they have latent depressive schemas that are more readily-accessible during remission/recovery (Scher et al., 2005). A longitudinal design removes the need to create artificial stressors used in previous studies to activate the schemas (e.g., sad mood induction), as stressful conditions in the form of life stressors would be expected to naturally occur in a subset of the sample in the period of time under study. Additionally, examining how schemas consolidate over time in a remitted/recovered sample, and how these schemas relate to relapse/recurrence, could provide information on appropriate timing of interventions that target schema restructuring (Friedmann, Lumley, & Lerman, 2016).

Schema structure has demonstrated trait-like properties in previous research on the etiology of depression (Dozois, 2007; Dozois & Dobson, 2001b), and is a possible predictor of relapse/recurrence when studied longitudinally. It is also potentially informative to study this construct in conjunction with schema content in this context. Negative schema content—such as cognitive distortions, and attentional and memory biases—tend to resolve following remission of a depressive episode (Barnett & Gotlib, 1990; Clark et al., 1999), whereas negative schema structure tends to remain stable (Dozois, 2007; Dozois, Bieling, Patellis-Siotis, Hoar, & Chudzik, 2009; Dozois & Dobson, 2001a). Monitoring schema content changes over time may provide an

indication of relapse/recurrence risk, as negative content may re-consolidate due to cognitive reactivity, leading to an increase in depressive symptoms and potentially a contribution to relapse/recurrence. Remitted individuals tend to endorse more dysfunctional attitudes and display more information processing biases than do never-depressed samples, ostensibly when the latent depressive schema is activated in artificial conditions, such as an induced dysphoric mood state (e.g., Ingram et al., 1994; Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990). These results suggest that these depressive schemas remain accessible following remission/recovery, and that they constitute a vulnerability factor. It is therefore possible that these schemas will become activated when exposed to natural stressors that meet an appropriate threshold to induce a negative mood state. Additionally, tightly- interconnected negative schema structure (and loosely-interconnected positive structure) is predictive of depressive symptoms in individuals enduring life stress (Seeds & Dozois, 2010). Thus, monitoring stressful occurrences along with schema content and structure longitudinally could provide insight into relapse/recurrence risk. Monroe, Slavich, Torres, and Gotlib (2007) also reported that chronic stress correlates more significantly with repeated episodes of depression than it does initial onset. However, which particular life stress domains most influence depressive recurrence remains unclear (Sheets & Craighead, 2014). Assessing life stress over multiple time points would presumably contribute to addressing this limitation.

Summary

In sum, considerable evidence implicates negative schema structure and content—such as automatic thoughts, dysfunctional attitudes, and information-processing biases—

as precipitants to depressive episodes, both in initial onset and specifically in remitted individuals (see Scher et al., 2005, for a review). However, the majority of studies have only assessed the extent to which these constructs relate to the etiology of depression in the context of the initial episode. It is important to study the development and stability of schemas over time, in order to determine the extent of their predictive ability toward depressive symptomatology, and future depressive episodes. This study aims to address these gaps in the literature base, as well as provide supporting evidence regarding previous findings on cognitive vulnerability to depression.

The current study

The current study examined schema stability and the predictive validity of its content and structure over three separate time points (baseline, and three and six months) in an undergraduate sample composed of individuals with one or more past episodes of depression. At initial testing, participants were administered the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002), which has been rated by clinicians as the gold-standard diagnostic measure. The SCID-I was used to select a sample of previously-depressed individuals from the broader student population. Schema structure was assessed using the Psychological Distance Scaling Task (PDST; Dozois, 2002; Dozois & Dobson, 2001a, 2001b, 2003; Dozois & Frewen, 2006). Schema content was assessed with the Dysfunctional Attitudes Scale (DAS) and the Cognitive Distortions Scale (CDS; Covin, Dozois, Ogniewicz, & Seeds, 2011). Participants also completed the Beck Depression Inventory – 2nd Edition (BDI-II),

and the Negative Life Events Questionnaire (NLEQ) to examine potential mediators and moderators of relapse/recurrence.

Hypotheses

Previous research has identified negative schema structure as a vulnerability factor in the development of depression, and tightly-interconnected negative schema structure—as well as loosely interconnected positive structure—has also been shown to interact with life events to predict depressive symptoms (Seeds & Dozois, 2010). The main objective of the present study was to determine whether the interaction of negative schema content and structure would predict relapse/recurrence and/or severity of depressive symptoms over time in a sample of previously-depressed individuals. A secondary goal of the study was to examine the stability of schema structure over a six-month period in the same group.

In the context of the current study, more tightly-interconnected negative schema structure (i.e., interstimulus distance calculated from adjective placement on the PDST) at earlier time points was expected to predict relapse/recurrence and greater severity of depressive symptoms over and above schema content. Scales pertaining to interpersonal subject matter on the two schema content measures (CDS social and DAS dependency) were also hypothesized to be more predictive of relapse/recurrence, and associated with greater depressive severity (controlling for baseline depression), than those pertaining to achievement content (CDS achievement and DAS perfectionism). This group difference was predicted based on established interpersonal risk factors for the development of depression discussed above, as well as previously-mentioned research showing the

positive relationship between interpersonal stressors and relapse/recurrence, and the assumption that university students would be more susceptible to experiencing such stressors given that they are at a life stage when the formation of a social identity would be expected to be a primary concern. Specifically, individuals with higher scores on interpersonal schema content measures were expected to have both a higher rate of relapse/recurrence, and a greater severity of depressive symptomatology, relative to individuals with lower scores on these measures. In line with previous research, individuals who did not show clinically-significant depressive symptoms at follow-up were nevertheless expected to continue to exhibit tighter negative schema organization (but not content), but to a lesser extent than those individuals who met relapse/recurrence criteria.

Method

Participants

Three-hundred-ninety-one Western University students were recruited to take part in the pre-screen process. Participants were recruited through posters set up on Western University campus, and through postings made on Facebook groups containing Western University subject matter. From this initial population, 288 were deemed ineligible for the study (by meeting DSM-5 criteria for current depression, or for having a history of mania), leaving 103 eligible participants. Of these 103 eligible participants, 49 were withdrawn from the study over the six-month time period: 37 who failed to respond to three consecutive attempts to schedule an in-lab appointment, and 11 who voluntarily withdrew from the study for various reasons (e.g., changes to workload or living arrangements). At Time 1, the sample consisted of 66 individuals ranging in age from 18-

36 ($M = 21.24$, $SD = 3.95$), and included 17 males and 49 females. At Time 2, the sample included 53 participants ranging in age from 18-36 ($M = 21.55$, $SD = 4.25$), including 14 males and 39 females. And at Time 3, there were 40 participants ranging in age from 18-36 ($M = 21.88$, $SD = 4.64$), including 12 males and 28 females.

Materials

The following measures were administered at each of Time 1, Time 2, and Time 3. All questionnaires/tasks, except for the SCID-I, were randomized and completed electronically.

Diagnostic Screening

Structured Clinical Interview for DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 2002. Mood disorders module

The SCID-I has been rated by clinicians as the gold-standard diagnostic measure of nosology. The SCID-I is a semi-structured interview for making diagnoses according to disorder-specific criteria outlined in the *Diagnostic and Statistical Manual of Mental Disorders – 4th Edition* (DSM-IV; APA, 2000). The DSM is the standard manual for classification of mental disorders used by clinicians and researchers. The SCID-I has excellent reliability for diagnosing Major Depressive Disorder specific to DSM-IV criteria ($\kappa = .80$; Zanarini et al., 2000). The SCID-I mood disorders module was used to screen participants for current depression at Time 1, and at Time 2 and 3 for relapse/recurrence. The measure was administered to participants privately and individually by one of four trained clinical psychology graduate students.

Schema structure

Psychological Distance Scaling Task (PDST; Dozois & Dobson, 2001b)

The PDST is a computer-based task that requires participants to place adjectives on a 21.5 cm by 23 cm rectangular grid in order to rate each adjective by degree of self-reference and valence. The left side of the grid's x-axis is labeled with the statement "not at all like me," and the right side of the axis is labeled with the statement "very much like me." The bottom of the y-axis is labeled with the statement "very negative," and the statement "very positive" appears at the top of the axis. Participants were asked to rate 60 target adjectives, which made up four categories comprising 15 adjectives each: sociotropic positive (e.g., "encouraged," "generous"), sociotropic negative (e.g., "alone," "rejected"), perfectionist positive (e.g., "ambitious," "intelligent"), and perfectionist negative (e.g., "defeated," "helpless"). To start the task, an adjective is placed at the centre of the grid. After each adjective placement, the participant is presented with a new grid and adjective until all 60 adjectives have been rated. For scoring, the computer records the x- and y-axis coordinate for each adjective. An idiographic formula is then used to calculate the average interstimulus distances for the self-referent positive and negative adjectives for each participant (see Dozois & Dobson, 2001b, for information on the development of the measure, and Seeds & Dozois, 2010, for a more detailed explanation of the formula). The assumption is that less distance among the adjectives indicates greater interconnectedness of the corresponding content, and greater distance indicates less interconnectedness of the corresponding content (Dozois & Frewen, 2006). The PDST has been used in a variety of studies assessing schema structure in depressive

and non-depressive samples (Dozois, 2007; Dozois & Dobson, 2001a, 2001b; Dozois & Dobson, 2003; Dozois & Frewen, 2006; Dozois, et al., 2014; Dozois, Eichstedt, Collins, Phoenix, & Harris, 2012; Lumley, Dozois, Hennig, & Marsh, 2012; Quilty, Dozois, Lobo, Ravindran, & Bagby, 2014).

Schema Content

Cognitive Distortions Scale (CDS; Covin, Dozois, Ogniewicz, & Seeds, 2011)

The CDS is a self-report measure that assesses the frequency with which individuals make a number of common cognitive distortions. The measure includes 10 cognitive distortions that relate to interpersonal and achievement schemas derived from Beck's cognitive model of depression. For each item, the CDS presents a brief description of the particular cognitive distortion, then asks participants to provide two responses: one for how often they engage in a given cognitive distortion in social situations (i.e., with friends, partner, or family), and one for achievement situations (i.e., school or work). The measure uses a 7-point Likert scale, ranging from 1 = never, to 7 = all the time, and items include: "Mind Reading" and "All-or-Nothing Thinking." The CDS has excellent overall reliability ($\alpha = .85$), as well as concurrent validity regarding stress and depression (Covin et al., 2011). Cronbach's alpha in this study was .90 at Time 1, .91 at Time 2, and .90 at Time 3.

Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978)

The DAS is a 40-item self-report measure developed to assess the degree to which respondents endorse dysfunctional attitudes toward themselves, others, and the world,

which are important components of Beck's cognitive model of depression. The DAS has demonstrated strong psychometric properties (e.g., Cane, Olinger, Gotlib, & Kuiper, 1986; Dobson & Shaw, 1986; Oliver and Baumgart 1985). The measure asks participants the extent to which they agree with a number of attitudes or beliefs (e.g., "It is difficult to be happy unless one is good looking, intelligent, rich, and creative," "If I fail at my work, then I am a failure as a person"), using a 7-point Likert scale, ranging from 1 = totally agree, to 7 = totally disagree. Cronbach's alpha in this study was .90 at Time 1, .92 at Time 2, and .92 at Time 3.

Life Stress

Negative Life Events Questionnaire (NLEQ; Metalsky & Joiner, 1992)

The NLEQ is a 70-item checklist measure that assesses life events in the interpersonal and achievement-related domains of functioning that are typically experienced by university-aged individuals. The measure has demonstrated strong validity in previous cognitive-vulnerability stress studies (e.g., Metalsky & Joiner, 1992). The NLEQ asks respondents to indicate the frequency that various events have occurred over the past 5 weeks (e.g., "Found out that a close family member has been criticizing you behind your back," "Did poorly on, or failed, an exam or major project in an important course"), using a 5-point scale ranging from A = never, to E = always.

Depressive symptomatology

Beck Depression Inventory – Second Edition (BDI-II; Beck, Steer, & Brown, 1996)

The BDI-II is a self-report measure that has shown both excellent sensitivity and specificity, and to be valid and reliable with regards to measuring the severity of

depressive symptoms (e.g., Arnau, Meagher, & Norris, 2001). The BDI-II includes 21 items that are responded to using a 4-point scale, ranging from 0 (in which a symptom is not at all present) to 3 (in which a symptom is severely present). Cronbach's alpha in this study was .90 at Time 1, .83 at Time 2, and .88 at Time 3.

Procedure

Upon receiving clearance from the research ethics board at Western University, recruitment materials were disseminated on Western University campus (see Appendix A), and on Facebook (see Appendix B). Interested participants were directed to an external website to complete the pre-screen measure for determining eligibility for the study. Participants were asked on the pre-screen if they had previously experienced an episode of depression (the study inclusion criterion), and were also screened for study exclusion criteria: current depression, a history of mania, and current mania. DSM-5 criteria were used throughout the pre-screen. The criteria for diagnosing Major Depressive Disorder did not change with the release of the DSM-5, and remains equivalent to that which appears in the mood disorders module of the SCID-I. Participants deemed eligible for the study were asked to provide consent to be contacted by a member of the research team to schedule an initial in-lab session, and were later contacted via their indicated preferred mode of contact (phone or email; see Appendices C and D for contact script). Additionally, all respondents to the pre-screen were presented with a list of community mental health resources upon completion of the measure.

At initial testing, participants were ushered into a private room by a research assistant, where they were tested individually and privately. After obtaining informed consent from participants (see Appendix E), the research assistant left the testing room, at

which time a trained clinical graduate student entered to administer the mood disorders module of the SCID-I to screen for current depression. Following this screening, participants were left alone to complete a series of computer-based questionnaires assessing schema structure and content, current depressive symptoms, and negative life events, as well as the PDST. Research assistants were instructed to monitor respondents' BDI-II scores while participants completed the study, and to inform the graduate student on duty if a respondent were to endorse a 2 or a 3 for item 9, which probes for suicidal ideation. The graduate student on duty would then conduct a risk assessment, and take further action to ensure the safety of the participant if it were deemed necessary. Upon completion of testing, participants were debriefed by a research assistant (see Appendix F for debriefing letter), and then given monetary compensation for their time (\$10-20 at Time 1, and \$20 for each of Time 2 and 3). Before leaving the testing area, participants were tentatively re-booked for a follow-up session exactly three months after their previous session.

For Time 2 data collection, the consent process from Time 1 was repeated, by first contacting participants via their preferred method of contact two weeks prior to their scheduled return date to ensure that this date remained feasible. All follow-up appointments were intended to be scheduled as close to three months after the previous assessment as possible. Eight participants withdrew themselves from the study prior to Time 2, citing changes to their workload or no longer residing in the city as reasons for withdrawal. All Time 1 procedures were repeated for Time 2, including re-booking participants for Time 2 testing three months after the second assessment. All procedures were repeated once more for Time 3 testing. Four participants withdrew from the study at

this stage, citing having moved away from the city for the summer as the reason for their withdrawal.

Results

Prediction of Relapse/Recurrence

Greater negative schema structure for interpersonal content was expected to predict relapse/recurrence and severity of depressive symptoms, over and above schema content. After controlling for baseline depression, higher scores on the schema content measures (CDS and DAS) were also hypothesized to predict relapse/recurrence and depressive severity. Lastly, tightly interconnected negative schema organization (but not content) will be evident even among individuals who do not relapse or show clinically-significant depressive symptoms at follow-up. The first series of analyses were conducted to ascertain whether self-schema structure and content is predictive of relapse/recurrence. A series of logistic regression analyses were conducted, with relapse/recurrence (based on SCID-I diagnoses) as the dependent variable. Gender differences in rate of relapse/recurrence were not tested, as the rate of relapse/recurrence was considered too small to draw any meaningful conclusions. The following Time 1 variables were entered into the first regression equation: PDST total scores for interpersonal negative adjectives, PDST total scores for interpersonal positive adjectives, and CDS social and DAS dependency (social) scale total scores. The second regression equation included PDST achievement negative and positive adjectives, and CDS and DAS achievement scale total scores. The same combination of variables was entered in the same order for the third and fourth regression equations (predicting Time 3 criteria) using Time 2 variables as

predictors. Results (presented in Table 1 and Table 2, for Time 1 and Time 2 variables respectively) revealed no significant effects. Considering only seven individuals (11% of the sample) relapsed/recurred, it is highly likely that there were simply not enough instances of clinical depression to detect meaningful differences between the two groups.

Prediction of Depressive Symptoms

The predictive utility of schema structure and content toward future depressive symptomatology was also of primary interest. To test this question, the Analysis of Partial Variance (APV) procedure was used. APV is a set-wise hierarchical regression procedure that allows for the “study of partial (residualized) variance that may use any type(s) of research factors as covariates and any type(s) of research factors whose covariate-adjusted effects are of interest” (Cohen & Cohen, 1983, p. 426). APV can be used to calculate change scores, when the dependent variable(s) is a post-score and the pre-score measure(s) is entered as a covariate (see Metalsky & Joiner, 1992), for the purpose of examining relationships with other factors (Cohen & Cohen, 1983). When conducting set-wise hierarchical regression analyses, Cohen and Cohen (1983) recommended entering the covariate(s) first, followed by sets of independent variables. In the present study, the first two hierarchical regressions examined the prediction of Time 2 depressive symptoms using Time 1 predictors. On the first step, Time 1 BDI-II total scores were entered as the covariate, for the purpose of controlling for baseline depressive symptoms. On the second step, a pairing of CDS and DAS total scores was entered [either CDS social and DAS dependency (social content) scales together, or CDS achievement and DAS perfectionist (achievement content) scales together], since we

Table 1

Logistic Regression: Predictors of Relapse/Recurrence at Time 2

| Predictor | B | S.E. | Wald | df | Sig. | Exp(B) |
|-----------|-------|------|-------|----|------|--------|
| PDST-IN | -.395 | .480 | .677 | 1 | .411 | .674 |
| PDST-IP | -.114 | .455 | .062 | 1 | .803 | .893 |
| CDS-S | -.012 | .050 | .055 | 1 | .814 | .988 |
| DAS-S | .413 | .473 | .761 | 1 | .383 | 1.511 |
| PDST-AN | .154 | .538 | .082 | 1 | .775 | 1.167 |
| PDST-AP | .461 | .401 | 1.328 | 1 | .249 | 1.586 |
| CDS-A | .000 | .056 | .000 | 1 | .999 | 1.000 |
| DAS-A | .263 | .591 | .198 | 1 | .656 | 1.301 |

Note:

BDI-II = Beck Depression Inventory II

CDS = Cognitive Distortions Scale

DAS = Dysfunctional Attitudes Scale

PDST = Psychological Distance Scaling Task

S = Social

A = Achievement

IN = Interpersonal Negative

IP = Interpersonal Positive

AN = Achievement Negative

AP = Achievement Positive

Hosmer and Lemeshow Test for interpersonal variables: $\chi^2 = 9.667$, $df = 8$, sig. = .289Hosmer and Lemeshow Test for achievement variables: $\chi^2 = 6.718$, $df = 7$, sig. = .459

Table 2

Logistic Regression: Predictors of Relapse/Recurrence at Time 3

| Predictor | B | S.E. | Wald | df | Sig. | Exp(B) |
|-----------|--------|-------|-------|----|------|--------|
| PDST-IN | -1.479 | 1.403 | 1.111 | 1 | .292 | .220 |
| PDST-IP | 1.512 | .211 | .513 | 1 | .474 | 4.535 |
| CDS-S | -.066 | .060 | 1.214 | 1 | .271 | .936 |
| DAS-S | .440 | .453 | .941 | 1 | .332 | 1.552 |
| PDST-AN | .326 | .526 | .385 | 1 | .535 | 1.386 |
| PDST-AP | -.489 | .721 | .459 | 1 | .498 | .613 |
| CDS-A | -.028 | .060 | 2.276 | 1 | .634 | .972 |
| DAS-A | -.027 | .642 | .002 | 1 | .966 | .973 |

Note:

BDI-II = Beck Depression Inventory II

CDS = Cognitive Distortions Scale

DAS = Dysfunctional Attitudes Scale

PDST = Psychological Distance Scaling Task

S = Social

A = Achievement

IN = Interpersonal Negative

IP = Interpersonal Positive

AN = Achievement Negative

AP = Achievement Positive

Hosmer and Lemeshow Test for interpersonal variables: $\chi^2 = 12.975$, $df = 7$, sig. = .073,

Hosmer and Lemeshow Test for achievement variables: $\chi^2 = 14.543$, $df = 8$, sig. = .069

sought to test the effects of interpersonal and achievement schemas separately). And lastly, on the third step, a cognitive organization predictor was entered (i.e., either PDST interpersonal negative total scores, or PDST achievement negative total scores). Time 2 BDI-II total scores was the dependent variable.

The next two regression analyses examined the prediction of Time 3 depressive symptomatology, first using Time 1 variables, and then Time 2 variables. For the first analysis, the same covariate and predictors from the regression above were added in identical order, and Time 3 BDI-II total scores were used as the dependent variable. For the next regression, Time 2 BDI-II total scores were used as the covariate, Time 2 predictors were substituted for their Time 1 counterparts, and Time 3 BDI-II total scores were used as the dependent variable.

Results of the general models pertaining to the prediction of Time 2 BDI-II scores (Table 3) revealed a significant main effect for both the CDS social scale ($\beta = .332$, $t = 2.11$, $p = .041$), and the PDST domain of interpersonal negative ($\beta = -.411$, $t = -3.20$, $p = .003$), independent of the other predictors in the equation. The regression for the achievement-related predictors revealed solely a marginally-significant main effect for PDST achievement negative ($\beta = -.34$, $t = -1.86$, $p = .070$; also see Table 3).

The regression analyses examining Time 3 depressive symptoms revealed no significant effects (Tables 4 and 5).

Stability of Self-Schema Structure

Bivariate correlations were performed to assess the stability of schema structure over a six-month time period. Table 6 shows the correlations of interpersonal and achievement-related content—categorized by valence—at Time 2 and Time 3. Schema

Table 3

Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 2

| Predictor | B | S.E. | t | Sig. | F | Sig. | Adjusted R Squared |
|------------------------------------|--------|-------|--------|------|--------------|-------------|--------------------------|
| Interpersonal Variables | | | | | | | |
| Step 1 | | | | | 3.941 | .053 | .057 |
| BDI-II | .215 | .108 | 1.985 | .053 | | | |
| Step 2 | | | | | 3.267 | .030 | .122 |
| CDS-S | .242 | .111 | 2.187 | .034 | | | |
| DAS-S | -.128 | 1.050 | -.122 | .904 | | | |
| Step 3 | | | | | 4.362 | .003 | .255 |
| PDST-IN | -3.033 | .947 | -3.201 | .003 | | | |
| PDST-IP | -.700 | .895 | -.783 | .438 | | | |
| Achievement Variables | | | | | | | |
| Step 1 | | | | | 2.358 | .132 | .031 |
| BDI-II | .182 | .118 | 1.536 | .132 | | | |
| Step 2 | | | | | 1.889 | .147 | .058 |
| CDS-A | .159 | .124 | 1.278 | .208 | | | |
| DAS-A | -.644 | 1.231 | -.523 | .604 | | | |

| | | | | | | | |
|---------------|--------|-------|--------|------|--------------|-------------|-------------|
| Step 3 | | | | | 1.878 | .121 | .093 |
| PDST-AN | -2.381 | 1.279 | -1.862 | .070 | | | |
| PDST-AP | -.493 | .997 | -.495 | .624 | | | |

Note:

BDI-II = Beck Depression Inventory II

CDS = Cognitive Distortions Scale

DAS = Dysfunctional Attitudes Scale

PDST = Psychological Distance Scaling Task

S = Social

A = Achievement

IN = Interpersonal Negative

IP = Interpersonal Positive

AN = Achievement Negative

AP = Achievement Positive

Table 4

Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 3 (Time 1 Predictors)

| Predictor | B | S.E. | t | Sig. | F | Sig. | Adjusted R Squared |
|--------------------------------|--------|-------|-------|------|-------------|-------------|--------------------|
| Interpersonal Variables | | | | | | | |
| Step 1 | | | | | .182 | .672 | -.023 |
| BDI-II | .065 | .152 | .427 | .672 | | | |
| Step 2 | | | | | .625 | .604 | -.032 |
| CDS-S | .180 | .181 | .994 | .327 | | | |
| DAS-S | -1.059 | 1.622 | -.653 | .518 | | | |
| Step 3 | | | | | .582 | .714 | -.062 |
| PDST-IN | -1.753 | 1.844 | -.951 | .349 | | | |
| PDST-IP | -.012 | 1.707 | -.007 | .995 | | | |
| Achievement Variables | | | | | | | |
| Step 1 | | | | | .040 | .842 | -.035 |
| BDI-II | -.035 | .174 | -.201 | .842 | | | |
| Step 2 | | | | | .458 | .714 | -.062 |
| CDS-A | -.252 | .226 | -.262 | .274 | | | |
| DAS-A | -1.864 | 2.247 | -.830 | .415 | | | |

| | | | | | | | |
|---------------|--------|-------|-------|------|-------------|-------------|-------------|
| Step 3 | | | | | .448 | .810 | -109 |
| PDST-AN | -1.721 | 2.060 | -.835 | .412 | | | |
| PDST-AP | .319 | 2.483 | .128 | .899 | | | |

Note:

BDI-II = Beck Depression Inventory II

CDS = Cognitive Distortions Scale

DAS = Dysfunctional Attitudes Scale

PDST = Psychological Distance Scaling Task

S = Social

A = Achievement

IN = Interpersonal Negative

IP = Interpersonal Positive

AN = Achievement Negative

AP = Achievement Positive

Table 5

Set-Wise Hierarchical Regression: Predictors of Depressive Symptom Severity at Time 3 (Time 2 Predictors)

| Predictor | B | S.E. | t | Sig. | F | Sig. | Adjusted R Squared |
|--------------------------------|-------|-------|-------|------|---------------|-------------|--------------------|
| Interpersonal Variables | | | | | | | |
| Step 1 | | | | | 11.796 | .002 | .241 |
| BDI-II | .621 | .181 | 3.435 | .002 | | | |
| Step 2 | | | | | 4.074 | .015 | .213 |
| CDS-S | -.124 | .142 | -.875 | .388 | | | |
| DAS-S | .292 | 1.277 | .229 | .820 | | | |
| Step 3 | | | | | 2.437 | .058 | .174 |
| PDST-IN | .896 | 1.445 | .620 | .540 | | | |
| PDST-IP | .627 | 1.424 | .441 | .663 | | | |
| Achievement Variables | | | | | | | |
| Step 1 | | | | | 5.595 | .025 | .141 |
| BDI-II | .520 | .220 | 2.365 | .025 | | | |
| Step 2 | | | | | 1.930 | .150 | .091 |
| CDS-A | -.122 | .183 | -.670 | .509 | | | |
| DAS-A | -.926 | 1.949 | -.475 | .639 | | | |

| | | | | | | | |
|---------------|-------|-------|-------|------|--------------|-------------|-------------|
| Step 3 | | | | | 1.121 | .377 | .021 |
| PDST-AN | -.824 | 1.905 | -.433 | .669 | | | |
| PDST-AP | -.725 | 2.238 | -.324 | .749 | | | |

Note:

BDI-II = Beck Depression Inventory II

CDS = Cognitive Distortions Scale

DAS = Dysfunctional Attitudes Scale

PDST = Psychological Distance Scaling Task

S = Social

A = Achievement

IN = Interpersonal Negative

IP = Interpersonal Positive

AN = Achievement Negative

AP = Achievement Positive

Table 6

Self-Schema Stability Over Time

| Variable | Time 2 | Time 3 |
|---------------------------|--------|--------|
| Interpersonal Negative | .350* | .236 |
| Interpersonal Positive | .510** | .494** |
| Achievement Negative | .527** | .202 |
| Achievement Positive | .590** | .722** |

Note: **Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

stability correlations between the same aspect of cognitive organization (e.g., achievement negative at Time x and achievement negative at Time x) that were significant at least at the .05 probability level were as follows, for Time 1 and Time 2: interpersonal negative, $r = .35$; achievement negative, $r = .53$; interpersonal positive, $r = .51$; achievement positive, $r = .59$.

For Time 2 and Time 3, stability correlations for these constructs were as follows (all significant at the .01 probability level): interpersonal negative, $r = .51$; achievement negative, $r = .57$; interpersonal positive, $r = .69$; achievement positive, $r = .74$.

Stability correlations for Time 1 and 3, which was the full length of the study, were as follows: interpersonal negative, $r = .24$ (*ns*); achievement negative, $r = .20$ (*ns*); interpersonal positive, $r = .49$ ($p = .01$); achievement positive, $r = .72$ ($p = .01$).

Discussion

The main objective of this study was to investigate the predictive validity of cognitive organization and the negative cognitive products of these self-structures (i.e., dysfunctional attitudes and cognitive distortions), as pertains to both relapse/recurrence of major depressive episodes and future depressive symptomatology. Cognitive products refer to deliberate negative thoughts and appraisals, as opposed to automatic processing, that occur as a result of schema activation (Clark et al., 1999). The secondary goal of the study was to examine the stability of cognitive organization over a six-month time period in a previously-depressed sample. The first hypothesis will be addressed somewhat briefly, as sampling limitations presented an obstacle to obtaining sufficiently

interpretable results for relapse/recurrence, and will be followed by a more involved discussion of the results of the latter two predictions.

Diathesis-stress models of depression posit that depressogenic schemas are a stable risk factor that remain latent until activated by stressful life events (Ingram, Miranda, & Segal, 2006), and that individuals with a history of depression are especially sensitive to negative events that may trigger these dormant self-structures (Clark et al., 1999; Dozois & Dobson, 2008). A history of multiple episodes is theorized to lower the activation threshold of depressive schemas. This process renders remitted/recovered individuals particularly vulnerable to re-experiencing maladaptive thinking patterns, and can potentially trigger a new depressive episode (Clark et al., 1999; Lau, Segal, & Williams, 2004) provided the correct conditions for schema activation are present (Segal et al., 2006). It was predicted that individuals in the study who relapsed/recurred would exhibit increased dysfunctional attitudes and cognitive distortions in the interpersonal domain, as well as more interconnected cognitive organization for interpersonal content as the study progressed and their latent cognitive vulnerability was ostensibly re-activated by the occurrence of stressful life events. This prediction was informed by the diathesis-stress conceptualization, as well as existing research suggesting that previously-depressed individuals are more vulnerable to cognitive reactivity (Clark et al., 1999; Segal et al., 2006; Lau et al., 2004). Recall that cognitive reactivity occurs when dormant depressive schemas are activated, and involves an individual's tendency to perceive and interpret information and events in a negatively-biased manner, rendering non-pathological sad mood states clinically-significant depressive episodes (Ingram et al., 1998). Furthermore, some cognitive theories for vulnerability to depression in previously-depressed

individuals have proposed a lingering type of cognitive reactivity that occurs when specific environmental conditions are present, such as a stressor of a significant magnitude (Segal et al., 2006).

However, it was unanticipated that only seven individuals in the sample (11.1%) would meet diagnostic criteria for relapse/recurrent of a major depressive episode during the period they were under observation. This percentage of relapse/recurrence falls far below what would be expected given the high rates of relapse/recurrence characteristic of the disorder. Previous research, for example, has demonstrated that between one-half and two-thirds of previously-depressed individuals experience a relapse/recurrence in any given year (Kessler & Wang, 2008). Therefore, it is reasonable to conclude that the small sample size rendered the study underpowered to accumulate a sufficient number of episodes to properly and thoroughly investigate our hypotheses regarding depressive relapse/recurrence. It is possible that the individuals in the sample did not experience the requisite conditions that would activate their respective cognitive diatheses during the six-month study period. For instance, perhaps this group did not experience a sufficiently impactful stressful life event—or combination of events—that met the threshold needed to actuate latent depressive schemas and set in motion the cascade of negative processes that could place them on the precipice of relapse/recurrence.

Interconnectedness of negative self-structures, specifically in the interpersonal domain, as well as cognitive distortions and dysfunctional attitudes pertaining to interpersonal content, were expected to be predictive of a) depressive relapse/recurrence and b) future depressive symptomatology. The first hypothesis was not supported. However, the second prediction was partially supported, in that the CDS social scale and

PDST scores in the interpersonal negative domain at Time 1 predicted depressive symptoms at Time 2. It was also hypothesized that negative self-structures would display moderate to strong stability in the sample throughout the course of the study. This last prediction was informed by research showing that cognitive by-products of depressive schema structures consistently improve with resolution of depressive symptoms (Clark et al., 1999; Dozois & Dobson, 2008; Seeds & Dozois, 2010), whereas schematic structure tends to remain stable both during depressive episodes and following remission (Dozois, 2007; Dozois & Dobson, 2001a; Dozois & Seeds, 2010). This enduring pattern of cognitive organization has been especially observable for negative interpersonal content, and has been suggestive of a trait-like characteristic for vulnerability to depression (Dozois, 2007; Dozois & Dobson, 2008; Seeds & Dozois, 2010). These findings reflect one of the main principles of Beck's theory of depression that negative schemas are present, yet dormant, in vulnerable individuals, and that when activated they can produce the depressogenic thinking patterns that are characteristic of negative mood states (Clark et al., 1999; Ingram et al., 2006).

Thus, in the current study, it was surprising that negative cognitive organization—particularly for interpersonal content—failed to maintain stability throughout the course of the study. Although negative interpersonal content did display some stability at Time 2 relative to Time 1 (it dissipated slightly yet remained significantly correlated to the baseline assessment), the correlation trended notably downward at Time 3. Moreover, cognitive organization for negative achievement-related content followed a similar pattern. Conversely, positive self-structures displayed marked stability throughout the course of the study; most notably the achievement positive domain, which had a

correlation of .72 from Time 1 to Time 3. This finding again runs counter to previous research in which organization for positive interpersonal content was observed to be less stable over time than negative interpersonal content (Dozois, 2007), an effect that is even more pronounced in individuals with recurrent or more severe depression (Dozois, 2002; Dozois & Dobson, 2003; Quilty et al., 2014).

If these findings represent true effects, the data could indicate that participants' negative and/or positive self-structures were repairing over time. The mechanism of change literature for the treatment of depression may place the obtained results in context, as skills obtained during the course of past or concurrent therapeutic intervention could be utilized to manage negative mood states. Unfortunately, we did not probe participants for current or past treatment status/history during the course of the study, so we can only speculate on whether they had received intervention at some point in time. However, it is logical to assume treatment was received either concurrently during the course of the study, or previously during an episode, considering the current sample comprised previously-depressed individuals with a history of recurrent depression (mean previous episodes = 3.90, SD = 3.10). The recent literature base has accumulated moderate support for the notion that two of the most prevalent forms of treatment for depression—Cognitive Behavioural Therapy (CBT), and pharmacotherapy (PT)—can affect change at the deeper, structural level of cognition in currently and previously-depressed individuals (Dozois et al., 2009; Quilty et al., 2014). Additionally, skills gained in cognitive therapy are associated with less cognitive reactivity in previously-depressed individuals, which presumably lessens one's risk of relapse (Strunk, Adler, & Hollars,

2013). If participants in this study had undergone cognitive therapy, it is possible that this could have lowered vulnerability to relapse/recurrence.

CBT is a product of Beck's (1967; Beck, Rush, & Emery, 1979) model of depression, and the modality's framework is designed to reduce depressive symptoms by changing cognitions at the three hypothesized levels of processing, including the deeper level of cognitive organization (Garratt & Ingram, 2007; Hundt, Mignogna, Underhill, & Cully, 2013). Treatment is designed not only to enact acute symptom relief and bring about remission from a current episode of depression, but also to equip individuals with skills they can use to combat negative automatic thoughts, dysfunctional attitudes, and other cognitive processes that may leave them vulnerable to reactivation of the underlying depressive schemas and potentially future relapse/recurrence (Garratt & Ingram, 2007). CBT has been shown in numerous studies to have a lasting beneficial effect that reduces the risk of relapse/recurrence compared to pharmacotherapy (DeRubeis, Webb, Tang, & Beck, 2010; Hollon et al., 2005). Dozois et al. (2009) suggested that cognitive therapy (CT) may enact change on depressive symptoms partially by modifying negative cognitive organization, and/or by strengthening opposing positive structures. Barber & DeRubeis (1989) proposed that a potential mechanism by which CT exerts its effects on cognitive organization is through equipping individuals with tools termed metacognitive skills, which is the ability to challenge automatic thoughts and other negative cognitions produced by depressive schemas, and to replace them with more realistic alternatives.

With the objective of assessing changes in cognitive structure and processing, Quilty et al. (2014) randomly assigned individuals with major depression to receive

treatment with either CBT or pharmacotherapy. The authors found that CBT and PT led to relatively equal improvements in cognitive structure, with negative structures becoming more diffuse over the treatment period, and positive structures becoming more interconnected. Additional research examining combination CT + PT treatment versus PT alone found that negative schemas dissipated in both conditions during treatment, with an additional post-treatment effect on compensatory positive self-structures for the combination treatment condition (Dozois et al., 2014). This was in contrast with an earlier study (Dozois et al., 2009) that compared combination CT + PT treatment with PT alone, which found that the combination treatment enacted significant pre-post treatment change in both positive and negative self-structures, in addition to positive changes in cognitive content and depressive symptoms during treatment (whereas the PT alone condition only affected cognitive products during treatment and had no significant effect on cognitive organization). Quilty et al. (2014) proposed that the characteristics of the Dozois et al. (2009) study's sample—specifically the latter's composition of participants with greater mean severity of depressive symptoms—is a potential explanation for the relatively conflicting results between the two similar studies.

These results provide evidence that stable cognitive vulnerability factors for depression are amenable to change with treatment, and also suggest that CT has an effect over and above PT in altering deeper cognitive self-structures. Further research is needed to elucidate the mechanism of change further, but there is converging evidence to suggest that CT and PT produce beneficial change on cognitive organization in depressed individuals, and that CT or combination treatment exerts an additional post-treatment protective effect that reduces relapse/recurrence risk. Mechanisms of change in cognitive

therapy for depression is an area that is in need of more research attention in general (Dozois et al., 2009; Garratt & Ingram, 2007). While there is evidence for its effectiveness in symptom reduction, consistent with Beck's hypothesized mechanisms of action (see Hollon & Ponniah, 2010, for a review), the exact manner by which CBT is effective at bringing about remission is still uncertain (Hundt et al., 2013; Longmore & Worrell, 2007). Continued research is needed to determine how cognitive structure is affected by the use of CT skills, and how this in turn relates to relapse/recurrence risk (Hundt et al., 2013). Other mechanisms by which CBT may alter cognitive organization should also be clarified, such as deactivation of negative thinking (Dozois et al., 2009), and accommodation (using learned skills to change negatively-biased beliefs about the self, then eventually becoming free of these biases altogether; DeRubeis, Siegle, & Hollon, 2008; Hollon, Evans, & DeRubeis, 1990).

Related to the current study's second hypothesis, set-wise hierarchical regressions revealed that both negative interpersonal PDST scores at Time 1 and higher scores on the CDS social subscale at Time 1 were strongly predictive of depressive symptomatology at Time 2. This result was especially striking considering this effect was produced despite controlling for baseline depressive symptoms. However, interpersonal schema content did not hold significant predictive validity when predicting Time 3 depressive symptoms. Taking these results in conjunction with those regarding schema stability (negative schema structure was somewhat stable at Time 1, but grew more diffuse as the study progressed, whereas positive schema structure was tightly-interconnected throughout, and displayed a marked increase in stability at Time 3), and a pattern appears to emerge: negative interpersonal structure and content is more salient earlier in the study, but less so

as the study wears on. It is possible that the observed Time 1 and Time 2 results captured the fallout from previous depressive episodes, and the residual effects of these episodes manifested in more interconnected negative cognitive organization and greater accessibility of interpersonal cognitive distortions. The subsequent overall trend of self-structures shifting and cognitive distortions no longer predicting depressive symptoms could then reflect the receding influence of these previous episodes. The apparent simultaneous repair of self-structures potentially allowed the majority of participants to combat these cognitive distortions, which prevented them from slipping into a prolonged depressed mood state. If an individual's most recent episode occurred further back in his/her personal history as opposed to the recent past, or it was less severe, it is possible that cognitive reactivity would be lower, and therefore vulnerability to relapse/recurrence may be lower as well as cognitive structures would have had more time to repair.

Unfortunately, we did not record data as to how recently participants' previous episodes occurred, so we cannot provide statistical evidence that participants were influenced by recent previous episodes. But if this hypothesis is true, the set-wise regression results may constitute further evidence that self-structures were repairing over time in the sample. This inference does not preclude participants continuing to experience negative self-focused cognitions as the study advanced, but rather that they potentially replaced these cognitions with more adaptive—or positive—thoughts (Quilty et al., 2014). This process could potentially occur through the use of metacognitive skills (DeRubeis & Barber, 1989; also see Wells, 2009), or perhaps be facilitated by the mechanism by which pharmacotherapy may exert its alterative effects on cognitive self-structures (e.g., symptom suppression providing increased energy and motivation,

thereby allowing participants to better challenge depressogenic thinking patterns, or to have a less negatively-biased outlook). It is unclear whether changes in positive or negative self-structures would be most influential in producing the particular pattern of results observed in the current study regarding organization of the self-system. Quilty et al. (2014) speculated that changes in positive self-structures could correlate with consequential changes in other aspects of cognitive organization, such as negative views of the self. Overall, more investigation is needed to clarify the mechanism by which cognitive therapy alters deeper cognitive structures and exerts protective effects over relapse/recurrence, and how these effects manifest in structural changes within the self-system.

Diathesis-stress congruence is another concept to consider when interpreting the lack of significant findings in the current study. This theory postulates that individuals who experience stressors that match their diathesis should be likely to develop clinical depression, whereas those who experience stressors that are incongruent with their diathesis should be no more likely than non-vulnerable individuals to experience depression (Alloy, Abramson, & Hogan, 1997). Segal, Shaw, Vella, & Katz (1992) found that individuals with self-critical traits incurred relapse at a higher rate after experiencing stressful life events categorized in the achievement domain than they did after experiencing events categorized in the interpersonal domain. These effects held regardless of whether stress was measured in terms of number of events, or degree of stressfulness experienced. In the current study, it is possible that the life events experienced by the majority of individuals during the period they were under observation were not congruent with the content of the schemas that confer their idiosyncratic

vulnerability to depression. Time 1 interpersonal cognitive distortions and Time 1 PDST scores in the interpersonal negative domain were found to be significant predictors of depressive symptoms at Time 2. Following a diathesis-stress congruence framework, it is logical to conclude that these individuals would be more susceptible to having latent depressive schemas reactivated through exposure to stressors in the interpersonal domain (such as relationship upheaval or rejection experiences). However, when interpersonal stressors—as measured by the NLEQ—were added to the regression equation, this variable failed to significantly add to the prediction of depressive symptoms at Time 2. Perhaps these individuals did not experience interpersonal stressors during the period under observation that were of sufficient magnitude to activate the schema, or the NLEQ was not sensitive enough to detect these events.

Future studies could continue to stratify individuals by vulnerability in domains such as interpersonal and achievement-related concerns, or parse these categories further, and then measure life events experienced in those areas during the time under assessment. Matching participants both on specific content or traits that may relate to their individual cognitive vulnerabilities, and the type of life event thought to function as an activating stressor, could provide a more fine-grained model that would be more predictive of depressive symptoms and/or relapse/recurrence. Additionally, a semi-structured, contextual measure of stress such as the Life Events and Difficulties Schedule (LEDS-II; Bifulco et al., 1989) would allow for more objective measurement of a broader range of life stress domains. Such a measure was not feasible for the current study, due to the time and rater training commitments required to employ it. However, it is possible that using such a measure would detect the presence of significant and depressogenic stressors that

were unaccounted for by a self-report measure such as the NLEQ (see Harkness, 2008; Rnic, Dozois, & Machado, N.d.)

Similarly, the continued development and use of idiographic measures for assessing self-schema content and structure has the potential to advance the investigative literature for relapse/recurrence. From a diathesis-stress perspective, and as discussed above, stressful life events that are congruent with one's underlying personality traits may be a vulnerability factor for depressive relapse/recurrence (Alloy et al., 1997; Ingram et al., 1998). Thus, utilizing assessment tools and methods that are more capable of personalizing the core schema constructs being investigated may be a worthwhile pursuit. For example, Solomon, Arnow, Gotlib, & Wind (2003) used an individualized assessment approach to demonstrate that previously-depressed individuals possessed strong irrational beliefs relative to a non-depressed control group. One of the issues that has plagued vulnerability to depression research is the difficulty finding consistent differences between previously-depressed and never-depressed individuals on cognitive measures in the absence of a cognitive prime or a sad mood induction (Clark et al., 1999; Miranda & Persons, 1988; Solomon et al., 1999). Using more sensitive and personalized assessment tools to conduct a more fine-grained investigation of the schema construct may allow researchers to identify reliable differences between remitted/recovered and never-depressed individuals out of episode that constitute stable vulnerability factors for depression as outlined in Beck's cognitive model of depression. Additionally, looking beyond overall positive and negative self-referent measures and further incorporating personality modes into the study of the relationship between cognitive organization and vulnerability to depression is another possible extension (Dozois, 2007).

As for the individuals in the current study who did relapse/recur during the study, or who will go on to do so at some point following completion of the study, it can be assumed that this group ultimately suffered from an inability to manage their negative mood states as well as their counterparts who maintained recovery. This is consistent with Beck's theory of depression, which posits that one is susceptible to depression when environmental stressors activate latent cognitive vulnerabilities. These stressors give rise to a number of negatively-biased processes, which result in dysphoria. Some individuals are able to self-correct (such as the group who remained remitted/recovered), whereas for others the maladaptive thoughts perpetuate, essentially trapping them in a "feedback loop" of negatively-biased thinking patterns and depressed mood state, as they are left without the cognitive resources required to break the cycle (Beevers & Carver, 2003; Beevers, 2005; Haefel, Abramson, Voelz, Metalsky, & Halberstadt, 2005).

In order to increase accurate differentiation between these two groups, future research should continue to investigate multiple predictors of relapse/recurrence risk, along with how changes in cognitive organization over time relates to future depressive episodes and symptoms. Much of past research in remitted/recovered individuals has used mood induction procedures to reveal latent negative cognitive structures and their by-products, and to show the endurance of these constructs past an acute episode. Future research should investigate other mechanisms by which dysfunctional depressogenic schemas may be activated in remitted/recovered samples, such as recently-occurring negative life events, or cognitive priming tasks. The exact process by which latent cognitive structures may influence vulnerability in remitted/recovered individuals remains an area requiring clarification, and the onus is on researchers interested in

identifying better predictive models of relapse/recurrence to investigate the circumstances in which underlying schemas can be primed or activated. One such area for further exploration is the conditions under which vulnerability may increase alongside the number of depressive episodes incurred. For instance, how exactly does the threshold for negative schema activation lower, or the range of stimuli that activate the underlying schemas broaden, as one accumulates a history of episodes? Teasdale's (1988) differential activation hypothesis asserts that depressive schemas and the associated negative thinking patterns are more readily triggered by sad mood states in remitted/recovered individuals than they are in those with no history of depression. According to Teasdale's (1988) hypothesis, this increased vulnerability in previously-depressed individuals is the product of these negative self-referential constructs being so strongly associated with dysphoric mood from previous episodes of depression. In contrast, cognitive models of depression attribute the heightened vulnerability observed in this population to greater cognitive reactivity to stressful life experiences that are similar semantically to underlying diatheses (Clark et al., 1999). Other theorists have proposed that rumination is the process that heightens vulnerability to a future episode (Nolen-Hoeksema & Wisco, 2008), and life stress has also been implicated (Monroe & Harkness, 2011).

Future research could also assess cognitive organization in previously-depressed samples who possess characteristics that are believed to place them at a high risk of relapse/recurrence. Examples include individuals with a greater number of previous episodes and/or a history of severe depression, or those who possess a combination of factors that have been associated with relapse/recurrence risk (e.g., early age of onset of

the index episode, greater severity of previous episodes, childhood maltreatment, etc.). Alloy, Abramson, Hogan, Whitehouse, & Rose (2000) retrospectively assessed lifetime vulnerability in a study in which participants were categorized as either high or low risk for major depression according to their level of depressogenic cognitive styles. Those individuals deemed to be high risk had a significantly higher incidence rate of depression than those placed in the low risk group. Furthermore, participants in the high risk group who had a history of depression were more likely to experience recurrence than those given the low risk label. A follow-up study using the same data discovered that rumination mediated the effects of this negative cognitive style on future depression (Spasojevic & Alloy, 2001).

Additional research has explored other negative cognitive styles and traits such as perfectionism, dysfunctional attitudes, and negative attributions as vulnerability factors for first onset (e.g., Brown, Hammen, Craske, & Wickens, 1995; Haefel et al., 2005; Lewinsohn, Joiner, & Rohde, 2001; Mongrain & Blackburn, 2005). Mongrain and Blackburn (2005) also found that negative attributional style and autonomy predicted recurrence of depression. However, unclear results when investigating some cognitive constructs illustrate that more investigative focus is needed to determine whether variables that have been implicated in first onset also constitute vulnerability factors for future depressive episodes (Abela & Hankin, 2008; Baks-Dermott, Dobson, & Jones, 2010; Dozois & Beck, 2008). For instance, Alloy et al. (2000) discovered that sociotropy was associated with a greater number of previous episodes of depression. Mongrain and Blackburn (2005) suggested that sociotropy could represent a 'scar' of depression, and confer vulnerability to future episodes by leaving an individual more susceptible to

interpersonal stressors. There is supportive evidence for this hypothesis in the form of sociotropy interacting with other personality traits to predict recurrence (e.g., Mazure & Maciejewski, 2003; Morse & Robins, 2005), as well as unsupportive findings for the construct's role in repeat episodes of depression (Backs-Dermott et al., 2010). These mixed findings in regard to cognitive-personality variables could be attributed to the relative newness of the prediction literature in regards to relapse/recurrence; therefore, the role of personality in recurrent depression is an area that requires continued investigation (Mongrain & Leather, 2006). Namely, there is a need for the development of more complex models (Backs-Dermott et al., 2010; Dozois & Beck, 2008) that better integrate a range of constructs toward the prediction of relapse/recurrence.

The current study had several strengths, the first being its longitudinal design, which consisted of three evenly-spaced assessments. Participants were followed quite closely, at three months apart, to ensure that relapse/recurrence would be captured relatively quickly if it did occur. The screening criteria was rigorous; participants completed a pre-screen measure probing for current depression and past or current mania (according to DSM-5 diagnostic criteria), and were again screened in-person for current depression prior to baseline testing. Moreover, the in-person diagnostic interviews were conducted by trained graduate-level clinical psychology students, using the SCID-I (which is widely considered the gold-standard diagnostic screening tool; Dozois & Dobson, 2010) mood disorders module. We also used the SCID-I as the criterion measure for relapse/recurrence, whereas some studies on the phenomenon have used BDI-II scores as an index. Self-report scales for depression can be unreliable when used for diagnosis, as their intended use is for determining severity of symptoms (Zimmerman & Coryell,

1987). Unlike self-report scales, clinical diagnosis (e.g., use of the SCID-I) only includes items that have been deemed relevant for diagnosis, and requires the clinician to make dichotomous decisions when determining the presence or absence of a given symptom(s) (Zimmerman & Coryell, 1987).

The small sample size of the study is a limitation. We were potentially underpowered to detect significant effects, and therefore the results should be interpreted with this information in mind. Clark et al. (1999) emphasized several methodological issues that researchers should be cognizant of when testing relapse/recurrence from a diathesis-stress perspective, when assessing how to improve the strength of a study from a sampling standpoint. Included among these recommendations were that samples should comprise the requisite number of individuals to provide adequate statistical power, and that all participants should be depression-free at baseline testing. In the current study, the process of ensuring the latter guideline came at the cost of the former. This is a limitation that will be remedied in the near future by recruiting additional participants, which will allow us to more thoroughly investigate the hypotheses and potentially reach more compelling conclusions regarding the relationship between schema structure and content, as well as future depressive episodes and symptomatology. Further to limitations, there was some variance in the time between participants' follow-up assessments. The procedure employed was to schedule a participant for an appointment exactly three months after the previous assessment, then to re-contact him/her to confirm the appointment two weeks prior to this pre-arranged date. The fluid nature of personal schedules meant that there was still some variance in the length of time between follow-ups, and therefore the assessment period was not quite equal across all participants.

This study adds to the burgeoning literature base on cognitive predictors of depressive relapse/recurrence, and provides a specific contribution to research on the relationship between self-schema structure and content and vulnerability to depression, as well as how these variables change over time. The current findings suggest that negative cognitive organization may not remain stable past an acute episode of major depression, and that negative self-structures and/or compensatory positive self-structures may repair themselves over time in a previously-depressed population. This result is contrary to previous evidence that negative cognitive organization—particularly for interpersonal content—remains stable into remission/recovery and therefore may be a trait-like characteristic for vulnerability to the disorder. The current study's findings also suggest that cognitive organization for negative interpersonal content, and cognitive distortions in the social domain, may be predictive of later depressive symptomatology. Gaining insight into specific schema content domains that may confer vulnerability to depression has the potential to shape therapeutic interventions, as well as inform an individual client's own strategy on how to approach and manage his or her disorder, and be aware of potential triggers for negative mood states. The most salient question at this time concerning the study at hand is whether the current findings can be replicated on a larger sample, which would also allow for better investigation of the hypotheses concerning relapse/recurrence and depressive symptoms. Ascertaining the predictors of relapse/recurrence has many important implications for depression prevention and treatment literature, given the high rate at which repeated episodes occur. Moreover, risk and vulnerability factors for index episodes versus additional episodes are not necessarily the same, and continuing to elucidate these differences is important. Depression continues to place a great strain on

society and burdens the lives of hundreds of millions of people yearly. Given the frequently-recurrent nature of the disorder, it is time for more research focus to be placed on depressive relapse/recurrence.

References

- Abela, J. R. Z. [Ed], & Hankin, B. L. [Ed]. (2008). *Handbook of Depression in Children and Adolescents*. New York, NY. Guilford Press.
- Abramson, L. Y., Alloy, L. B., & Hogan, M. E. (1997). Cognitive/personality subtypes of depression: Theories in search of disorders. *Cognitive Therapy and Research*, 21, 247–265.
- Abramson, L. Y., Alloy, L. B., & Metalsky, G. I. (1988). The cognitive diathesis-stress theories of depression: Toward an adequate evaluation of the theories' validities. In *Cognitive Processes in Depression* (pp. 3–30). Guilford Press, New York, NY.
- Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., ... Lapkin, J. B. (2000). The Temple-Wisconsin cognitive vulnerability to depression project: Lifetime history of axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*, 109, 403–418.
- An Open Pilot Study of Interpersonal Psychotherapy for Panic Disorder (IPT-PD)*. - *PsycINFO - ProQuest*. (n.d.). Retrieved from <http://search.proquest.com.proxy1.lib.uwo.ca/psycinfo/docview/621346640/484B0E57840046E9PQ/1?accountid=15115>
- American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Washington, DC: Author.
- Arnau, R. C., Meagher, M. W., Norris, M. P., & Bramson, R. (2001). Psychometric evaluation of the Beck Depression Inventory-II with primary care medical patients. *Health Psychology : Official Journal of the Division of Health Psychology*, 20, 112-

119.

- Backs-Dermott, B. J., Dobson, K. S., & Jones, S. L. (2010). An evaluation of an integrated model of relapse in depression. *Journal of Affective Disorders, 124*, 60–67.
- Barber, J. P., & DeRubeis, R. J. (1989). On second thought: Where the action is in cognitive therapy for depression. *Cognitive Therapy and Research, 13*, 441–457.
- Barkow, K. (2003). Risk factors for depression at 12-month follow-up in adult primary health care patients with major depression: an international prospective study. *Journal of Affective Disorders* 76, 157-169.
- Barnett, P. A., & Gotlib, I. H. (1990). Cognitive vulnerability to depressive symptoms among men and women. *Cognitive Therapy and Research, 14*(1), 47-61.
- Beck, A. (1971). Cognition, affect, and psychopathology. *Archives of General Psychiatry, 24*, 495–500.
- Beck, A., Steer, R., & Brown, G. (1996). *Beck Depression Inventory-II. San Antonio.*
- Beck, A. T. (1964). Thinking and depression: II. Theory and therapy. *Archives of General Psychiatry, 10*, 561–571.
- Beck, A. T. (1967a). *Depression. Depression*. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=psych&AN=1967-35022-000&site=ehost-live>
- Beck, A. T. (1967b). *Depression*. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true{&}db=psych{&}AN=1967-35022-000{&}site=ehost-live>
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. (Vol. 8).

[http://doi.org/10.1016/S0005-7894\(77\)80293-1](http://doi.org/10.1016/S0005-7894(77)80293-1)

Beck, A. T. (2005). The current state of cognitive therapy: a 40-year retrospective.

Archives of General Psychiatry, 62, 953–9.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for the Beck depression inventory-II. *San Antonio, TX: Psychological Corporation*, 1–82.

Beck, J. S. (2011). *Cognitive behavior therapy: Basics and beyond*. New York, NY: Guilford Press.

Beevers, C. G. (2005). Cognitive vulnerability to depression: A dual process model. *Clinical Psychology Review*, 25, 975-1002.

Beevers, C. G., & Carver, C. S. (2003). Attentional bias and mood persistence as prospective predictors of dysphoria. *Cognitive Therapy and Research*, 27, 619–637.

Bellón, J. Á., Moreno-Peral, P., Moreno-Küstner, B., Motrico, E., Aiarzagüena, J. M., Fernández, A., ... Amezcua, M. (2014). Patients' opinions about knowing their risk for depression and what to do about it. The predictD-qualitative study. *PLoS ONE*, 9.

Berlanga, C. (1999). A phenomenological evaluation of the depressive disorder. *SALUD MENTAL*, 22, 26–32.

Bernecker, S. L., Constantino, M. J., Atkinson, L. R., Bagby, R. M., Ravitz, P., & McBride, C. (2016). Attachment style as a moderating influence on the efficacy of cognitive-behavioral and interpersonal psychotherapy for depression: A Failure to replicate. *Psychotherapy*, 53, 22-33.

- Bifulco, A., Brown, G., Edwards, A., Harris, T., Neilson, E., Richards, C., & Robinson, R. (1989). *Life Events and Difficulties Schedule (LEDS-2): Vol. 1*. London, England: Royal Holloway and Bedford New College, University of London.
- Birmaher, B., Ryan, N. D., Williamson, D. E., Brent, D. A., Kaufman, J., Dahl, R. E., ... Nelson, B. (1996). Childhood and adolescent depression: a review of the past 10 years. Part I. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 1575-1583.
- Blackwell, E., & Conrod, P.J. (2003). A five dimensional measure of drinking motives. Unpublished manuscript, Department of Psychology, University of British Columbia, Vancouver, Canada.
- Bleiberg, K. L., & Markowitz, J. C. (2005). A pilot study of interpersonal psychotherapy for posttraumatic stress disorder. *The American Journal of Psychiatry*, 162, 181–3.
- Bockting, C. L. H., Spinhoven, P., Koeter, M. W. J., Wouters, L. F., & Schene, A. H. (2006). Prediction of recurrence in recurrent depression and the influence of consecutive episodes on vulnerability for depression: A 2-year prospective study. *Journal of Clinical Psychiatry*, 67, 747–755.
- Brown, G. P., Hammen, C. L., Craske, M. G., & Wickens, T. D. (1995). Dimensions of dysfunctional attitudes as vulnerabilities to depressive symptoms. *Journal of Abnormal Psychology*, 104, 431–435.
- Burcusa, S. L., & Iacono, W. G. (2007). Risk for recurrence in depression. *Clinical Psychology Review*, 27, 959-985.
- Calvete, E. (2011). Integrating sociotropy, negative inferences and social stressors as explanations for the development of depression in adolescence: Interactive and

- mediational mechanisms. *Cognitive Therapy and Research*, 35, 477–490.
- Calvete, E., & Cardeoso, O. (2005). Gender differences in cognitive vulnerability to depression and behavior problems in adolescents. *Journal of Abnormal Child Psychology*, 33, 179–192.
- Cane, D. B., Olinger, L. J., Gotlib, I. H., & Kuiper, N. a. (1986). Factor structure of the dysfunctional attitude scale in a student population. *Journal of Clinical Psychology*, 42, 307–309.
- Clark, D. A., Beck, A. T., & Alford, B. A. (1999). Scientific foundations of cognitive theory and therapy of depression. Philadelphia: Wiley.
- Cohen, J., & Cohen, P. (1983). Applied multiple regression/correlation for the behavioral sciences. *Hillsdale, NJ: Lawrence Earlbaum*.
- Covin, R., Dozois, D. J. A., Ogniewicz, A., & Seeds, P. M. (2011). Measuring cognitive errors: Initial development of the cognitive distortions scale (CDS). *International Journal of Cognitive Therapy*, 4, 297–322.
- Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: a meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology*, 76, 909–22.
- Daley, S. E., Hammen, C., & Rao, U. (2000). Predictors of first onset and recurrence of major depression in young women during the 5 years following high school graduation. *Journal of Abnormal Psychology*, 109, 525–533.
- DeLucia-Waack, J. L. [Ed], Gerrity, D. A. [Ed], Kalodner, C. R. [Ed], & Riva, M. T. [Ed]. (2004). Handbook of group counseling and psychotherapy. *Handbook of Group Counseling and Psychotherapy*. Retrieved from

<http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=psyc4&NEWS=N&AN=2004-21655-000>

- DeRubeis, R. J., Siegle, G. J., & Hollon, S. D. (2008). Cognitive therapy versus medication for depression: treatment outcomes and neural mechanisms. *Nature Reviews Neuroscience*, 9, 788–796.
- DeRubeis, R. J., Webb, C. a, Tang, T. Z., & Beck, A. T. (2010). Cognitive Therapy Overview. *Handbook of CognitiveBehavioral Therapies*, 1–27.
- Dobson, K. S., & Dozois, D. J. A. (2004). The prevention of anxiety and depression: Promises and prospects. In K. Dobson & D. Dozois (Eds.). (2004). *The prevention of anxiety and depression: Theory, research, and practice* (pp. 283–295). Washington, DC: American Psychological Association.
- Dobson, K. S., & Dozois, D. J. A. (Eds.). (2008). *Risk factors in depression*. San Diego, CA: Elsevier Academic Press.
- Dobson, K. S., & Shaw, B. F. (1986). Cognitive assessment with major depressive disorders. *Cognitive Therapy and Research*, 10, 13–29.
- Dozois, D., & Dobson, K. (2003). The structure of the self-schema in clinical depression: Differences related to episode recurrence. *Cognition & Emotion*, 17, 933–941.
- Dozois, D. J. a, Bieling, P. J., Patelis-Siotis, I., Hoar, L., Chudzik, S., McCabe, K., & Westra, H. a. (2009). Changes in self-schema structure in cognitive therapy for major depressive disorder: a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 77, 1078–1088.
- Dozois, D. J. A. (2002). Cognitive organization of self-schematic content in nondysphoric, mildly dysphoric, and moderately-severely dysphoric individuals.

Cognitive Therapy and Research, 26, 417–429.

Dozois, D. J. A. (2007). Stability of negative self-structures: A longitudinal comparison of depressed, remitted, and nonpsychiatric controls. *Journal of Clinical Psychology*, 63, 319–338.

Dozois, D. J. A., & Beck, A. T. (2008). Cognitive schemas, beliefs and assumptions. In K. Dobson & D. Dozois (Eds.). *Risk factors in depression* (pp. 119–143). San Diego, CA: Elsevier Academic Press.

Dozois, D. J. A., Bieling, P. J., Evraire, L. E., Patelis-Siotis, I., Hoar, L., Chudzik, S., & Westra, H. A. (2014). Changes in core beliefs (early maladaptive schemas) and self-representation in cognitive therapy and pharmacotherapy for depression. *International Journal of Cognitive Therapy*, 7, 217–234.

Dozois, D. J. A., & Dobson, K. S. (2010). Depression. In M. Antony & D. Barlow (Eds.). *Handbook of assessment and treatment planning for psychological disorders* (pp. 344–389). New York, NY: Guilford Press.

Dozois, D. J. A., Eichstedt, J. A., Collins, K. A., Phoenix, E., & Harris, K. (2012). Core beliefs, self-perception, and cognitive organization in depressed adolescents. *International Journal of Cognitive Therapy*, 5, 99–112.

Dozois, D. J. A., & Frewen, P. A. (2006). Specificity of cognitive structure in depression and social phobia: A comparison of interpersonal and achievement content. In *Journal of Affective Disorders*, 90, 101–109.

Dozois, D. J. A., Mikail, S. F., Alden, L. E., Bieling, P. J., Bourgon, G., Clark, D. A., ... Johnston, C. (2014). The CPA presidential task force on evidence-based practice of psychological treatments. *Canadian Psychology/Psychologie Canadienne*, 55, 153–

160.

- Dozois, D. J., & Dobson, K. S. (2001a). A longitudinal investigation of information processing and cognitive organization in clinical depression: stability of schematic interconnectedness. *Journal of Consulting and Clinical Psychology, 69*, 914–925.
- Dozois, D. J., & Dobson, K. S. (2001b). Information processing and cognitive organization in unipolar depression: specificity and comorbidity issues. *Journal of Abnormal Psychology, 110*, 236–246.
- Driessen, E., & Hollon, S. D. (2010). Cognitive behavioral therapy for mood disorders: Efficacy, moderators and mediators. *Psychiatric Clinics of North America, 33*, 537–555.
- Durbin, C. E., & Wilson, S. (2009). Assessment of mood disorders in children and adolescents. In J. Matson, F. Andrasik, & M. Matson (Eds.) *Assessing childhood psychopathology and developmental disabilities* (pp. 241–271). New York, NY: Springer Science + Business Media.
- Eberhart, N. K., Auerbach, R. P., Bigda-Peyton, J., & Abela, J. R. Z. (2011). Maladaptive schemas and depression: Tests of stress generation and diathesis-stress models. *Journal of Social and Clinical Psychology, 30*, 75–104.
- Eccleston, D., & Scott, J. (1991). Treatment, prediction of relapse and prognosis of chronic primary major depression. *International Clinical Psychopharmacology*. Retrieved from <http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=psyc3&NEWS=N&AN=1992-31394-001>
- Elkin, I., Shea, M. T., Watkins, J. T., Imber, S. D., Sotsky, S. M., Collins, J. F., ...

- Parloff, M. B. (1989). NIMH Treatment of depression collaborative research program: General effectiveness of treatments. *Archives of General Psychiatry*, 46, 971–982.
- Epp, A. M., Dobson, K. S., Dozois, D. J. A., & Frewen, P. A. (2012). A systematic meta-analysis of the Stroop task in depression. *Clinical Psychology Review*, 32, 316–328.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). Structured Clinical Interview for DSM-IV Axis I Disorders. *New York State Psychiatric Institute*. Retrieved from http://www.scid4.org/revisions/november_2001_02.htm
- Frank, E., Prien, R. F., Jarrett, R. B., Keller, M. B., Kupfer, D. J., Lavori, P. W., ... Weissman, M. M. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder: remission, recovery, relapse, and recurrence. *Archives of General Psychiatry*, 48, 851–855.
- Friedmann, J. S., Lumley, M. N., & Lerman, B. (2016). Cognitive schemas as longitudinal predictors of self-reported adolescent depressive symptoms and resilience. *Cognitive Behaviour Therapy*, 45, 32–48.
- Garratt, G., & Ingram, R. E. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology Science and Practice*, 14, 224–239.
- Giles, D. E., Jarrett, R. B., Biggs, M. M., Guzick, D. S., & Rush, A. J. (1989). Clinical predictors of recurrence in depression. *American Journal of Psychiatry*, 146(6), 764–767.
- Goldfried, M. R. (2013). What should we expect from psychotherapy? *Clinical Psychology Review*, 33, 862–869.

- Gonzales, L., Lewinsohn, P., & Clarke, G. (1985). Longitudinal follow-up of unipolar depressives: An investigation of predictors of relapse. *J Consult Clin Psychol*, 53, 461–469.
- Gotlib, I. H., Lewinsohn, P. M., & Seeley, J. R. (1998). Consequences of depression during adolescence: marital status and marital functioning in early adulthood. *Journal of Abnormal Psychology*, 107, 686–690.
- Gotlib, I. H., & MacLeod, C. (1997). Information processing in anxiety and depression: A cognitive-developmental perspective. *Attention, development, and psychopathology*. Retrieved from <http://ezproxy.library.wisc.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=psyh&AN=1997-09032-013&site=ehost-live>
- Gotlib, I. H., Whiffen, V. E., Wallace, P. M., & Mount, J. H. (1991). Prospective investigation of postpartum depression: factors involved in onset and recovery. *Journal of Abnormal Psychology*, 100, 122–132.
- Greenberg, P. E., Fournier, A.-A., Sisitsky, T., Pike, C. T., & Kessler, R. C. (2015). The economic burden of adults with major depressive disorder in the United States (2005 and 2010). *The Journal of Clinical Psychiatry*, 76, 155–162.
- Gunlicks, M. L., & Weissman, M. M. (2008). Change in child psychopathology with improvement in parental depression: a systematic review. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 379–389.
- Haefel, G. J., Abramson, L. Y., Voelz, Z. R., Metalsky, G. I., Halberstadt, L., Dykman, B. M., ... Alloy, L. B. (2003). Cognitive vulnerability to depression and lifetime history of Axis I psychopathology: A comparison of negative cognitive styles

(CSQ) and dysfunctional attitudes (DAS). *Journal of Cognitive Psychotherapy*, 17, 3–22.

Hames, J. L., Hagan, C. R., & Joiner, T. E. (2013). Interpersonal processes in depression. *Annual Review of Clinical Psychology*, 9, 355–77.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, 100, 555–561.

Hammen, C. (2009). Adolescent depression: Stressful interpersonal contexts and risk for recurrence. *Current Directions in Psychological Science*, 18, 200–204.

Hammen, C., & Watkins, E. (2008a). Depression (2nd ed.). Clinical psychology: A modular course. Retrieved from [https://www.lib.uwo.ca/cgi-bin/ezpauthn.cgi?url=http://search.proquest.com/docview/622091860?accountid=15115&\\$nhttp://sfx.scholarsportal.info/western?url_{_}ver=Z39.88-2004{&}rft_{_}val_{_}fmt=info:ofi/fmt:kev:mtx:book{&}genre=book{&}sid=ProQ:PsycINFO{&}ati](https://www.lib.uwo.ca/cgi-bin/ezpauthn.cgi?url=http://search.proquest.com/docview/622091860?accountid=15115&$nhttp://sfx.scholarsportal.info/western?url_{_}ver=Z39.88-2004{&}rft_{_}val_{_}fmt=info:ofi/fmt:kev:mtx:book{&}genre=book{&}sid=ProQ:PsycINFO{&}ati)

Hammen, C., & Watkins, E. (2008b). Depression (2nd ed.). Clinical psychology: A modular course. Retrieved from https://www.lib.uwo.ca/cgi-bin/ezpauthn.cgi?url=http://search.proquest.com/docview/622091860?accountid=15115\nhttp://sfx.scholarsportal.info/western?url_ver=Z39.88-2004&rft_val_fmt=info:ofi/fmt:kev:mtx:book&genre=book&sid=ProQ:PsycINFO&atitle=&title=Depre

Hammen, C., & Gotlib, I. Handbook of depression (3rd ed.) (2014). New York, NY: Guilford Press.

Hankin, B. L., Abramson, L. Y., Miller, N., & Haefel, G. J. (2004). Cognitive

- vulnerability-stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. *Cognitive Therapy and Research*, 28, 309–345.
- Hardeveld, F., Spijker, J., De Graaf, R., Nolen, W. A., & Beekman, A. T. F. (2010). Prevalence and predictors of recurrence of major depressive disorder in the adult population. *Acta Psychiatrica Scandinavica*, 147, 225-231.
- Harkness, K. L. (2008). Life events and hassles. In K. Dobson & D. Dozois (Eds.), *Risk factors in depression* (pp. 317-341). San Diego, CA: Elsevier Academic Press.
- Hollon, S. D., Evans, M. D., & DeRubeis, R. J. (1990). Cognitive mediation of relapse prevention following treatment for depression: Implications of differential risk. In R. E. Ingram (Ed.), *Contemporary psychological approaches to depression* (pp. 117–136). New York: Plenum.
- Hollon, S. D., & Ponniah, K. (2010). A review of empirically supported psychological therapies for mood disorders in adults. *Depression and Anxiety*, 27, 891-932.
- Hollon, S. D., Shelton, R. C., Wisniewski, S., Warden, D., Biggs, M. M., Friedman, E. S., ... Rush, A. J. (2006). Presenting characteristics of depressed outpatients as a function of recurrence: Preliminary findings from the STAR*D clinical trial. *Journal of Psychiatric Research*, 40, 59–69.
- Hollon, Steven D., Robert J. DeRubeis, Richard C. Shelton, Jay D. Amsterdam, Ronald M. Salomon, John P. O'Reardon, Margaret L. Lovett, et al. 2005. Prevention of relapse following cognitive therapy vs medications in moderate to severe depression. *Archives of General Psychiatry* 62, 417-422.
- Hooley, J. M., & Teasdale, J. D. (1989). Predictors of relapse in unipolar depressives:

- expressed emotion, marital distress, and perceived criticism. *Journal of Abnormal Psychology*, 98, 229–235.
- Hundt, N. E., Mignogna, J., Underhill, C., & Cully, J. A. (2013). The relationship between use of cbt skills and depression treatment outcome: A theoretical and methodological review of the literature. *Behavior Therapy*, 44, 12-26.
- Hunsley, J., Elliott, K., & Therrien, Z. (2014). The efficacy and effectiveness of psychological treatments for mood, anxiety, and related disorders. *Canadian Psychology/Psychologie Canadienne*, 55, 161–176.
- Ingram, R. E., Bernet, C. Z., & McLaughlin, S. C. (1994). Attentional allocation processes in individuals at risk for depression. *Cognitive Therapy and Research*, 18, 317–332.
- Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). Cognitive vulnerability to depression. New York, NY: Guilford Press.
- Ingram, R. E., Miranda, J., & Segal, Z. (2006). Cognitive vulnerability to depression. In L. Alloy & J. Riskind (Eds.), *Cognitive vulnerability to emotional disorders* (pp. 63-91). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.
- Jakobsen, J. C., Hansen, J. L., Simonsen, S., Simonsen, E., & Gluud, C. (2012). Effects of cognitive therapy versus interpersonal psychotherapy in patients with major depressive disorder: a systematic review of randomized clinical trials with meta-analyses and trial sequential analyses. *Psychological Medicine*, 42, 1343–1357.
- Johansson, O., Lundh, L.-G., & Bjärehed, J. (2015). 12-Month Outcome and predictors of recurrence in psychiatric treatment of depression: A retrospective study. *Psychiatric Quarterly*, 86, 407–417.

- Joiner, T. E. (2000). Depression's vicious scree: Self-propagating and erosive processes in depression chronicity. *Clinical Psychology: Science and Practice*, 7, 203–218.
- Joiner Jr., T. E., & Timmons, K. A. (2009). Depression in its interpersonal context. In *Handbook of depression* (pp. 322–339). New York, NY: Guilford Press.
- Joyce, P. R., McKenzie, J. M., Carter, J. D., Rae, A. M., Luty, S. E., Frampton, C. M. A., & Mulder, R. T. (2007). Temperament, character and personality disorders as predictors of response to interpersonal psychotherapy and cognitive-behavioural therapy for depression. *British Journal of Psychiatry*, 190, 503–508.
- Kessler, R. C., McGonagle, K. a, Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., ... Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry*, 51, 8-19.
- Kessler, R. C., & Wang, P. S. (2008). The descriptive epidemiology of commonly occurring mental disorders in the United States. *Annual Review of Public Health*, 29, 115–129.
- Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York: Basic Books.
- Kovacs, M., & Beck, A. T. (1978). Maladaptive cognitive structures in depression. *American Journal of Psychiatry*, 135, 525–533.
- Kupfer, D., Frank, E., & Wamhoff, J. (1996). Mood disorders: Update on prevention of recurrence. In: Mundt, C., Goldstein., M.J. (Eds.), *Interpersonal factors in the origin and course of affective disorders* (pp. 289-302). London, England: Gaskell/Royal College of Psychiatrists.

- Lau, M. A., Segal, Z. V., & Williams, M. G. (2004). Teasdale's differential activation hypothesis: Implications for mechanisms of depressive relapse and suicidal behaviour. *Behaviour Research and Therapy*, 42, 1001–1007.
- Leahy, R. L. (2002). Cognitive therapy: Current problems and future directions. In R. Dowd & E. Thomas (Eds.), *Clinical advances in cognitive psychotherapy: Theory and application* (pp. 418-434). New York, NY: Springer Publishing Co.
- Lewinsohn, P. M., Allen, N. B., Seeley, J. R., & Gotlib, I. H. (1999). First onset versus recurrence of depression: differential processes of psychosocial risk. *Journal of Abnormal Psychology*, 108, 483–489.
- Lewinsohn, P. M., Joiner, T. E., & Rohde, P. (2001). Evaluation of cognitive diathesis-stress models in predicting major depressive disorder in adolescents. *Journal of Abnormal Psychology*, 110, 203–215.
- Lewinsohn, P. M., Steinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-related cognitions: Antecedent or consequence? *Journal of Abnormal Psychology*, 90, 213–219.
- Longmore, R. J., & Worrell, M. (2007). Do we need to challenge thoughts in cognitive behavior therapy? *Clinical Psychology Review*, 27, 173-187.
- Lumley, M. N., Dozois, D. J. A., Hennig, K. H., & Marsh, A. (2012). Cognitive organization, perceptions of parenting and depression symptoms in early adolescence. *Cognitive Therapy and Research*, 36, 300-310.
- Lumley, M. N., & Harkness, K. L. (2007). Specificity in the relations among childhood adversity, early maladaptive schemas, and symptom profiles in adolescent depression. *Cognitive Therapy and Research*, 31, 639–657.

- Luty, S. E., Carter, J. D., McKenzie, J. M., Rae, A. M., Frampton, C. M. a, Mulder, R. T., & Joyce, P. R. (2007a). Randomised controlled trial of interpersonal psychotherapy and cognitive-behavioural therapy for depression. *The British Journal of Psychiatry : The Journal of Mental Science*, 190, 496–502.
- Luty, S. E., Carter, J. D., McKenzie, J. M., Rae, A. M., Frampton, C. M. A., Mulder, R. T., & Joyce, P. R. (2007b). Randomised controlled trial of interpersonal psychotherapy and cognitive-behavioural therapy for depression. *The British Journal of Psychiatry : The Journal of Mental Science*, 190, 496–502.
- Luxton, D. D., Ingram, R. E., & Wenzlaff, R. M. (2006). Uncertain self-esteem and future thinking in depression vulnerability. *Journal of Social and Clinical Psychology*, 25, 840–854.
- MacLeod, C. M. (1991). Half a century of research on the Stroop effect: an integrative review. *Psychological Bulletin*, 109, 163–203.
- Markowitz, J. C., Lipsitz, J., & Milrod, B. L. (2014). Critical review of outcome research on interpersonal psychotherapy for anxiety disorders. *Depression and Anxiety*, 31, 316-325.
- Mazure, C. M., & Maciejewski, P. K. (2003). A model of risk for major depression: Effects of life stress and cognitive style vary by age. *Depression and Anxiety*, 17,
- McBride, C., Atkinson, L., Quilty, L. C., & Bagby, R. M. (2006). Attachment as moderator of treatment outcome in major depression: a randomized control trial of interpersonal psychotherapy versus cognitive behavior therapy. *Journal of Consulting and Clinical Psychology*, 74, 1041–1054.
- Metalsky, Gerald I.; Joiner, T. E. (1997). The Hopelessness Depression Symptom

- Questionnaire. *Cognitive Therapy and Research*, 21, 359–384.
- Metalsky, G. I., & Joiner, T. E. (1992). Vulnerability to depressive symptomatology: a prospective test of the diathesis-stress and causal mediation components of the hopelessness theory of depression. *Journal of Personality and Social Psychology*, 63, 667–75.
- Miranda, J., & Persons, J. B. (1988). Dysfunctional attitudes are mood-state dependent. *Journal of Abnormal Psychology*, 97, 76–79.
- Miranda, J., Persons, J. B., & Byers, C. N. (1990). Endorsement of dysfunctional beliefs depends on current mood state. *Journal of Abnormal Psychology*, 99, 237–241.
- Mongrain, M., & Blackburn, S. (2005). Cognitive vulnerability, lifetime risk, and the recurrence of major depression in graduate students. *Cognitive Therapy and Research*, 29, 747–768.
- Mongrain, M., & Leather, F. (2006). Immature dependence and self-criticism predict the recurrence of major depression. *Journal of Clinical Psychology*, 62, 705–713.
- Monroe, S. M., & Harkness, K. L. (2011). Recurrence in major depression: A conceptual analysis. *Psychological Review*, 118, 655–674.
- Monroe, S. M., & Simons, a D. (1991). Diathesis-stress theories in the context of life stress research: implications for the depressive disorders. *Psychological Bulletin*, 110, 406–425.
- Monroe, S. M., Slavich, G. M., Torres, L. D., & Gotlib, I. H. (2007). Major life events and major chronic difficulties are differentially associated with history of major depressive episodes. *Journal of Abnormal Psychology*, 116, 116–124.
- Morse, J. Q., & Robins, C. J. (2005). Personality-life event congruence effects in late-life

- depression. *Journal of Affective Disorders*, 84, 25–31.
- Mueller, T. I., Leon, A. C., Keller, M. B., Solomon, D. A., Endicott, J., Coryell, W., ... Maser, J. D. (1999). Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. *American Journal of Psychiatry*, 156, 1000–1006.
- Mundt, C. [Ed], Goldstein, M. J. [Ed], Hahlweg, K. [Ed], Fiedler, P. [Ed], & al, et. (1996). Interpersonal factors in the origin and course of affective disorders. Retrieved from <http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=psyc3&NEWS=N&AN=1996-97969-000>
- Nanni, V., Uher, R., & Danese, A. (2012). Childhood maltreatment predicts unfavorable course of illness and treatment outcome in depression: A meta-analysis. *American Journal of Psychiatry*, 169, 141–151.
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking Rumination. *Perspectives on Psychological Science*, 3, 400–424.
- O’Leary, D., Costello, F., Gormley, N., & Webb, M. (2000). Remission onset and relapse in depression: An 18-month prospective study of course for 100 first admission patients. *Journal of Affective Disorders*, 57, 159–171.
- Oliver, J. M., & Baumgart, E. P. (1985). The Dysfunctional Attitude Scale: Psychometric properties and relation to depression in an unselected adult population. *Cognitive Therapy and Research*, 9, 161–167.
- Olthuis Janine, V., Watt Margo, C., & Stewart Sherry, H. (2011). Therapist-delivered distance cognitive behavioural therapy for anxiety disorders in adults. *Cochrane*

- Database of Systematic Reviews*. <http://doi.org/10.1002/14651858.CD009028>
- Otte, C. (2011). Cognitive behavioral therapy in anxiety disorders: Current state of the evidence. *Dialogues in Clinical Neuroscience*, 13, 413–421.
- Parsons, T. (1951). Illness and the role of the physician: a sociological perspective. *The American Journal of Orthopsychiatry*, 21, 452–460.
- Pezard, L., Nandrino, J. L., Renault, B., Massiou, F. El, Allilaire, J. F., Müller, J., ...
Martinerie, J. (1996). Depression as a dynamical disease. *Biological Psychiatry*, 39, 991–999.
- Post, R. M. (1992). Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. *American Journal of Psychiatry*, 149, 999–1110.
- Puig-Antich, J., Lukens, E., Davies, M., Goetz, D., Brennan-Quattrock, J., & Todak, G. (1985). Psychosocial functioning in prepubertal major depressive disorders. II. Interpersonal relationships after sustained recovery from affective episode. *Archives of General Psychiatry*, 42, 511–517.
- Quilty, L. C., Dozois, D. J. A., Lobo, D. S. S., Ravindran, L. N., & Bagby, R. M. (2014). Cognitive structure and processing during cognitive behavioral therapy vs. pharmacotherapy for depression. *International Journal of Cognitive Therapy*, 7, 235–250.
- Ratnasingham, S., Cairney, J., Manson, H., Rehm, J., Lin, E., & Kurdyak, P. (2013a). The burden of mental illness and addiction in Ontario. *Canadian Journal of Psychiatry*, 58, 529–537.
- Ratnasingham, S., Cairney, J., Manson, H., Rehm, J., Lin, E., & Kurdyak, P. (2013b). The burden of mental illness and addiction in Ontario. *Canadian Journal of*

Psychiatry, 58, 529–537.

- Rnic, K., Dozois, J.A., & Machado, D. (2016). A meta-analytic review of stress generation in unipolar depression. Unpublished manuscript, Department of Psychology, Western University, London, Canada.
- Riskind, J. H., & Alloy, L. B. (2006). Cognitive vulnerability to psychological disorders: Overview of theory, design, and methods. *Journal of Social and Clinical Psychology*, 25, 705–725.
- Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review*, 25, 487–510.
- Schmidt, N. B., Schmidt, K. L., & Young, J. E. (1999). Schematic and interpersonal conceptualizations of depression: An integration. In T. Joiner & J. C. Coyne (Eds.), *The interactional nature of depression* (pp. 127–148). Washington, DC: American Psychological Association
- Schmidt, N. B., Joiner, T. E., Young, J. E., & Telch, M. J. (1995). The schema questionnaire: Investigation of psychometric properties and the hierarchical structure of a measure of maladaptive schemas. *Cognitive Therapy and Research*, 19, 295–321.
- Seeds, P. M., & Dozois, D. J. A. (2010). Prospective evaluation of a cognitive vulnerability-stress model for depression: The interaction of schema self-structures and negative life events. *Journal of Clinical Psychology*, 66, 1307–1323.
- Segal, Z. V., & Gemar, M. (1997). Changes in cognitive organisation for negative self-referent material following cognitive behaviour therapy for depression: A primed

- Stroop study. *Cognition & Emotion*, 11, 501–516.
- Segal, Z. V., & Vella, D. D. (1990). Self-schema in major depression: Replication and extension of a priming methodology. *Cognitive Therapy and Research*, 14, 161–176.
- Segal, Z. V. (1988). Appraisal of the self-schema construct in cognitive models of depression. *Psychological Bulletin*, 103, 147–162.
- Segal, Z. V, Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, 63, 749–755.
- Segal, Z. V, Shaw, B. F., Vella, D. D., & Katz, R. (1992). Cognitive and life stress predictors of relapse in remitted unipolar depressed patients: test of the congruency hypothesis. *Journal of Abnormal Psychology*, 101, 26–36.
- Segal, Z. V, Williams, J. M., Teasdale, J. D., & Gemar, M. (1996). A cognitive science perspective on kindling and episode sensitization in recurrent affective disorder. *Psychological Medicine*, 26, 371–380.
- Segrin, C., & Abramson, L. Y. (1994). Negative reactions to depressive behaviors : A communication theories analysis. *Journal of Abnormal Psychology*, 103, 655–668.
- Sheets, E. S., & Craighead, W. E. (2014). Comparing chronic interpersonal and noninterpersonal stress domains as predictors of depression recurrence in emerging adults. *Behaviour Research and Therapy*, 63, 36–42.
- Solomon, A., Arnow, B. A., Gotlib, I. H., & Wind, B. (2003). Individualized measurement of irrational beliefs in remitted depressives. *Journal of Clinical Psychology*, 59, 439–455.

- Spasojević, J., & Alloy, L. B. (2001). Rumination as a common mechanism relating depressive risk factors to depression. *Emotion, 1*, 25–37.
- Starr, L. R., & Davila, J. (2008). Excessive reassurance seeking, depression, and interpersonal rejection: A meta-analytic review. *Journal of Abnormal Psychology, 117*, 762–775.
- Strunk, D. R., Adler, A. D., & Hollars, S. N. (2013). Cognitive therapy skills predict cognitive reactivity to sad mood following cognitive therapy for depression. *Cognitive Therapy and Research, 37*, 1214–1219.
- Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion, 2*, 247–274.
- Teasdale, J. D., & Dent, J. (1987). Cognitive vulnerability to depression: An investigation of 2 hypotheses. *British Journal of Clinical Psychology, 26*, 113–126.
- Timbremont, B., & Braet, C. (2004). Cognitive vulnerability in remitted depressed children and adolescents. *Behaviour Research and Therapy, 42*, 423–437.
- Van Loo, H. M., Aggen, S. H., Gardner, C. O., & Kendler, K. S. (2015). Multiple risk factors predict recurrence of major depressive disorder in women. *Journal of Affective Disorders, 180*, 52–61.
- van Zoonen, K., Buntrock, C., Ebert, D. D., Smit, F., Reynolds, C. F., Beekman, A. T. F., & Cuijpers, P. (2014). Preventing the onset of major depressive disorder: A meta-analytic review of psychological interventions. *International Journal of Epidemiology, 43*, 318–329.
- Wang, J. L., Patten, S., Sareen, J., Bolton, J., Schmitz, N., & MacQueen, G. (2014). Development and validation of a prediction algorithm for use by health

professionals in prediction of recurrence of major depression. *Depression and Anxiety*, 31, 451–457.

- Weissman, A. N., & Beck, A. T. (1978). Development and validation of the Dysfunctional Attitude Scale: A preliminary investigation. *Report: ED167619*. 33pp. Mar 1978. Retrieved from <http://eric.ed.gov/ERICWebPortal/recordDetail?accno=ED167619>
- Wells, A. (2009). *Metacognitive therapy for anxiety and depression* Guilford Press, New York, NY. Retrieved from <https://www.lib.uwo.ca/cgi-bin/ezpauthn.cgi?url=http://search.proquest.com/docview/621609691?accountid=15115>
- Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1988). *Cognitive psychology and emotional disorders*. Chichester, NY: Wiley.
- Wilson, M., Joffe, R. T., & Wilkerson, B. (2000). *The unheralded business crisis in Canada: Depression at work*. Toronto: Global Business and Economic Roundtable on Addiction and Mental Health
- Zanarini, M. C., Skodol, a E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., ... Gunderson, J. G. (2000). The collaborative longitudinal personality disorders study: Reliability of Axis I and II diagnoses. *Journal of Personality Disorders*, 14, 291–299.
- Zimmerman, M., & Coryell, W. (1987). The Inventory to Diagnose Depression (IDD): A self-report scale to diagnose major depressive disorder. *Journal of Consulting and Clinical Psychology*, 55, 55–59.

Appendix A

How Do Our Thoughts Affect Our Mood? Participate in this study!

Western University students 18 or over who have previously suffered from depression are eligible to participate in a study that examines how thinking affects mood over time.

This study involves completing questionnaires and a computer-based task in a lab setting 3 times over a 6-month period (45-55 mins each time). You will receive one research participation credit for each of the 3 in-lab sessions.

Appendix B

Western University students 18 years and over who have previously suffered from depression are eligible to participate in a study that examines how thinking affects mood over time.

This study involves completing questionnaires and a computer-based task in a lab setting 3 times over a 6 month period (45-55 min each time). You will be compensated \$20 for the 1st session and \$10 each time thereafter.

For more information and to complete initial online screening to determine eligibility for the study (no compensation for initial screening, which is expected to take ~ 6 mins), go to:

Appendix C

Subject Line: Mood and Thinking Study

Hello [insert name of participant],

I am contacting you to book an appointment for the lab component of the Mood and Thinking Study. This component of the study is expected to take between 30 minutes and 1 hour to complete. The following times are available:

Please let me know what time you would be available to come in. Our lab is located at Westminster Hall. You can park in one of the spots in front of the building (spots are marked by a "research participants" sign). If you are driving, then we will provide you with a parking pass once you arrive. If you have any questions or concerns, please do not hesitate to contact us by email or telephone at:

Thank you,

[insert your name]

Research Assistant

Western University

Appendix D

Hello, may I please speak to **Insert name of participant here?***

I am contacting you to book an appointment for the lab component of the Mood and Thinking Study. This component of the study is expected to take between 30 minutes and 1 hour to complete. May I list several days and times that we have available and you could select one that is most convenient for you?

If no, ask when would be a good time to call back. Our lab is located at Westminster Hall. You can park in one of the spots in front of the building (spots are marked by a "research participants" sign). If you are driving then we will provide you with a parking pass once you arrive. If you have any questions or concerns, please do not hesitate to contact us by email or telephone at

*If the participant is not available, do not leave a message. Say you will call back another time.

Appendix E

Letter of Information

1. Invitation to Participate

This study explores the cognitive predictors of depressive relapse. You have been invited to participate in an initial in-lab session and two additional follow-up sessions (roughly three and six months in the future). Each of the three in-lab sessions will take approximately 45-55 minutes. For each in-lab session you attend, you will be compensated \$20.

2. Purpose of the Letter

The purpose of this letter is to provide you with information required for you to make an informed decision regarding participation in this research.

3. Purpose of this Study

The purpose of this study is to examine how people organize information and beliefs about the self, and how this changes over time. This will help us to better understand the factors involved in the onset, recurrence and maintenance of depression, which is an area in need of further research.

4. Inclusion Criteria

Individuals who are students at Western University, and have experienced one or more episodes of depression in the past are eligible to participate in this study.

5. Exclusion Criteria

Individuals who are not students at Western University are not eligible to participate in this study. Also ineligible are individuals who are currently depressed, or who have experienced a manic episode at any point in the past.

6. Study Procedures

The initial screening process to determine eligibility for the study will take approximately 6 minutes. There will be three in-lab sessions (conducted in

the Mood Lab at Western University). At each session, you will be asked to complete several written questionnaires, as well as a computer-based task. The initial session and each of the two follow-ups (at roughly three and six months in the future) will take approximately 45-55 minutes. After each session, you will be debriefed by the researcher and asked if you are willing to be contacted in the future to participate in other studies conducted by the Mood Lab. You may withdraw from any given session at any time should you decide you would no longer like to participate, without any loss in compensation for that particular session. Similarly, refusal to answer questions will not result in loss of compensation.

6. Possible Risks and Harms

You may experience some mild discomfort when completing the questionnaires and/or tasks, but this should be transient. Further, you will be provided with a debriefing form at the end of each session that provides resources on campus and in the community that you can use if you are distressed.

7. Possible Benefits

This study gives you the opportunity to learn more about how psychological research is conducted. Additionally, information gathered may provide benefits to society as a whole, including learning more about the course of depression and its associated risk factors.

8. Compensation

For each in-lab session you attend, you will be compensated \$20.

9. Voluntary Participation

Participation in this study is voluntary. You may refuse to participate, refuse to answer any questions or withdraw from the study at any time with no effect on your academic status or relationship to the university. If you refuse to participate partway through the study, any data collected up to that point will not be used.

10. Confidentiality

All data collected will remain confidential and accessible only to the investigators of this study. Data is stored by Western University Psychology Department's secure server and all forms are stored in locked filing cabinets. If the results are published, your name will not be used. If you choose to withdraw from this study, your data will be removed and

destroyed from our database. All data will be destroyed 5 years after final publication of results.

11. Contacts for Further Information

If you require any further information regarding this research project or your participation in the study you may contact the Principal Investigators:
Dr. David Dozois
Daniel Machado

If you have any questions about your rights as a research participant or the conduct of this study, you may contact The Office of Research Ethics

12. Publication

If the results of the study are published, your name will not be used. If you would like to receive a copy of any potential study results, please contact:

This letter is yours to keep for future reference.

Consent Form

Project Title: Predictors of Depressive Relapse

Study Investigators' Names:

Daniel Machado, MSc candidate, Western University

David Dozois, PhD, Western University

I have read the Letter of Information, have had the nature of the study explained to me and I agree to participate. All questions have been answered to my satisfaction.

Participant's Name (please print):

Participant's Signature:

Date:

Person Obtaining Informed Consent (please print):

Signature:

Date:

Appendix F

Debriefing

Project Title: How Thinking Affects Mood Over Time

Thank you for your participation in this study. Research has linked dysfunctional thinking to the development and maintenance of depression. The diathesis-stress model holds that depression results from the development of negative self-schemas that interact with stressful life events (Beck & Dozois, 2011; Dozois & Beck, 2008). Schemas are patterns of thinking that we use to organize and make sense of information in the world around us. Schemas are believed to be made up of both structure (organization of thoughts) and content (beliefs) (Dozois, 2007, 2014; Dozois & Beck, 2008; Dozois & Dobson, 2001a, 2001b). Considerable evidence also links thinking styles that result from negative schema content to the occurrence of depressive episodes. Although negative content tends to wane following improvement from depression, organization of these negative structures tends to remain (Dozois, 2007; Dozois & Bieling, 2010; Dozois & Dobson, 2001a), representing a potential risk factor for relapse.

The goal of this study is to examine these cognitive structures and processes over time, in individuals who have experienced one or more episodes of depression in the past, with the hope of identifying what distinguishes those who relapse with those who maintain recovery. This research is important, because depression is a highly recurrent disorder, and sufferers will often experience several episodes throughout the course of their lifetime. It is hoped that this study will contribute to a better understanding of vulnerability factors that influence the development and maintenance of depression, and possible reasons for relapse. This information will also add to the existing literature base regarding how our thoughts influence the development of depression, as well as potentially inform future clinical practice.

Thank you again for your participation,

Sincerely,

Daniel Machado, M.Sc. candidate

Should you have any questions or concerns about this study, please contact:

Daniel Machado or Dr. David Dozois

Below are a variety of resources if you are interested in learning more about depression, how you can help yourself, or how you can arrange for professional help.

Self-Help References:

If you would like to look up some good self-help books on changing negative thinking, please see:

- ❖ Burns, D. D. (1980). *Feeling good*. New York: Penguin.
- ❖ Burns, D. D. (1989). *The feeling good handbook*. New York: Penguin.
- ❖ Greenberger, D., & Padesky, C. A. (1995). *Mind over mood: Change the way you feel by changing the way you think*. Guilford Press.
- ❖ Wright, J. H., & McCray, L. W. (2011). *Breaking free from depression: Pathways to wellness*. Guilford Press

Available Services

There are several ways in which individuals can access psychological or psychiatric help both on campus and within the City of London, Ontario. If you are feeling depressed or anxious or feel that you could benefit from some individual assistance, the following information may be of use to you.

The Student Development Centre

- Individual appointments are available for students. To make an appointment you can call
- Psychological Services Staff will make every effort to respond as quickly as possible when an individual student requires an emergency appointment.
- Psychological Services Staff can help you deal with a variety of issues including those related to Traumatic Events, Sexual or Physical Assault, Date rape, Interpersonal Violence, and Gay, Lesbian, Bisexual, or Transgendered situations.
- More information about the services offered at SDC can be found on the World Wide Web at

London Crisis Centres

Psychological Services Staff will make every effort to respond as quickly as possible when an individual requires an emergency appointment. If you are in crisis when the office is closed please call one of the numbers listed below.

- **Mental Health Crisis Centre:**
- **Sexual Assault Centre London Crisis Line:**
 - Also 24 hour support line for sex trade workers:
- **Women's Community House Help Line:**
 - Out-of-Town calls:
- **Zhaawanong (Atenlos) Shelter:**
 - Outside of the London area code:
 - 24 hour crisis line:
- **St. Joseph's Sexual Assault and Domestic Violence Centre:**

Student Health Services Counselling Centre

- SHS is located in. Main telephone line:
- The Student Health Services Counselling Centre provides individual counselling for students. The Counselling Centre can be reached at

- The Counselling Centre's Hours of Operation are as follows: Monday to Friday 8:30 a.m.- 4:30 p.m. (Please note the Counselling Centre will be closed when the university is closed.)

London & District Distress Centre

- This is a 24-hour Distress Line:
- Crisis Response Line:
- Access by e-mail at:
- Each problem is handled in an atmosphere of confidentiality, anonymity & impartiality. You do not have to give your name nor does the service use call display; they will not try to identify the caller.

Addiction Services of Thames Valley

- Alcohol & Drug Services of Thames Valley is located at:
- A community service, funded by the Provincial Ministry of Health, Ontario Substance Abuse Bureau. There are currently no charges for clinical services, although fees may be charged for training or seminars.
- Service is available to any resident of Middlesex, Elgin or Oxford County. There are no admission restrictions.
- Provide early intervention to persons who are concerned about substance use and/or problem gambling.
- ADSTV is a gay, lesbian, bisexual, transsexual, and transgender positive environment
- Services include assessment of individuals who have an alcohol and/or drug related problem. Assessments are also available for problem gambling. Based on these assessments the ADS will develop treatment plans for clients and assist with referrals to provide outpatient counselling and aftercare.
- Hours of operation in London are as follows: Monday to Friday - 8:30 a.m. to 4:30 p.m.; Tuesdays- 8:30 a.m. to 9:00 p.m. (closed 12 until 1 p.m. each day and 4:30 to 5:30 p.m. on Tuesdays).
- Self-referrals are welcome, call:

Emergencies After Hours

- If you are in distress during an after-hours time, please go to the **nearest hospital emergency room**.
- **On Campus:** University Hospital:
 - **South London:** Victoria Hospital:
 - **North London:** St. Joseph's Hospital:

Referrals to Other Resources

- Family physicians can provide you with counselling services, and can make referrals to other community resources as needed.
- Specialized services for emotional and interpersonal problems are available, however, a referral from a physician is often necessary.

We hope that this information is helpful to those who need it.

If you are suffering from distress, we encourage you to seek help from an appropriately qualified individual or service centre. Please contact a University or Community Agency that can help you, or to speak with a physician who can refer you to the appropriate resource.

Appendix G



**Western
Research**

Research Ethics

Western University Health Science Research Ethics Board NMREB Delegated Initial Approval Notice

Principal Investigator: Prof. David Dozois
Department & Institution: Social Science/Psychology, Western University

NMREB File Number: 106608
Study Title: Cognitive Schemas as Longitudinal Predictors of Depressive Relapse
Sponsor:

NMREB Initial Approval Date: May 28, 2015
NMREB Expiry Date: May 28, 2016

Documents Approved and/or Received for Information:

| Document Name | Comments | Version Date |
|-------------------------------------|--------------------------------------|--------------|
| Revised Western University Protocol | | 2015/05/04 |
| Letter of Information | | 2015/05/04 |
| Other | Debriefing revised | 2015/05/04 |
| Instruments | Beck Depression Inventory II revised | 2015/05/04 |
| Other | Screening protocol revised | 2015/05/04 |
| Recruitment Items | Study poster ad clean | 2015/05/04 |
| Recruitment Items | Facebook ad revised | 2015/05/04 |
| Recruitment Items | Classroom ad revised | 2015/05/04 |
| Other | Consent Form | 2015/03/18 |
| Instruments | Young Schema Questionnaire | 2015/03/18 |
| Instruments | Negative Life Events Scale | 2015/03/18 |
| Instruments | Cognitive Distortions Scale | 2015/03/18 |
| Instruments | Dysfunctional Attitudes Scale | 2015/03/18 |

The Western University Non-Medical Research Ethics Board (NMREB) has reviewed and approved the above named study, as of the NMREB Initial Approval Date noted above.

NMREB approval for this study remains valid until the NMREB Expiry Date noted above, conditional to timely submission and acceptance of NMREB Continuing Ethics Review.

The Western University NMREB operates in compliance with the Tri-Council Policy Statement Ethical Conduct for Research Involving Humans (TCPS2), the Ontario Personal Health Information Protection Act (PHIPA, 2004), and the applicable laws and regulations of Ontario.

Members of the NMREB who are named as Investigators in research studies do not participate in discussions related to, nor vote on such studies when they are presented to the REB.

under the IRB registration number IRB 00000941.

ber

This is an official document. Please retain the original in your files.

Curriculum Vitae

Daniel Machado

Education

September 2014 – Present: Master of Science
Clinical Psychology
Western University, London, Ontario, Canada
Supervisor: Dr. David Dozois, Ph.D., C. Psych.
Master's Thesis (in progress): Cognitive Schemas as Longitudinal Predictors of depressive relapse

Fall 2012: Bachelor of Arts
Honours Psychology
University of Waterloo, Waterloo, Ontario, Canada
Supervisor: Dr. Joanne Wood
Honours thesis: Communication Context and Expressivity: Online versus face-to-face communication as a function of self-esteem and agreeableness

Awards and Honours

- **Dean's Honours List:** 2008 – 2012

Publications

Rnic, K., Dozois, D. J. A., & Machado, D. (In revision). A meta-analytic review of stress generation in unipolar depression. *Psychological Bulletin*.

Conference Presentations

Machado, D., Dozois, D. J. A., Shumlich, E., Rnic, K., & Maiolino, N. (Accepted). Cognitive schemas as longitudinal predictors of depressive relapse. Talk submitted to the 2016 meeting of the International Congress of Psychology, Yokohama, Japan.

Rnic, K., Dozois, D. J. A., & **Machado, D.** (October, 2015). A meta-analytic review of stress generation in unipolar depression. Poster presented at the 2015 meeting of the Society for Research in Psychopathology, New Orleans, LA.

Research Experience

May 2013 – August 2014: Research Assistant

- Employed by Dr. Erik Woody
- University of Waterloo, Waterloo, ON

January 2011 – January 2012: Honours Thesis – Communication Context and Expressivity: Online versus face-to-face communication as a function of self-esteem and agreeableness

- Supervised by Dr. Joanne Wood
- University of Waterloo, Waterloo, ON

July 2010 – August 2014: Research Assistant

- Employed by Dr. Joanne Wood
- University of Waterloo, Waterloo, ON

Teaching Experience

Teaching Assistant for Human Sexuality (September 2014 – April 2015)

Clinical Experience

January 2016 – April 2015: Initial Assessment Practicum

- Under the supervision of Gloria Grace, Ph.D., C. Psych at University Hospital – Neuropsychological Assessment

January 2016 – April 2015: Initial Assessment Practicum

- Under the supervision of Ellen Vriezen, Ph.D., C. Psych at London Health Sciences Centre – Children's Hospital, Neuropsychological Assessment

May 2015 – August 2015: Initial Intervention Practicum

- Under the supervision of Kathy Dance and Mercedes Umana Garcia Ph.D., C. Psych at the Student Development Center at Western University.

Graduate Level Courses

Professional Foundations of Clinical Psychology
Clinical Skills Pre-Practicum
Research Design and Statistics

Assessment
Adult Psychopathology and Diagnosis
Psychotherapy Approaches

Volunteer Work

September 2015 – Present: Member of Clinical Students Advisory Committee

October 2015 – May 2015: Secretary for Advocacy Through Action