

**THE LINK BETWEEN EARLY LIFE STRESS AND DEPRESSIVE ILLNESS:
THE MEDIATING ROLE OF COPING, COGNITIVE FLEXIBILITY,
AND RESILIENCE**

by

Samantha Santoni

A Thesis submitted to the Faculty of Graduate Studies and Postdoctoral Affairs

in partial fulfillment of the requirements for

Master's of Science degree

in

Neuroscience

Carleton University

Ottawa, Ontario, Canada

©2017 Samantha Santoni

Abstract

The relationship between early life stress and depression might be affected by several different factors, and several variables might likewise mediate this relationship. Among others, the relationship between early life stressors and later depression might operate through resilience, later negative experiences, cognitive flexibility, and coping. Moreover, these relations may be moderated by the brain-derived neurotrophic factor (BDNF) Val66Met polymorphism. The present investigation demonstrated that in Carleton University undergraduates (N=248), depressive symptoms were positively correlated with early life trauma, emotion-focused and avoidant coping, and later negative experiences, and were negatively linked with problem-focused coping, resilience, and cognitive flexibility. The latter variables (coping style, recent negative events, resilience, and cognitive flexibility) also served as mediators between early-life stressors and depression. There was no significant moderating effect of BDNF on these relationships; however, this should not be taken to imply that BDNF does not have a role in the evolution of depressive illness. The data were discussed in terms of these mediating contributions on the development of depressive illness.

Acknowledgements

First and foremost, I owe a debt of gratitude to my esteemed supervisor, Dr. Hymie Anisman, for his endless guidance and support. Dr. Anisman's patience and genuine caring has been invaluable, both academically and personally (whether he'd like to admit it or not). I would also like to thank Dr. Kim Hellemans and Diane Trenouth for their continued support and mentorship throughout my many years at Carleton. Further, I'd like to thank the other members of the Anisman lab, especially Robbie Woods, for his collaboration on this project. Finally, I would not have made it this far without the tremendous love and support of my family and friends, both near and far away. And to my Grandpa Bob and Grandma Donna who still hope I'll be the first doctor in the family, I dedicate this thesis.

Table of Contents

Title Page	i
Abstract	ii
Acknowledgements	iii
Table of Contents	iv
List of Tables	v
List of Figures	vi
List of Appendices	vii
Introduction	1
Methods	13
Results	17
Discussion	24
References	30
Appendices	44

List of Tables

Table 1. Mean scores for all variables across each BDNF genotype (Val/Val, Val/Met, Met/Met).

Table 2. Bivariate correlations depicting relations between depressive symptoms, coping styles, resilience, and cognitive flexibility.

Table 3. Bivariate correlations depicting relations between depressive symptoms, early-life trauma, and recent negative events.

List of Figures

Figure 1. Schematics of the multiple mediation analysis examining the mediating role of recent negative events and resilience in the relationship between total early life trauma and depression.

Figure 2. Schematics of the multiple mediation analyses examining the mediating role of recent negative events and resilience in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression.

Figure 3. Schematic of the mediation analysis examining the mediating role of cognitive flexibility in the relationship between total early life trauma and depression.

Figure 4. Schematics of the mediation analyses examining the mediating role of cognitive flexibility in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression.

Figure 5. Schematic of the multiple mediation analysis examining the mediating role of different coping strategies (emotion-focused, problem-focused, and avoidant) in the relationship between total early life trauma and depression

Figure 6. Schematics of the multiple mediation analyses examining the mediating role of emotion-focused, problem-focused, and avoidant coping strategies in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression.

List of Appendices

Appendix A	Recruitment Notice
Appendix B	Informed Consent
Appendix C	Debriefing
Appendix D	General Information
Appendix E	Beck Depression Inventory (BDI)
Appendix F	Additional Debriefing Form
Appendix G	Suicidal Ideation Protocol
Appendix H	Early Life Trauma Inventory (ELTI)
Appendix I	Recent Life Events Scale (RLES)
Appendix J	Survey of Coping Profiles Endorsed (SCOPE)
Appendix K	Cognitive Flexibility Questionnaire (CFQ)
Appendix L	Connor-Davidson Resilience Scale (CD-RISC)

Introduction

Depression is the most widespread psychiatric illness, affecting millions of people worldwide, accounting for a large proportion of missed work and disability. While many different perspectives have been examined concerning the etiology of this illness, there has been an increasing focus on multi-dimensional approaches to understanding depressive illness, wherein experiential, cognitive, and biological contributions are considered.

Childhood trauma and maltreatment may lead to increased psychopathology later in life (Kendler et al., 1999; Kendler et al., 2004; van Loo et al., 2015). In this regard, experiencing traumatic events in childhood may predispose individuals to further psychological stressor encounters ('stress generation'), potentially resulting in symptoms of depression (Foley et al., 1996; Liu & Alloy, 2010). In contrast to the effects of relatively intense or chronic stressors, moderate challenges may have the opposite effect, essentially preparing the individual to contend effectively with subsequent stressors (Lyons & Parker, 2007; Parker et al., 2006). Aside from these influences, individual difference factors may be related to or interact with early life adversity in predicting later psychological disturbances. These include coping style, cognitive flexibility, and resilience.

Beyond these interactions, the presence of particular genes could moderate the impact of early and later stressor experiences. In this regard, brain-derived neurotrophic factor (BDNF), which is fundamental for neuroplasticity, neurogenesis, and synaptogenesis, serves to generate, propagate, and aid in the survival of new neurons as well as to help the lasting neural connections remain malleable (Park & Poo, 2013). Thus, BDNF is arguably fundamental for learning and memory processes (Bath & Lee, 2006; Egan et al., 2003; Lu & Gottschalk, 2000), and there has been evidence supporting the view that diminished BDNF functioning within the

hippocampus may contribute to the emergence and maintenance of depression (Duman et al., 1999; Duman & Monteggia, 2006). As such, it might be expected that a Val66Met polymorphism on the gene coding for brain-derived neurotrophic factor (BDNF), would be linked to the development of depression, or that the influence of this polymorphism would be most notable when accompanied by recent or early life stressors, as previously observed in relation to the serotonin transporter (Caspi et al., 2003).

In relationship to psychopathology, it was initially believed that higher levels of BDNF would be protective against mental illness, in that individuals without the polymorphism would be better off than those carrying a Met variant (Jiang et al., 2005; Perea et al., 2012). However, the relative level of BDNF may not be as important as the environment with which it is matched. That is, given that BDNF is involved in learning, some people who have experienced traumas may be better off if they are not learning the maladaptive coping strategies and cognitive styles that come along with such an experience. Therefore, it has been suggested that lower levels of BDNF, as is found in Met carriers, may actually be protective against a transition to mental illness when combined with a certain set of traumatic or stressful life experiences (Caldwell et al., 2013).

The current investigation assessed the link between early-life stress and depressive affect, and the mediating roles of recent life stress, coping style, resilience and cognitive flexibility. As well, it was hypothesized that these linkages would be moderated by the BDNF polymorphism.

Depression

Major depressive disorder (MDD) is characterized by melancholic mood and/or anhedonia. In addition to these symptoms, MDD is also accompanied by feelings of guilt and

worthlessness, changes to sleep and appetite, and suicidal thoughts and actions. Depending on the nature of these symptoms (e.g. increased vs. decreased appetite, increased or decreased sleep), depression can be categorized as being typical or atypical, which might involve different underlying mechanisms and treatment strategies (Anisman et al., 1999). Depressive illness can also manifest in a milder form in which symptoms are present despite not meeting diagnostic criteria, referred to as minor depression. This type of depressive illness, as well as other less severe forms of depression (such as dysthymia) present strong reasoning for evaluating depressive symptomatology on a continuum.

The World Health Organization estimates that major depression affects more than 300 million people worldwide (WHO, 2017). In Canada, an estimated 10.1% of the population aged 15 or older has symptoms consistent with a mental illness, 5.4% of which were mood disorders. Notably, more youth (aged 15 – 24) fit these criteria than any other age group, and females outnumber males by nearly double (Statistic Canada, 2012). Furthermore, among female youth, antidepressants are one of the most commonly prescribed medications, second only to hormonal contraceptives (Rotermann et al., 2014). The economic toll of these illnesses in Canada is also significant, and a recent report estimated that the cost of depression is \$32.3 billion annually, mainly due to missed work (Conference Board of Canada, 2016).

Despite its prevalence, the etiology of depression is still poorly understood. There is no disputing that stressors play an important role in the development of depressive illness, and both retrospective and prospective studies have indeed found that they are strongly linked (Kendler et al., 1999). Likewise, coping processes, cognitive mechanisms, personality factors, and several neurobiological processes have been implicated in the evolution and maintenance of depressive disorders. While the individual contributions of these factors have frequently been assessed,

there has also been a focus on an integrative understanding of depressive disorders, wherein experiential, genetic, and cognitive perspectives are considered concurrently.

Stress and Depression

Stressful experience, as already indicated, have been strongly linked to the development of depression, as well as its recurrence. When asked to recall major stressful events in the period leading up to the onset of depression, patients report higher incidences of negative events than non-depressed controls (Hammen, 2005; Mazure, 1998). Depressed individuals also report higher occurrences of minor stressful events and daily hassles than their healthy counterparts (Ravindran et al., 1995). Moreover, just as stressful events contribute to a risk for depression, depression itself may contribute to the occurrence of more stressful life events (Stress generation). That is, individuals who display signs of depressive disorders tend to place themselves into situations in which they are more likely to become stressed (Foley et al., 1996; Liu & Alloy, 2010). That said, not everyone who experiences stressful life events will develop a depressive disorder, indicating that there other factors ought to be considered in the evolution of this disorder.

Negative experiences early in life may dispose individuals to depressive illness. Among other things, stressful life events encountered during childhood or adolescence, can affect the way that individuals deal with stressors later in life (Tsoory et al., 2007). Likewise, early life stressors might prime neurobiological systems (sensitization) so that later stressor challenges promote greater neurochemical disturbances that culminate in the onset of depression in adulthood (Anisman et al., 2003). For example, the biological effects of acute stressors can be elicited more readily if mice have previously encountered a stressful experience (Anisman et al.,

2008; De Kloet et al., 2005; Uchida et al., 2010). This said, low levels of early life stressors (tolerable stressors) could actually have the opposite effect, by preparing the individual to handle stress later on. That is, when exposed to mild or moderate early life stressors (such as early handling or maternal separation), both rodents and primates show marked reductions in HPA axis responses upon subsequent stressor encounters (Lyons & Parker, 2007; Parker et al., 2006; Shonkoff et al., 2009).

Appraisals and Coping

In response to a potential stressor, appraisals of the situation are initiated in which the individual makes an assessment as to the type of challenge and the threat it represents, largely based on their past experiences (Lazarus, 1966). Following the decision that this event poses a threat, a secondary appraisal is made concerning whether the situation can be dealt with effectively (Lazarus & Folkman, 1984). These appraisals form the basis upon which particular coping strategies will be made, although individuals likely will already have developed coping *styles* that guide the actions that will be taken.

Coping methods typically fall into three broad categories: problem-focused, emotion-focused, and avoidant strategies. Problem-focused coping methods are used to manage the problem that is causing the distress and includes tactics such as information gathering and seeking advice. Avoidant coping, by contrast, entails behaviours such as finding distractions and distancing from the problem, whereas emotion-focused coping involves strategies such as rumination, day-dreaming, or blaming others (Folkman, 2013; Matheson & Anisman, 2003). While some behaviours seem to fit seamlessly into one category or another, others can be harder to classify. For example, seeking social support could fit into any of the three coping strategies depending on the nature of this behaviour; be it a shoulder to cry on (emotion-focused), a way to

get objective advice (problem-solving) or as a distraction (avoidant). Even the coping strategies themselves can be viewed as either adaptive or maladaptive depending on the context (Folkman, 2013).

Generally, problem-focused coping is regarded as the most productive strategy for dealing with a given stressor, while emotion-focused and avoidant strategies are less so. That said, it is unlikely that any one of these coping strategies is inherently good or bad, and that instead, engaging in one method could be superior to the others in the context of a specific situation. For example, among caregivers of people with dementia, only emotion-focused coping seems to protect against the adverse effects of caregiver burden (Cooper et al., 2008). By contrast, when dealing with the stress of medical school, many students' perceived stress was decreased when they engaged in avoidant-coping strategies such as sleeping or participating in recreational activities (Shaikh et al., 2004). Despite there being many ways to cope successfully in a given situation, individuals suffering from depressive disorders tend to use inappropriate coping more frequently (Ravindran et al., 1995). However, following treatment, the attenuation of depressive symptomatology may also be associated with improvements in coping methods, as well as in perception of stressful events (Ravindran et al., 1995).

Appropriate appraisals and coping and the ability to be flexible in these processes (by engaging multiple behaviours) is likely very important to an individual's well-being and may contribute to a resilience to depressive episodes. For example, among university students, resilience associated with potentially stressful events was directly related to coping flexibility (Galatzer-Levy et al., 2012). Furthermore, resilient adolescents tend to engage in more problem-focused coping than their vulnerable counterparts (Dumont & Provost, 1999).

Cognitive Flexibility

Cognitive flexibility refers to the ability to exert cognitive control in switching between “cognitive sets” and to recruit various mental resources in order to adapt to changing environmental stimuli. Depressed individuals frequently display cognitive rigidity (Young et al., 2001), which could undermine appropriate appraisals and coping, and may ultimately promote maladaptive beliefs and automatic negative thoughts, leading to a depressive episode (Fossati et al., 2001; Teasdale et al., 1995).

There are several techniques by which cognitive flexibility can be measured, including behavioural paradigms (e.g. Wisconsin Card Sorting Task (WCST), Stroop test) and self-report questionnaires. The WCST evaluates the participant’s ability to modify their behavior in the context of non-reinforced responses. In this paradigm, individuals with impairments in cognitive flexibility show perseverance by responding to previously, but no longer, correct stimuli. Self-report questionnaires, alternatively, have respondents evaluate the degree to which they identify with statements such as “I can’t focus on anything when I’m upset” and “I take the time to think of more than one way to resolve the problem”. Using these approaches, impaired cognitive flexibility has been associated with depressive illness (Austin et al., 2001; Grant et al., 2001; Trivedi & Greer, 2014), and may be linked to the recurrence of depressive episodes (Alexopoulos et al., 2000). Interestingly, impaired cognitive flexibility may even persist into remission (Paradiso et al., 1997), raising the possibility of this feature being a state characteristic predictive of relapse.

By contrast, cognitive flexibility has been associated with a propensity to engage in adaptive coping strategies, and hence limited presence of depressive symptomatology (Dennis & Vander Wal, 2010). Moreover, treatments that attenuate depressive symptomatology, such as

cognitive behavioural therapy, are accompanied by enhanced cognitive flexibility (Hollon et al., 1996), and this effect might be attributed to its involvement in cognitive restructuring (Johnco et al., 2013). As such, cognitive flexibility could be an important resilience factor that acts against depressive illness.

The Role of BDNF

Brain-derived neurotrophic factor (BDNF) is a prominent growth factor involved in modulating neuroplastic changes via processes such as synaptogenesis, and long-term potentiation (Bath & Lee, 2006; Laske & Eschweiler, 2006). Primarily, it serves to generate, propagate, and aid in the survival of new neurons, as well as to assure neuronal malleability. It seems that BDNF could also contribute to the development of depressive symptomatology, and has been linked with many aspects of this illness. For example, serum BDNF is lower in depressed patients and has been linked to hippocampal atrophy seen in these individuals (Duman & Monteggia, 2006; Schmidt & Duman, 2007). It is also associated with the severity and recurrence of depressive episodes, and with the positive effects of antidepressant treatments (Gonul et al., 2005; Larsen et al., 2010; Ricci et al., 2010). Moreover, the effectiveness of rapidly acting antidepressant agents, such as ketamine, has been at least partially attributed to changes in BDNF (Duman et al., 1999; Duman et al., 2012; Lepack et al., 2015).

Much of the data in humans pertaining to the BDNF-depression linkage has come from studies of BDNF levels in blood (Gupta et al., 2016; Ricken et al., 2013) and from studies assessing the relationship between BDNF polymorphisms and incidences of depression. In humans, a single nucleotide polymorphism (SNP), involving a valine to a methionine substitution on the BDNF gene, has been associated with altered BDNF activity and disturbed

memory and hippocampal functioning, as well as depressive symptoms, perhaps due to impairments in intracellular trafficking and activity-dependent secretion of BDNF (Chepenik et al., 2009; Egan et al, 2003; Liu et al., 2012). To a significant extent, the outcomes of these studies, as well as those that considered serum levels of BDNF, have been in line with a role for BDNF in relation to depression (Duman & Monteggia, 2006; Sen et al., 2008). However, it has also become clear that attributing depression to this neurotrophin is likely an over simplification, given that depression involves multiple environmental and neurobiological triggers.

Resilience

Most studies of depressive illness have revolved around vulnerabilities (such as those discussed in the previous sections), but it is also important to consider factors that contribute to hardiness. Resilience refers to the elimination of already existing illnesses or factors that contribute to quicker recovery. As well, resilience is also used to refer to individual characteristics that act against illness occurrence. Several factors have been identified that may contribute to an individual's resilience to stressors, including genetic factors, early experiences, age, hedonia, adaptability, and problem-solving capabilities (Anisman, 2014; Charney, 2004). Furthermore, resilience is closely related to social support and social identity, in that having a strong social identity and multiple group memberships can be protective against depressive illness (Haslam et al., 2015; Ysseldyk, et al., 2013). In contrast, the loss of such social identities has been associated with an increase in depressive symptomatology (Seymour-Smith et al., 2016). Predictably, interventions such as Groups 4 Health, that serve to augment social identity and group membership, improve resilience and reduce vulnerability to depression and anxiety (Haslam et al., 2016).

Just as having many vulnerabilities can culminate in the onset of depression, the presence of multiple resilience factors may be optimal to protect against illness. For example, having social support may or may not be sufficient in the presence of personality disorders that predispose to depression. Ideally, resilience would be best in the context of having good social support, positive early experiences, and effective problem-solving skills. However, it is also possible that the presence of even a single strong resilience factor, such as close social support, may in some instances be sufficient to counteract other vulnerabilities. In essence, it is possible that resilience is dependent on the way these pieces come together, and that this process varies considerably between individuals and across circumstances (Anisman, 2014).

Aside from these personality and psychosocial factors, it is likely that there are neurobiological and genetic factors that contribute to resilience. It is thought that components of the nervous system, such as norepinephrine (NE) and neuropeptide Y (NPY), may be among these correlates. Indeed, reductions in NE and NPY are thought to contribute to mental illnesses such as post-traumatic stress disorder (PTSD), whereas elevated levels are associated with resilience. For example, during highly stressful training, resilient Special Forces operatives have reduced anxiety compared to their average military counterparts, and this was accompanied by elevated levels of NE and NPY (Morgan et al., 2000; Morgan et al., 2002).

Findings in both human and animal studies have implicated brain-derived neurotrophic factor (BDNF) as another neurochemical contributor to resilience. In this regard, resilience is inferred from the finding that adequate or high BDNF levels are accompanied by the absence of a depressive-like state that is more likely to occur with low levels of BDNF. Specifically, hippocampal BDNF levels were predictive of resilience to chronic stress in rodents, and reductions of BDNF were associated with prolonged corticosterone secretion (Taliaz et al.,

2011). Furthermore, in rats, higher BDNF and its precursors within the prefrontal cortex were accompanied by greater resilience regarding the impact of stressors on performance in a learned helplessness paradigm (B. Yang et al., 2016). Interestingly, these BDNF actions were significantly related to BDNF levels within the medial prefrontal cortex and dentate gyrus, and it was suggested that brain-region specific levels of BDNF may contribute to resilience (C. Yang et al., 2015). It has also been reported that rats that tended to be stress-resilient, defined as not succumbing to behavioural disturbances in response to strong stressors, exhibited greater activation of the BDNF gene (Farhang et al., 2014).

The data in humans pointing to BDNF in relation to resilience are scant, and as already indicated, largely being inferred from the findings that diminished BDNF may be linked to depressive mood (Duman & Monteggia, 2006). This said, much like the findings with NPY, it has been reported that an epigenetic effect on the BDNF promoter gene was linked to combat related post-traumatic stress disorder (Kim et al., 2017). Further, among children who experienced a natural disaster in the form of a hurricane, the appearance of PTSD and depression was linked to the perceived severity of the stressor, and this effect was stronger for children with a BDNF polymorphism (La Greca et al., 2013). Whether high levels of BDNF limit depression owing to the instigation of better coping is also uncertain. However, given its actions on neuroplasticity it could have an important influence on experience-dependent memory, hence affecting coping strategies and styles. From this perspective, BDNF would be a fundamental ingredient that contributes to stress-resilience. There have similarly been reports that the presence of a BDNF polymorphism may be accompanied by increased incidence of depressive mood (van Winkel et al., 2014). Yet, it has been suggested that BDNF may play more of a role in affecting sensitivity to environmental stimuli (irrespective of whether these are negative or

positive) rather than disposing individuals directly to a depressive-like condition (Caldwell et al., 2013; Cruz-Fuentes et al., 2014). In all likelihood, diminished BDNF does not act alone in relation to depression or PTSD, but instead, is part of a network or constellation of neurobiological changes that favor psychological disorders. For instance, it has been reported that although a polymorphism related to catechol-O-methyltransferase (COMT) may be linked to resilience, this outcome may be moderated by the presence of a BDNF mutation/polymorphism (Kang et al., 2013).

It seems that our understanding of how the BDNF polymorphism is related to stress and depression is still incomplete. Having more BDNF appears to be advantageous, and in this sense Val homozygous individuals would be more resilient than individuals carrying at least one Met allele. However, it is equally possible that BDNF serves to make environmental stimuli more salient, and thus, individuals without the polymorphism would not only gain from positive life experiences, but also be negatively affected by adverse events. In contrast, individuals carrying the BDNF polymorphism would be expected not to gain from positive early life events, but by the same token the potential adverse effects of negative early life experiences would also not be realized.

In the present investigation, it is hypothesized that:

1. Early life stress would be linked to depression, and that this relationship would be mediated by recent life stressors and resilience.
2. The relationship between early life stress and depression would be mediated by the individual's coping style and cognitive flexibility, and this would be moderated by the individual's BDNF genotype.

3. Early life stress would be associated with depression, and this relationship would be moderated by a BDNF polymorphism.

Methods

Participants and Procedure

The study protocol was approved by the Psychology Research Ethics Board at Carleton University. Participants were recruited online by the SONA system at Carleton University between fall 2014 and winter 2015 (Appendix A). Participants comprised 158 male and 335 female undergraduate students ranging from ages 17 - 46. Upon arrival to the testing session, participants were given an informed consent form explaining the nature of the study (Appendix B). As part of this process, participants were asked to choose one of three options regarding the use of the saliva sample that they would be providing. After having given consent, participants filled out a series of questionnaires consisting of: General Information, the Beck Depression Inventory (BDI, Beck, 1972), the Survey of Coping Endorsements (SCOPE; Matheson & Anisman, 2003), the Early Life Trauma Inventory (ELTI; Bremner, et al., 2000), and a modified version of the Life Experiences Survey (RLES; Sarason, et al., 1978). Once the booklet was completed, participants provided a 2 ml saliva sample. Upon completion of the study, all participants were debriefed (Appendix C).

Measures

General Information. Participants provided demographic details, medical history, and information regarding current and past places of residence for screening purposes (Appendix D).

Depressive Symptoms. Depressive symptomatology was assessed using the Beck Depression Inventory (BDI - 21 item version; Beck, 1972; Appendix E). The BDI is a widely used self-administered questionnaire to assess the intensity of depressive symptoms (e.g. sadness, worthlessness, suicidal ideation) in both clinical and non-clinical individuals. For each item, participants indicate which statement best represents their level of depressive symptomatology, where lower numbers (e.g. 0) indicate less severe symptoms, and higher numbers (e.g. 4) indicate more severe symptoms. The overall intensity of depression symptoms is computed by summing the scores across the 21 items, where 0-9 indicates minimal depression, 10-18 indicates mild-moderate depression, 19-29 indicates moderate-severe depression, and 30+ indicates severe depression.

Early Life Trauma. Early life trauma was assessed using the Early Life Trauma Inventory (ELTI; Bremner et al., 2000; Appendix F). This 23-item measure assess exposure to a variety of potentially traumatic experiences between the ages of 0-5, 6-12, and 13-18. Events are categorized as General Traumas (e.g. exposure to natural disaster), Physical Punishment (e.g. being slapped in the face), Emotional Abuse (e.g. being put down or ridiculed), and Sexual Abuse (e.g. being forced or coerced to touch someone in an intimate part of their body). The frequency of these events is assessed using a 6-point scale, on which participants indicate whether each event has occurred from never (0) to more than ten times (5). Due to the age range of the participants in the study, Early life trauma was operationalized as events that occurred at or prior to the age of 12. As such, early life trauma was computed by summing up all events that occurred at or prior to the age of 13.

Recent Life Experiences. The negative impact of life events over the past year were assessed using a modified version of the Recent Life Experiences Survey (RLES; Sarason et al.,

1978; Appendix G). The RLES is a self-report scale that comprises 49 items, both positive and negative. Respondents indicate whether or not they have experienced a particular event in the past year, and if so, whether it had a negative or positive impact on their life at that time. The modified version of this scale used in the present investigation also contains potentially stressful events specific to student populations (e.g. failing an important exam). Respondents indicate the extent to the negative or positive experience on a scale of -100 (extremely negative) to +100 (extremely positive), with 0 having no impact. The number of positive or negative experiences is calculated by summing all the instances that the respondent indicated an event took place, and extent of impact is computed by summing the total amount of impact for negative or positive experiences.

Coping Style: Coping styles were assessed using the Survey of Coping Profile Endorsement (SCOPE; Matheson & Anisman, 2003; Appendix H). The SCOPE is a 50-item measure comprised of 13 different coping methods that fall into 3 broad categories; problem-focused, emotion-focused, and avoidant strategies. Participants indicate the extent to which they resort to each of the behaviours as a way of dealing with general stressors using a scale of 0 (Never) to 4 (Frequently).

Cognitive Flexibility. The Cognitive Flexibility Questionnaire (CFQ; Gabrys, Matheson & Anisman, *unpublished*; Appendix I) was used to assess the degree to which individuals are flexible in their feelings, thoughts, and actions when confronted with stressful experiences. This 28-item scale has respondents indicate from 1 (Strongly Disagree) to 7 (Strongly Agree), their endorsement of various strategies for managing negative thoughts, memories, and emotions that may be provoked by stressful situations (e.g. “I can’t focus on anything when I’m upset”, “I take

the time to think of more than one way to resolve the problem”, “It’s difficult to let go of intrusive thoughts or emotions”).

Resilience. Resilience was assessed using the Connor-Davidson Resilience Scale (CD-RISC; Connor & Davidson, 2003; Appendix J). The CD-RISC is a self-report scale that comprises 25 items that assess the participant’s ability to cope with stress (e.g. “I tend to bounce back after illness or hardship”, “I like challenges”, “I can deal with whatever comes”) and thus, be resilient to depressive illness and stress reactions. Respondents rate each statement on a 5-point scale from 0 (Not true at all) to 4 (True nearly all of the time) how much it relates to them for general stressors in the last month.

Genotyping

NORGEN Saliva DNA Isolation Kits were used to collect saliva samples (~2ml) for subsequent isolation and purification of DNA from the preserved samples, which were then stored at -80°C. Steps for collection, preservation, and isolation were followed as outlined in the kits’ instructions. All samples were diluted in ultrapure water to a concentration of 20ng/ul and measured to 35µl prior to shipment. DNA samples were sent to the McGill University and Genome Quebec Innovation Centre in Montreal, Quebec for SNP genotyping using Sequenom® iPLEX® Gold Genotyping Technology.

Although data were collected from non-Caucasian participants (n=221), only Caucasian participants (n=248) were used for analyses involving genotype, due to the notable ethnic differences seen in past genetic studies. Specifically, the frequency of the Met polymorphism appears only 25-32% in Caucasians (Cargill et al., 1999; Shimizu et al., 2004), whereas in Asian populations it may occur as much as 40-50% (Choi et al., 2006; Itoh et al., 2004). While it has

been suggested that these differences may play a role in the apparent disparities seen in the occurrence of major depression between these populations (Verhagen et al., 2010), the contribution of the polymorphism is still unclear. Therefore, a homogeneous Caucasian sample were used in the present investigation.

Results

The statistical analyses were performed using IBM SPSS Statistics 20 for Mac (Armonk, NY, USA: IBM Corp.). Mean scores were tabulated for depressive symptoms, recent negative events, resilience, cognitive flexibility, each of the coping styles, and early life trauma for each of the BDNF genotypes (Val/Val, Val/Met, Met/Met) and are shown in Table 1.

Table 1. Mean scores for all variables across each BDNF genotype (Val/Val, Val/Met, Met/Met)

Mean Score	Val/Val (N = 164)	Val/Met (N = 73)	Met/Met (N = 11)
Depressive Score (BDI)	9.5	9.0	8.7
Recent Negative Events	3.6	3.5	2.6
Resilience	68.4	68.8	69.7
Cognitive Flexibility	4.1	4.0	4.0
Coping			
Emotion-focused	1.8	1.9	1.9
Problem-focused	2.4	2.4	2.5
Avoidant	2.1	2.2	2.1
Early Life Trauma			
Total	16.3	12.7	28.5
General trauma	4.7	2.9	6.3
Physical abuse	3.9	3.5	7.5
Emotional abuse	6.9	5.9	13.7
Sexual abuse	0.7	0.4	0.9

Pearson correlations were calculated to assess the relations between self-reported scores for cognitive flexibility, resilience, coping, and depressive symptomatology. As expected, as

shown in Table 2, depression scores were positively correlated with emotion-focused and avoidant coping, and negatively correlated with problem-focused coping. Predictably, depressive symptoms were also negatively associated with resilience and cognitive flexibility.

Table 2. Bivariate correlations depicting relations between depressive symptoms, coping styles, resilience, and cognitive flexibility.

	1	2	3	4	5
(1) Depressive Symptoms					
<i>Coping Styles</i>					
(2) Emotion-focused	.59***				
(3) Problem-focused	-.32***	.02			
(4) Avoidant	.48***	.58***	-.08		
(5) Resilience	-.56***	-.41***	.54***	-.26***	
(6) Cognitive Flexibility	-.57***	-.53***	.41***	-.35***	.61***

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

The same analysis was conducted to assess the relationships between different types of early-life trauma (general trauma, physical abuse, emotional abuse, and sexual abuse), recent negative events, and depressive symptoms. As expected, total trauma, as well as each type of trauma, was positively correlated with scores of depression (Table 3). As well, number of recent negative events was positively predictive of increased depressive symptoms.

Table 3. Bivariate correlations depicting relations between depressive symptoms, early-life trauma, and recent negative events.

	1	2	3	4	5	6
(1) Depressive Symptoms						
<i>Early-Life Trauma</i>						
(2) Total Trauma	.39***					
(3) General Traumas	.27***	.74***				
(4) Physical Abuse	.13*	.67***	.31***			
(5) Emotional Abuse	.40***	.90***	.54***	.44***		
(6) Sexual Abuse	.25***	.45***	.21***	.20**	.30***	
(7) Recent Negative Events	.34***	.25***	.18**	.14*	.25***	.09

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

It was of interest to determine the mediating influence of recent negative events and resilience on the relationship between early life stress and depression. Multiple mediation analyses were conducted using bootstrapping procedures and confidence intervals based on 5000 iterations (Preacher & Hayes, 2008). These analyses revealed that total early life stress was positively correlated with depressive symptomatology and that this relationship was mediated by both reported recent negative events (95% CI: 0.01, 0.05) and resilience (95% CI: 0.02, 0.07) as shown in Figure 1.

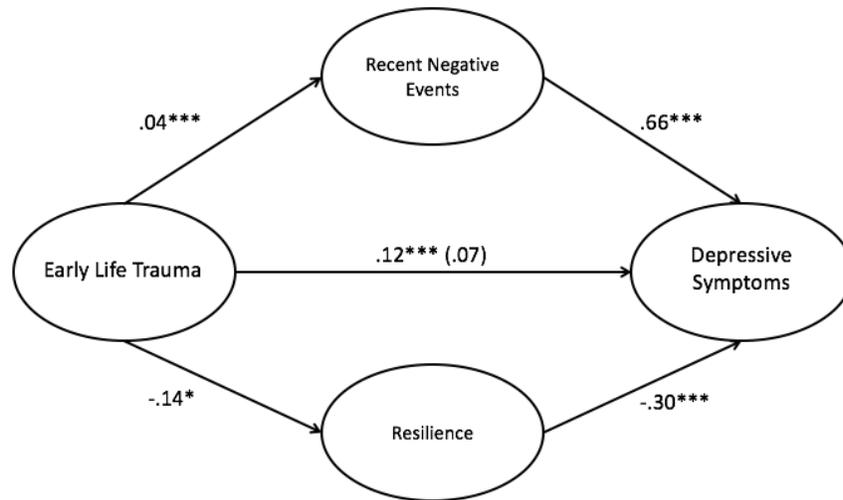


Figure 1. Schematics of the multiple mediation analysis examining the mediating role of recent negative events and resilience in the relationship between total early life trauma and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

To further examine these relationships, the same mediation analysis was conducted separately for each of the four types of early life trauma. As shown in Figure 2, general trauma, emotional abuse, and sexual abuse were significantly correlated with depressive symptoms. Recent negative events, but not resilience, mediated the link between general traumas and physical abuse with depressive symptoms [(95% CI: 0.02, 0.15) (95% CI: 0.005, 0.16), respectively], whereas resilience, but not recent negative events, mediated the relationship between sexual abuse and depressive symptomatology (95% CI: 0.14, 0.62). By contrast, both recent negative events and resilience partially mediated the relationship between early life emotional abuse with depressive symptoms [(95% CI:0.02, 0.10) (95% CI: 0.04, 0.14), respectively].

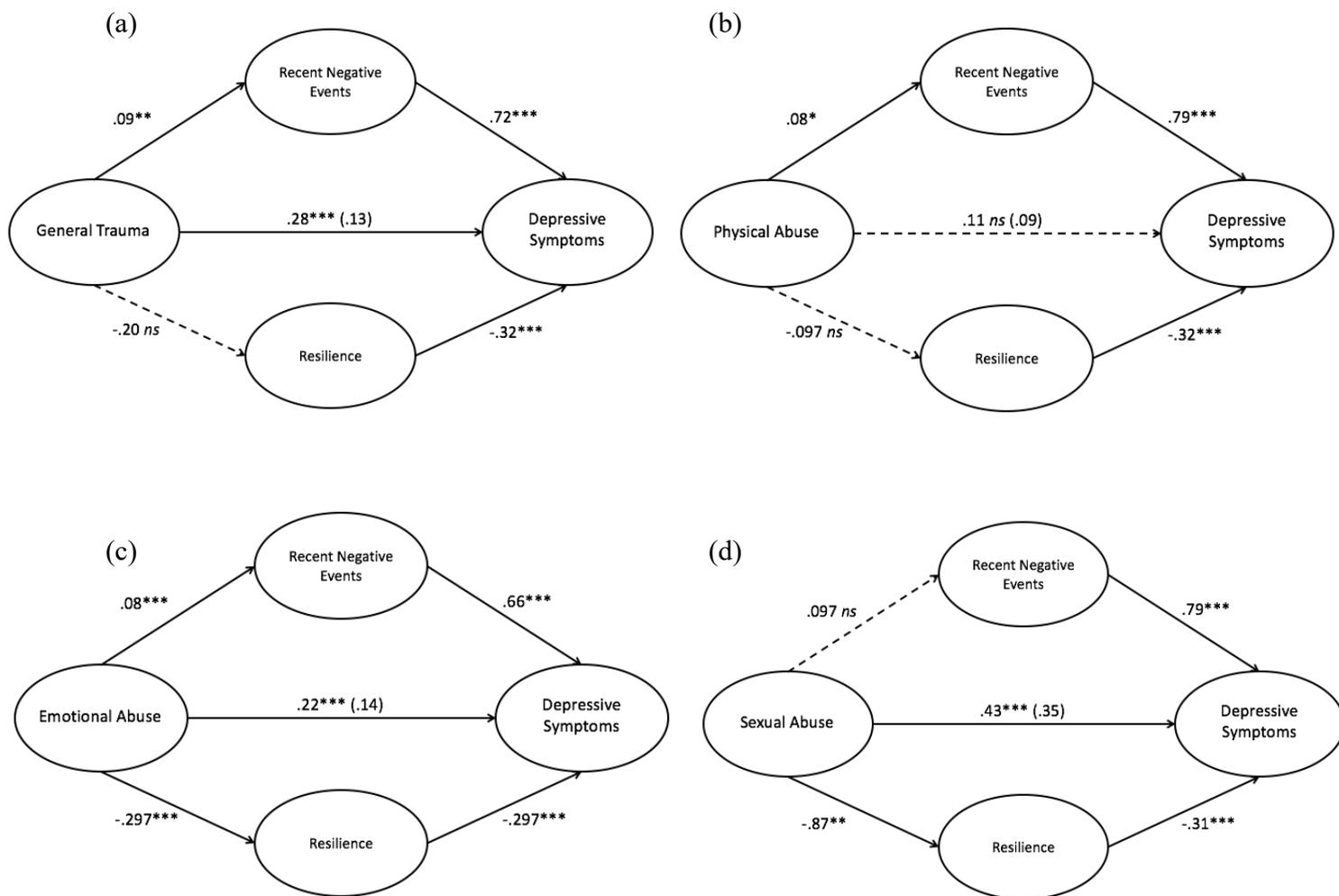


Figure 2. Schematics of the multiple mediation analyses examining the mediating role of recent negative events and resilience in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Once more, the same statistical method was employed to examine the mediating role of cognitive flexibility in the link between early life stress and depression. As predicted, cognitive flexibility was negatively correlated with total early life trauma, and was a significant mediator between its link with depressive symptomatology (95% CI: 0.10, 0.20), shown in Figure 3. Using the same approach, mediation analyses were conducted for each type of trauma, shown in Figure

4. As in the previous analyses, cognitive flexibility significantly mediated the relationship between both emotional and sexual abuse with depression [(95% CI: 0.02, 0.15), (95% CI: 0.08, 0.52), respectively], while it was not a significant mediator for the link between depression and general trauma or physical abuse. The BDNF polymorphism did not act as a moderator in this relationship.

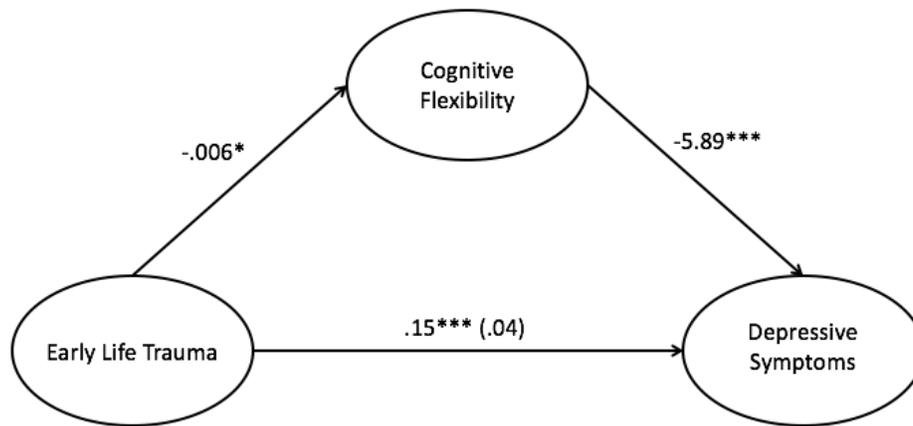


Figure 3. Schematic of the mediation analysis examining the mediating role of cognitive flexibility in the relationship between total early life trauma and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

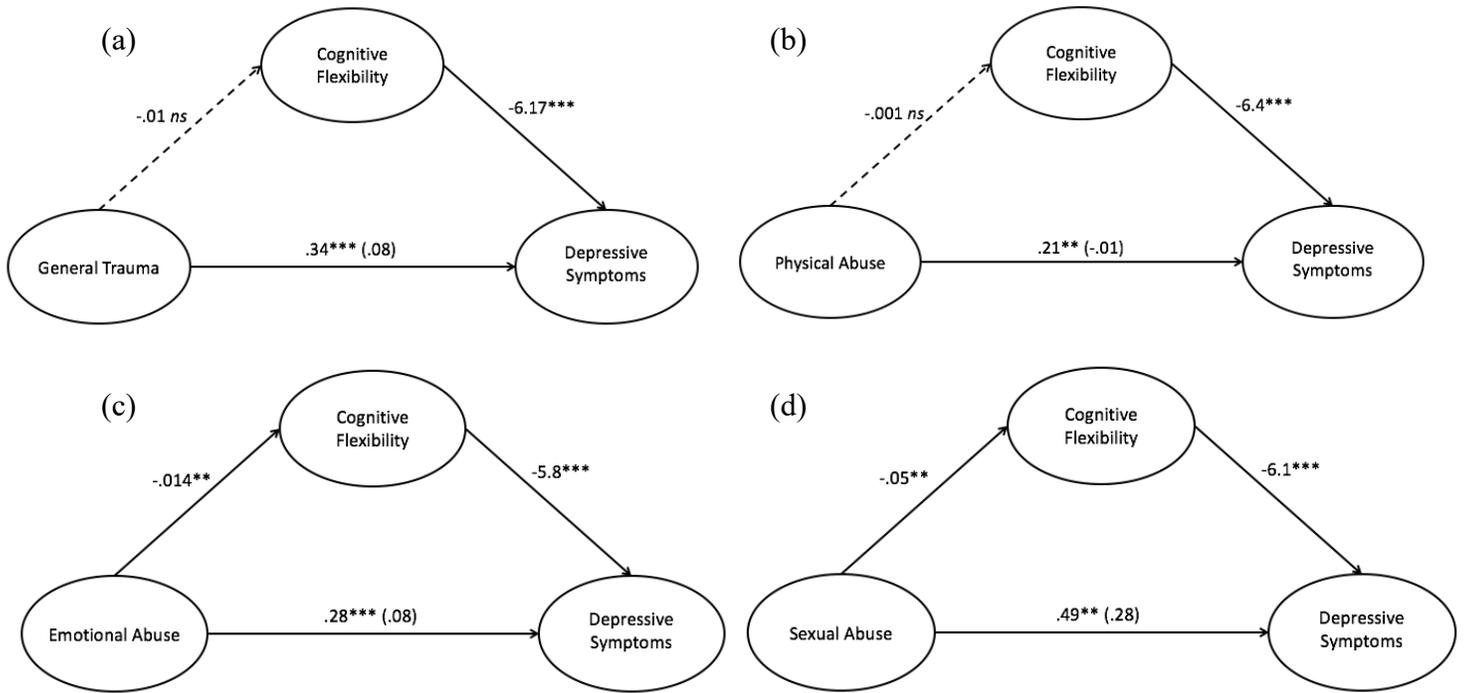


Figure 4. Schematics of the mediation analyses examining the mediating role of cognitive flexibility in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Finally, multiple mediation analyses were conducted to examine the mediating role of different coping strategies (emotion-focused, problem-focused, and avoidant) in the link between early life stress and depression. When all four types of trauma were considered together, only emotion-focused coping was a significant mediator in this relationship (95% CI: 0.03, 0.08), as seen in Figure 5. The BDNF polymorphism did not moderate this relationship.

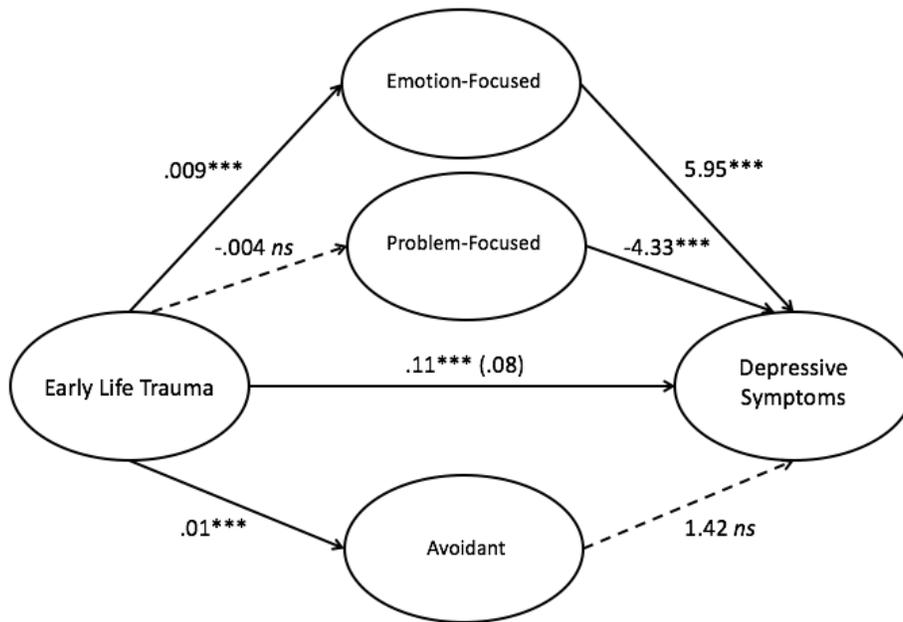


Figure 5. Schematic of the multiple mediation analysis examining the mediating role of different coping strategies (emotion-focused, problem-focused, and avoidant) in the relationship between total early life trauma and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Once again, this relationship was examined by separately analysing each of the four types of early life trauma. As seen in Figure 6, in the case of general trauma, only emotion-focused coping was a significant mediator in the link with depressive symptoms (95% CI: 0.04, 0.21), whereas for emotional abuse, all three types of coping were significantly mediators of this relationship [(95% CI: 0.06, 0.16), (95% CI: 0.008, 0.069), (95% CI: 0.003, 0.058) respectively]. None of the coping styles were significant mediators in the link between physical abuse or sexual abuse with depression.

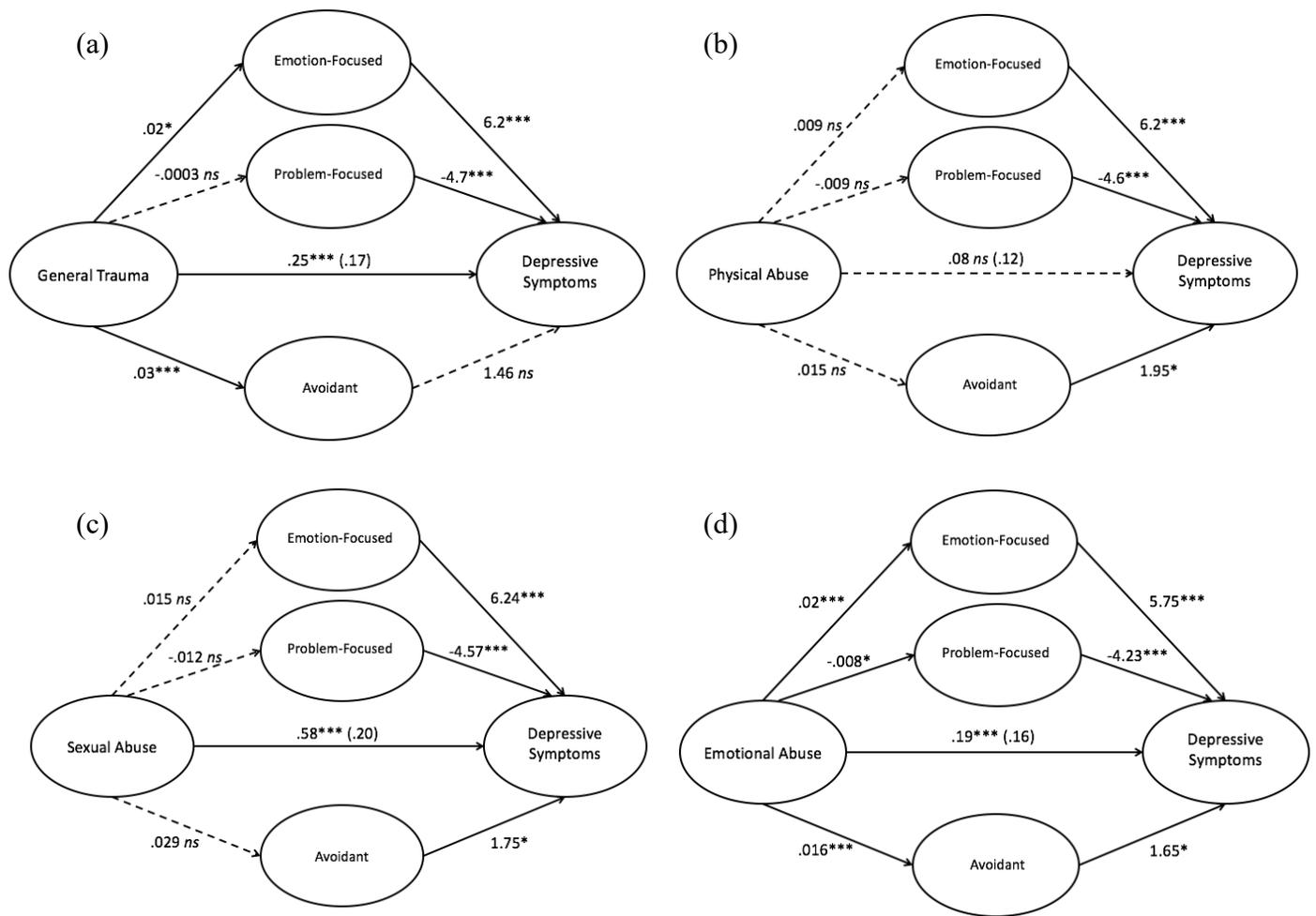


Figure 6. Schematics of the multiple mediation analyses examining the mediating role of emotion-focused, problem-focused, and avoidant coping strategies in the relationship between (a) general trauma, (b) physical abuse, (c) emotional abuse, and (d) sexual abuse and depression. (beta values are provided along each line) Note * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Discussion

Consistent with earlier reports (Kendler et al., 1999; Kendler et al., 2004; van Loo et al., 2015), a strong linkage was found between early life trauma and depressive symptoms. Although it has been well established that early life stressful events are tied to the occurrence of depressive symptoms, as in the present investigation, most studies involved self-report measures, which were of a retrospective nature, and thus were subject to the common biases associated with these procedures (Colman et al., 2016, Schraedley et al., 2002). Nonetheless, in the context of other

reports of a prospective nature, these results showed the same profile (Colman et al., 2014; Kendler et al., 1999). Importantly, what distinguishes this study from other experiments is that not all trauma experiences seem to be linked to depression in the same fashion. For instance, depressive symptoms were directly related to earlier emotional abuse, and to a somewhat lesser extent, general trauma, sexual abuse, and physical abuse. Whether this is reflective of the severity or the age at which they occurred cannot be known from the present study. Furthermore, consistent with other findings (Caldwell et al., 2013; Matheson & Anisman, 2003; Nolen-Hoeksema, 1991), in the present investigation, increased emotion-focus coping and avoidant coping were both significantly correlated with depressive symptoms, whereas problem-focused coping was negatively associated with depressive symptomatology.

It has been maintained that early life trauma alters an individual's developmental trajectory so that they will be more likely to encounter further trauma, and that these re-traumatizations may favour the evolution of depression (Banyard et al., 2003; Liu, 2013). In line with these findings, the relationship between early life experiences and depression was mediated by the combination of recent negative experiences and the individual's resilience. It will be recalled, resilience refers to an individual's hardiness against illness, or the ability to "bounce back" after an illness or hardship has been experienced, and is associated with decreased incidence and severity of depressive symptomatology (Charney, 2004; Haslam et al., 2015; Haslam et al., 2016). Moreover, resilience factors such as social support, problem-solving skills, and positive early-life experiences, are those that promote well-being and contribute to better health outcomes. By contrast, lower levels of resilience or loss of resilience factors may contribute to the onset of depression (Seymour-Smith et al., 2016). As expected, recent stressful events partially mediated the link between negative early life experience and adult depression.

However, the mediating role of recent negative events was dependent upon the nature of the early life trauma experienced. In this regard, recent events acted as a mediator between each of general trauma, physical abuse, and emotional abuse with self-reported depression, but not with sexual abuse. This was unexpected, as there was no reason to anticipate that the link between sexual abuse and depression would not be mediated by recent events. In essence, early life sexual abuse was directly tied to adult depression, and this occurred irrespective of subsequent adverse events. This should not be taken to imply that sexual abuse was a more potent predictor of later depression, as emotional abuse was actually most highly related to depression. As with recent negative events, the mediating role of resilience was dependent on the nature of the early life trauma experienced. Specifically, resilience mediated the relationship between both emotional abuse and sexual abuse, but not general trauma or physical abuse with depression. Why this is the case is unclear, particularly considering the broad age range in which these stressors were measured (age 0 – 12) and the low number and severity of reported events overall.

Consistent with the view that coping styles are important in predicting depression, both problem-focused and emotion-focused coping were linked to depressive symptoms, whereas this relationship was not as strong in relation to avoidant coping. Early life trauma was similarly linked to emotion-focused as well as avoidant coping, but not to problem-focused efforts. These findings are largely consistent with earlier studies (Matheson & Anisman, 2012), although in some reports, problem-focused coping was also associated with depressive symptomatology. It appeared that the link between early life trauma and depressive symptoms was partially mediated by these three coping styles (emotion-focused, problem-focused, avoidant), with the most pronounced contribution coming from emotion-focused coping. In a sense, the coping styles or strategies that would be most effective in dealing with stressors are those that allow or encourage

flexibility in how situations are appraised or dealt with (Cheng et al., 2014; Lam & McBride-Chang, 2007; Roussi et al., 2007).

Cognitive flexibility, which refers to an individual's ability/propensity to shift from one strategy or another ("cognitive sets") as the situation demands (Young et al., 2001), was found to be a strong mediator between trauma experiences and the later emergence of depression. Of particular significance was that the link between total early life trauma and depressive symptoms operated through cognitive flexibility, as did the link between both emotional and sexual abuse in relation to depressive symptoms. Cognitive rigidity, by contrast, is often observed in depressed individuals (Austin et al., 2001; Grant et al., 2001; Trivedi & Greer, 2014), and may impede appropriate appraisals and coping processes by promoting the perseverance of maladaptive beliefs and automatic negative thoughts (Fossati et al., 2001; Moore, 1996; Teasdale et al., 1995).

It has been said that appraisals are fundamental in relation to depression (Folkman et al., 1986; Lazarus, 1966), and while not denying this, the present investigation focused on coping without evaluating appraisals. Changes of appraisal processes could, no doubt, affect coping, and could enact a chain of events wherein variations of personality factors, such as self-esteem and identity, could favor the development of depressed mood.

The neurotrophin BDNF has been implicated in the development of depression (Duman & Monteggia, 2006; Schmidt & Duman, 2007; Sen et al., 2008), thus it was expected that a BDNF polymorphism would be linked to depressed mood, but this was not observed in this study. Although the original reports seem to be impressive, increasingly there have been questions concerning the BDNF-depression link. Among other things, there have been a number of studies assessing BDNF in blood, but a link between blood and brain BDNF wasn't reliably

observed (Kyeremanteng et al., 2012). This aside, changes of BDNF within the blood doesn't necessarily imply that serum BDNF reflects functioning in the specific brain regions (e.g. hippocampus, prefrontal cortex) thought to be fundamental in subserving depressive symptoms. Besides, even if BDNF were associated with depression, it might operate in conjunction with several other factors, including other neurotrophins. Moreover, there is some debate as to whether a BDNF polymorphism acts as a vulnerability or resilience factor for depressive illness (Verhagen et al., 2010). It has been suggested that the Met allele is associated with depression owing to reduced hippocampal activity (Chen et al., 2004; Egan et al., 2003), and might also be tied to poor episodic memory, a common symptom among depressed individuals (Veiel, 1997). By contrast, the Val allele has been associated with yet other risk factors for depression, such as trait anxiety and neuroticism (Lang et al., 2005; Sen et al., 2003), and has also been linked to childhood-onset mood disorders (Strauss et al., 2005). Other investigations, however, have failed to show any linkage between the Val66Met polymorphism and depression (Cohen et al., 2004; Oswald et al., 2005; Schumacher et al., 2005; Surtees et al., 2007). Indeed, a meta-analysis in this line of inquiry found no significant link between the Val66Met polymorphism and depression when including all ethnicities and genders, however a gender-stratified analysis revealed that the Met allele was significantly linked to depression in men only (Verhagen et al., 2010). The small sample size and over-representation of females in the current investigation would not yield the appropriate statistical power to warrant this analysis.

The present investigation had a number of limitations which should be taken into consideration. As previously described, the measures used to assess childhood trauma were retrospective in nature, and are therefore subject to the common biases of these procedures. Measures of depressive symptoms were also unable to distinguish between different depressive

subtypes, which are likely underpinned by different actions and systems. Additionally, the sample size was modest, especially considering the number of individuals who carried the Met allele, which further impacted the present findings as the small number of polymorphism-carrying individuals meant that Val/Met and Met/Met individuals had to be evaluated together, despite possible differences within these two groups. Furthermore, the amount of childhood trauma observed in the current sample (mainly consisting of non-clinically depressed university students) is relatively low, and perhaps not well representative of the population at large. Moreover, the over-representation of females in this sample did not allow for the analysis of certain sex-differences that likely exist within this group.

In summary, the present findings are consistent with the view that several forms of early life trauma were linked to depressive symptoms, operating through general resilience, recent negative experiences, the way in which individuals coped with the trauma experience, and their cognitive flexibility, which can be considered a form of coping that facilitates the development of different coping mechanisms. Although it had been expected that at least some of these relationships, particularly resilience and coping style, would be moderated by a BDNF polymorphism, this was actually not observed; nevertheless, it would be premature to dismiss a role for BDNF in the link between early life negative experiences and later depression.

References

- Alexopoulos, G. S., Meyers, B. S., Young, R. C., Kalayam, B., Kakuma, T., Gabrielle, M., & Hull, J. (2000). Executive dysfunction and long-term outcomes of geriatric depression. *Archives of general psychiatry*, *57*(3), 285-290.
- Anisman, H. (2014). *An introduction to Stress and Health*. Sage.
- Anisman, H., Hayley, S., & Merali, Z. (2003). Sensitization associated with stressors and cytokine treatments. *Brain, Behaviour, and Immunity*, *17*, 86-93.
- Anisman, H., Merali, Z., & Hayley, S. (2008). Neurotransmitter, peptide and cytokine processes in relation to depressive disorder: comorbidity between depression and neurodegenerative disorders. *Progress in neurobiology*, *85*(1), 1-74.
- Anisman, H., Ravindran, A., Griffiths, J., & Merali, Z. (1999). Endocrine and cytokine correlates of major depression and dysthymia with typical or atypical. *Molecular psychiatry*, *4*, 182-188.
- Austin, M. P., Mitchell, P., & Goodwin, G. M. (2001). Cognitive deficits in depression. *The British Journal of Psychiatry*, *178*(3), 200-206.
- Banyard, V. L., Williams, L. M., & Siegel, J. A. (2003). Retraumatization among adult women sexually abused in childhood: Exploratory analyses in a prospective study. *Journal of child sexual abuse*, *11*(3), 19-48.
- Bath, K. G., & Lee, F. S. (2006). Variant BDNF (Val66Met) impact on brain structure and function. *Cognitive, Affective, & Behavioral Neuroscience*, *6*(1), 79-85.
- Beck, A. T. (1972). Measuring depression: The depression inventory. *Recent advances in the psychobiology of the depressive illnesses*, 299-302.

- Bremner, J. D., Vermetten, E., & Mazure, C. M. (2000). Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: the Early Trauma Inventory. *Depression and anxiety, 12*(1), 1-12.
- Caldwell, W., McInnis, O. A., McQuaid, R. J., Liu, G., Stead, J. D., Anisman, H., & Hayley, S. (2013). The role of the Val66Met polymorphism of the brain derived neurotrophic factor gene in coping strategies relevant to depressive symptoms. *PLoS One, 8*(6), e65547.
- Cargill, M., Altshuler, D., Ireland, J., Sklar, P., Ardlie, K., Patil, N., & Ziaugra, L. (1999). Characterization of single-nucleotide polymorphisms in coding regions of human genes. *Nature genetics, 22*(3), 231-238.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386-389.
- Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability: implications for successful adaptation to extreme stress. *Am. J. Psychiatry, 161* (2004), pp. 195–216
- Chen, Z. Y., Patel, P. D., Sant, G., Meng, C. X., Teng, K. K., Hempstead, B. L., & Lee, F. S. (2004). Variant brain-derived neurotrophic factor (BDNF)(Met66) alters the intracellular trafficking and activity-dependent secretion of wild-type BDNF in neurosecretory cells and cortical neurons. *Journal of Neuroscience, 24*(18), 4401-4411.
- Cheng, C., Lau, H. P. B., & Chan, M. P. S. (2014). Coping flexibility and psychological adjustment to stressful life changes: A meta-analytic review.

- Chepenik, L. G., Fredericks, C., Papademetris, X., Spencer, L., Lacadie, C., Wang, F., Pittman, B., Duncan, J.S., Staib, L.H., Duman, R.S., & Gelernter, J. (2009). Effects of the brain-derived neurotrophic growth factor val66met variation on hippocampus morphology in bipolar disorder. *Neuropsychopharmacology*, *34*(4), 944-951
- Choi, M. J., Kang, R. H., Lim, S. W., Oh, K. S., & Lee, M. S. (2006). Brain-derived neurotrophic factor gene polymorphism (Val66Met) and citalopram response in major depressive disorder. *Brain research*, *1118*(1), 176-182.
- Cohen, S., Rosa, A., Corsico, A., Sterne, A., Owen, M., Korzsun, A., & McGuffin, P. (2004). The brain derived neurotrophic factor (BDNF) Val66Met polymorphism and recurrent unipolar depression. In *American Journal of Medical Genetics*. 130: 37 – 38.
- Colman, I., Jones, P. B., Kuh, D., Weeks, M., Naicker, K., Richards, M., & Croudace, T. J. (2014). Early development, stress and depression across the life course: pathways to depression in a national British birth cohort. *Psychological medicine*, *44*(13), 2845-2854.
- Colman, I., Kingsbury, M., Garad, Y., Zeng, Y., Naicker, K., Patten, S., Jones, P.B., Wild, T.C., & Thompson, A. H. (2016). Consistency in adult reporting of adverse childhood experiences. *Psychological medicine*, *46*(03), 543-549.
- Conference Board of Canada (2016). Healthy Brains at Work: Employer-Sponsored Mental Health Benefits and Programs.
- Connor, K. M., & Davidson, J. R. (2003). Development of a new resilience scale: The Connor-Davidson resilience scale (CD-RISC). *Depression and anxiety*, *18*(2), 76-82.
- Cooper, C., Katona, C., Orrell, M., & Livingston, G. (2008). Coping strategies, anxiety and depression in caregivers of people with Alzheimer's disease. *International journal of geriatric psychiatry*, *23*(9), 929-936.

- Cruz-Fuentes, C. S., Benjet, C., Martínez-Levy, G. A., Pérez-Molina, A., Briones-Velasco, M., & Suárez-González, J. (2014). BDNF Met66 modulates the cumulative effect of psychosocial childhood adversities on major depression in adolescents. *Brain and behavior, 4*(2), 290-297.
- Dennis, J. P., & Vander Wal, J. S. (2010). The cognitive flexibility inventory: Instrument development and estimates of reliability and validity. *Cognitive therapy and research, 34*(3), 241-253.
- De Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: from adaptation to disease. *Nature Reviews Neuroscience, 6*(6), 463-475.
- Duman, R. S., Malberg, J., & Thome, J. (1999). Neural plasticity to stress and antidepressant treatment. *Biological psychiatry, 46*(9), 1181-1191.
- Duman, R. S., Li, N., Liu, R. J., Duric, V., & Aghajanian, G. (2012). Signaling pathways underlying the rapid antidepressant actions of ketamine. *Neuropharmacology, 62*(1), 35-41.
- Duman, R. S., & Monteggia, L. M. (2006). A neurotrophic model for stress-related mood disorders. *Biological psychiatry, 59*(12), 1116-1127.
- Dumont, M., & Provost, M. A. (1999). Resilience in adolescents: Protective role of social support, coping strategies, self-esteem, and social activities on experience of stress and depression. *Journal of youth and adolescence, 28*(3), 343-363.
- Egan, M. F., Kojima, M., Callicott, J. H., Goldberg, T. E., Kolachana, B. S., Bertolino, A., & Lu, B. (2003). The BDNF val66met polymorphism affects activity-dependent secretion of BDNF and human memory and hippocampal function. *Cell, 112*(2), 257-269.

- Farhang, S., Barar, J., Fakhari, A., Mesgariabbasi, M., Khani, S., Omid, Y., & Farnam, A. (2014). Asymmetrical expression of BDNF and NTRK3 genes in frontoparietal cortex of stress-resilient rats in an animal model of depression. *Synapse*, 68(9), 387-393.
- Foley, D. L., Neale, M. C., & Kendler, K. S. (1996). A longitudinal study of stressful life events assessed at interview with an epidemiological sample of adult twins: the basis of individual variation in event exposure. *Psychological Medicine*, 26(06), 1239-1252.
- Folkman, S. (2013). *Stress: appraisal and coping* (pp. 1913-1915). Springer New York.
- Folkman, S., Lazarus, R. S., Gruen, R. J., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of personality and social psychology*, 50(3), 571.
- Fossati, P., Ergis, A. M., & Allilaire, J. F. (2001). Executive functioning in unipolar depression: a review. *L'encéphale*, 28(2), 97-107.
- Galatzer-Levy, I. R., Burton, C. L., & Bonanno, G. A. (2012). Coping flexibility, potentially traumatic life events, and resilience: A prospective study of college student adjustment. *Journal of Social and Clinical Psychology*, 31(6), 542-567.
- Gonul, A. S., Akdeniz, F., Taneli, F., Donat, O., Eker, Ç., & Vahip, S. (2005). Effect of treatment on serum brain-derived neurotrophic factor levels in depressed patients. *European archives of psychiatry and clinical neuroscience*, 255(6), 381-386.
- Grant, M. M., Thase, M. E., & Sweeney, J. A. (2001). Cognitive disturbance in outpatient depressed younger adults: evidence of modest impairment. *Biological psychiatry*, 50(1), 35-43.
- Gupta, R., Gupta, K., Tripathi, A. K., Bhatia, M. S., & Gupta, L. K. (2016). Effect of mirtazapine treatment on serum levels of brain-derived neurotrophic factor and tumor necrosis factor-

- α in patients of major depressive disorder with severe depression. *Pharmacology*, 97(3-4), 184-188.
- Hammen, C. (2005). Stress and depression. *Annu. Rev. Clin. Psychol.*, 1, 293-319.
- Haslam, C., Cruwys, T., Haslam, S. A., Dingle, G., & Chang, M. X. L. (2016). Groups 4 Health: Evidence that a social-identity intervention that builds and strengthens social group membership improves mental health. *Journal of affective disorders*, 194, 188-195.
- Haslam, C., Cruwys, T., Milne, M., Kan, C. H., & Haslam, S. A. (2015). Group ties protect cognitive health by promoting social identification and social support. *Journal of aging and health*, 0898264315589578.
- Hollon, S. D., DeRubeis, R. J., & Evans, M. D. (1996). Cognitive therapy in the treatment and prevention of depression. In P. M. Salkovskis (Ed.), *Frontiers of cognitive therapy* (pp. 293–317). New York: Guilford Press.
- Itoh, K., Hashimoto, K., Kumakiri, C., Shimizu, E., & Iyo, M. (2004). Association between brain-derived neurotrophic factor 196 G/A polymorphism and personality traits in healthy subjects. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 124(1), 61-63.
- Jiang, X., Xu, K., Hoberman, J., Tian, F., Marko, A. J., Waheed, J. F., Harris, C. R., Marini, A. M., Enoch, M-A., & Lipsky, R. H. (2005). BDNF variation and mood disorders: a novel functional promoter polymorphism and Val66Met are associated with anxiety but have opposing effects. *Neuropsychopharmacology*, 30(7), 1353.
- Johnco, C., Wuthrich, V. M., & Rapee, R. M. (2013). The role of cognitive flexibility in cognitive restructuring skill acquisition among older adults. *Journal of anxiety disorders*, 27(6), 576-584.

- Kang, J. I., Kim, S. J., Song, Y. Y., Namkoong, K., & An, S. K. (2013). Genetic influence of COMT and BDNF gene polymorphisms on resilience in healthy college students. *Neuropsychobiology*, *68*(3), 174-180.
- Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry*.
- Kendler, K. S., Kuhn, J. W., & Prescott, C. A. (2004). Childhood sexual abuse, stressful life events and risk for major depression in women. *Psychological medicine*, *34*(8), 1475.
- Kyeremanteng, C., James, J., Mackay, J. & Merali, Z. (2012). A study of brain and serum brain-derived neurotrophic factor protein in Wistar and Wistar-Kyoto rat strains after electroconvulsive stimulus. *Pharmacopsychiatry*, *45*, 244-249.
- Kim, T. Y., Kim, S. J., Chung, H. G., Choi, J. H., Kim, S. H., & Kang, J. I. (2017). Epigenetic alterations of the BDNF gene in combat-related post-traumatic stress disorder. *Acta Psychiatrica Scandinavica*, *135*(2), 170-179.
- La Greca, A. M., Lai, B. S., Joormann, J., Auslander, B. B., & Short, M. A. (2013). Children's risk and resilience following a natural disaster: Genetic vulnerability, posttraumatic stress, and depression. *Journal of affective disorders*, *151*(3), 860-867.
- Lam, C. B., & McBride-Chang, C. A. (2007). Resilience in young adulthood: The moderating influences of gender-related personality traits and coping flexibility. *Sex Roles*, *56*(3-4), 159-172.
- Lang, U. E., Hellweg, R., Kalus, P., Bajbouj, M., Lenzen, K. P., Sander, T., Kunz, D., & Gallinat, J. (2005). Association of a functional BDNF polymorphism and anxiety-related personality traits. *Psychopharmacology*, *180*(1), 95-99.

- Larsen, M. H., Mikkelsen, J. D., Hay-Schmidt, A., & Sandi, C. (2010). Regulation of brain-derived neurotrophic factor (BDNF) in the chronic unpredictable stress rat model and the effects of chronic antidepressant treatment. *Journal of psychiatric research, 44*(13), 808-816.
- Laske, C., & Eschweiler, G. W. (2006). Brain-derived neurotrophic factor. *Der Nervenarzt, 77*(5), 523-537.
- Lazarus, R. S. (1966). Psychological stress and the coping process.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer publishing company.
- Lepack, A. E., Fuchikami, M., Dwyer, J. M., Banasr, M., & Duman, R. S. (2015). BDNF release is required for the behavioral actions of ketamine. *International Journal of Neuropsychopharmacology, 18*(1), pyu033.
- Liu, R. T. (2013). Stress generation: Future directions and clinical implications. *Clinical Psychology Review, 33*(3), 406-416.
- Liu, R. T., & Alloy, L. B. (2010). Stress generation in depression: A systematic review of the empirical literature and recommendations for future study. *Clinical psychology review, 30*(5), 582-593.
- Liu, R. J., Lee, F. S., Li, X. Y., Bambico, F., Duman, R. S., & Aghajanian, G. K. (2012). Brain-derived neurotrophic factor Val66Met allele impairs basal and ketamine-stimulated synaptogenesis in prefrontal cortex. *Biological psychiatry, 71*(11), 996-1005.
- Lu, B., & Gottschalk, W. (2000). Modulation of hippocampal synaptic transmission and plasticity by neurotrophins. *Progress in brain research, 128*, 231.

- Lyons, D. M., & Parker, K. J. (2007). Stress inoculation-induced indications of resilience in monkeys. *Journal of traumatic stress, 20*(4), 423-433.
- Matheson, K., & Anisman, H. (2003). Systems of coping associated with dysphoria, anxiety and depressive illness: a multivariate profile perspective. *Stress, 6*(3), 223-234.
- Matheson, K., & Anisman, H. (2012). Biological and psychosocial responses to discrimination. *The social cure: Identity, health and well-being, 133-153*.
- Mazure, C. M. (1998). Life stressors as risk factors in depression. *Clinical Psychology: Science and Practice, 5*(3), 291-313.
- Morgan, C. A., Rasmusson, A. M., Wang, S., Hoyt, G., Hauger, R. L., & Hazlett, G. (2002). Neuropeptide-Y, cortisol, and subjective distress in humans exposed to acute stress: replication and extension of previous report. *Biological psychiatry, 52*(2), 136-142.
- Morgan, C. A., Wang, S., Southwick, S. M., Rasmusson, A., Hazlett, G., Hauger, R. L., & Charney, D. S. (2000). Plasma neuropeptide-Y concentrations in humans exposed to military survival training. *Biological psychiatry, 47*(10), 902-909.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of abnormal psychology, 100*(4), 569.
- Oswald, P., Del-Favero, J., Massat, I., Souery, D., Claes, S., Van Broeckhoven, C., & Mendlewicz, J. (2005). No implication of brain-derived neurotrophic factor (BDNF) gene in unipolar affective disorder: Evidence from Belgian first and replication patient-control studies. *European neuropsychopharmacology, 15*(5), 491-495.
- Paradiso, S., Lamberty, G. J., Garvey, M. J., & Robinson, R. G. (1997). Cognitive impairment in the euthymic phase of chronic unipolar depression. *The Journal of nervous and mental disease, 185*(12), 748-754.

- Parker, K. J., Buckmaster, C. L., Sundlass, K., Schatzberg, A. F., & Lyons, D. M. (2006). Maternal mediation, stress inoculation, and the development of neuroendocrine stress resistance in primates. *Proceedings of the National Academy of Sciences of the United States of America*, *103*(8), 3000-3005.
- Park, H., & Poo, M. M. (2013). Neurotrophin regulation of neural circuit development and function. *Nature Reviews Neuroscience*, *14*(1), 7-23.
- Perea, C. S., Paternina, A. C., Gomez, Y., & Lattig, M. C. (2012). Negative affectivity moderated by BDNF and stress response. *Journal of affective disorders*, *136*(3), 767-774.
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior research methods*, *40*(3), 879-891.
- Ravindran, A. V., Griffiths, J., Waddell, C., & Anisman, H. (1995). Stressful life events and coping styles in relation to dysthymia and major depressive disorder: variations associated with alleviation of symptoms following pharmacotherapy. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *19*(4), 637-653.
- Ricci, V., Pomponi, M., Martinotti, G., Bentivoglio, A., Loria, G., Bernardini, S., & Angelucci, F. (2010). Antidepressant treatment restores brain-derived neurotrophic factor serum levels and ameliorates motor function in Parkinson disease patients. *Journal of clinical psychopharmacology*, *30*(6), 751-753.
- Ricken, R., Adli, M., Lange, C., Krusche, E., Stamm, T. J., Gaus, S., Koehler, S., Nase, S., Bschor, T., Richter, C., Steinacher, B., Heinz, A., Rapp M.A., Borgwadt, S., Hellweg, R., & Lang, U.E. (2013). Brain-derived neurotrophic factor serum concentrations in acute

- depressive patients increase during lithium augmentation of antidepressants. *Journal of clinical psychopharmacology*, 33(6), 806-809.
- Rotermann, M., Sanmartin, C., Hennessy, D., & Arthur, M. (2014). Prescription medication use by Canadians aged 6 to 79. *Health reports*, 25(6), 3.
- Roussi, P., Krikeli, V., Hatzidimitriou, C., & Koutri, I. (2007). Patterns of coping, flexibility in coping and psychological distress in women diagnosed with breast cancer. *Cognitive Therapy and Research*, 31(1), 97-109.
- Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: development of the Life Experiences Survey. *Journal of consulting and clinical psychology*, 46(5), 932.
- Schmidt, H. D., & Duman, R. S. (2007). The role of neurotrophic factors in adult hippocampal neurogenesis, antidepressant treatments and animal models of depressive-like behavior. *Behavioural pharmacology*, 18(5-6), 391-418.
- Schraedley, P. K., Turner, R. J., & Gotlib, I. H. (2002). Stability of retrospective reports in depression: traumatic events, past depressive episodes, and parental psychopathology. *Journal of Health and Social Behavior*, 307-316.
- Schumacher, J., Jamra, R. A., Becker, T., Ohlraun, S., Klopp, N., Binder, E. B., Schulze, T.G., Deschner, M., Schmal, C., Hofels, S., & Zobel, A. (2005). Evidence for a relationship between genetic variants at the brain-derived neurotrophic factor (BDNF) locus and major depression. *Biological psychiatry*, 58(4), 307-314.
- Sen, S., Nesse, R. M., Stoltenberg, S. F., Li, S., Gleiberman, L., Chakravarti, A., Weber, A.B., & Burmeister, M. (2003). A BDNF coding variant is associated with the NEO personality inventory domain neuroticism, a risk factor for depression.

- Sen, S., Duman, R., & Sanacora, G. (2008). Serum brain-derived neurotrophic factor, depression, and antidepressant medications: meta-analyses and implications. *Biological psychiatry*, 64(6), 527-532.
- Seymour-Smith, M., Cruwys, T., Haslam, S. A., & Brodribb, W. (2016). Loss of group memberships predicts depression in postpartum mothers. *Social Psychiatry and Psychiatric Epidemiology*, 1-10.
- Shaikh, B. T., Kahloon, A., Kazmi, M., Khalid, H., Nawaz, K., Khan, N., & Khan, S. (2004). Students, stress and coping strategies: a case of Pakistani medical school. *Education for Health-Abingdon-Carfax Publishing Limited*, 17, 346-353.
- Shimizu, E., Hashimoto, K., & Iyo, M. (2004). Ethnic difference of the BDNF 196G/A (val66met) polymorphism frequencies: the possibility to explain ethnic mental traits. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 126(1), 122-123.
- Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *Jama*, 301(21), 2252-2259.
- Statistics Canada (2012). Canadian Community Health Survey: Mental Health 2012.
- Strauss, J., Barr, C. L., George, C. J., Devlin, B., Vetro, A., Kiss, E., Baji, I., King, N., Shaikh, S., Lanktree, M., & Kovacs, M. (2005). Brain-derived neurotrophic factor variants are associated with childhood-onset mood disorder: confirmation in a Hungarian sample. *Molecular psychiatry*, 10(9), 861-867.
- Surtees, P. G., Wainwright, N. W., Willis-Owen, S. A., Sandhu, M. S., Luben, R., Day, N. E., & Flint, J. (2007). No association between the BDNF Val66Met polymorphism and mood

- status in a non-clinical community sample of 7389 older adults. *Journal of psychiatric research*, 41(5), 404-409.
- Taliaz, D., Loya, A., Gersner, R., Haramati, S., Chen, A., & Zangen, A. (2011). Resilience to chronic stress is mediated by hippocampal brain-derived neurotrophic factor. *Journal of Neuroscience*, 31(12), 4475-4483.
- Teasdale, J. D., Segal, Z., & Williams, J. M. G. (1995). How does cognitive therapy prevent depressive relapse and why should attentional control (mindfulness) training help?. *Behaviour Research and therapy*, 33(1), 25-39.
- Trivedi, M. H., & Greer, T. L. (2014). Cognitive dysfunction in unipolar depression: implications for treatment. *Journal of affective disorders*, 152, 19-27.
- Tsoory, M., Cohen, H., & Richter-Levin, G. (2007). Juvenile stress induces a predisposition to either anxiety or depressive-like symptoms following stress in adulthood. *European Neuropsychopharmacology*, 17(4), 245-256.
- Uchida, S., Hara, K., Kobayashi, A., Funato, H., Hobara, T., Otsuki, K., & Watanabe, Y. (2010). Early life stress enhances behavioral vulnerability to stress through the activation of REST4-mediated gene transcription in the medial prefrontal cortex of rodents. *Journal of Neuroscience*, 30(45), 15007-15018.
- 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 Winkel, M., Peeters, F., van Winkel, R., Kenis, G., Collip, D., Geschwind, N., & Myin-Germeys, I. (2014). Impact of variation in the BDNF gene on social stress sensitivity and

- the buffering impact of positive emotions: replication and extension of a gene–environment interaction. *European Neuropsychopharmacology*, 24(6), 930-938.
- Verhagen, M., Van Der Meij, A., Van Deurzen, P. A. M., Janzing, J. G. E., Arias-Vasquez, A., Buitelaar, J. K., & Franke, B. (2010). Meta-analysis of the BDNF Val66Met polymorphism in major depressive disorder: effects of gender and ethnicity. *Molecular psychiatry*, 15(3), 260-271.
- Veiel, H. O. (1997). A preliminary profile of neuropsychological deficits associated with major depression. *Journal of clinical and experimental neuropsychology*, 19(4), 587-603.
- WHO (2017). Depression. <http://www.who.int/mediacentre/factsheets/fs369/en/>. (accessed February 28, 2017).
- Yang, B., Yang, C., Ren, Q., Zhang, J. C., Chen, Q. X., Shirayama, Y., & Hashimoto, K. (2016). Regional differences in the expression of brain-derived neurotrophic factor (BDNF) pro-peptide, proBDNF and preproBDNF in the brain confer stress resilience. *European archives of psychiatry and clinical neuroscience*, 266(8), 765-769.
- Yang, C., Shirayama, Y., Zhang, J. C., Ren, Q., & Hashimoto, K. (2015). Regional differences in brain-derived neurotrophic factor levels and dendritic spine density confer resilience to inescapable stress. *International Journal of Neuropsychopharmacology*, 18(7), pyu121.
- Young, J. E., Weinberger, A. D., & Beck, A. T. (2001). Cognitive therapy for depression. *Clinical handbook of psychological disorders: A step-by-step treatment manual*, 3, 264-308
- Ysseldyk, R., Haslam, S. A., & Haslam, C. (2013). Abide with me: religious group identification among older adults promotes health and well-being by maintaining multiple group memberships. *Aging & Mental Health*, 17(7), 869-879.

Appendix A

Recruitment Notice

Study Name: Biological underpinning to multiple social identities: The role of the oxytocin receptor gene

Description: The purpose of this study is to assess how genes influence one's ability to identify with social groups.

You will be asked to complete a series of questionnaires (some of which may be sensitive in nature as they pertain to depression, anxiety, unsupportive social interactions etc.) and provide one saliva sample for genetic analyses. This study will take approximately 1 hour.

This study has received clearance by the Carleton University Psychology Research Ethics Board (Reference #14-131).

Eligibility: Student at Carleton University.

Compensation: 1.25% Course Credit

Researchers: Robbie Woods (Principal Investigator); Samantha Santoni (Principle Investigator); Dr. Kim Matheson (Faculty Sponsor); Dr. Hymie Anisman (Other Personnel)

Principal Investigator: Robbie Woods Phone: 613 520-2600 ext. 2683 EMAIL: Robbie.Woods@carleton.ca

Principal Investigator: Samantha Santoni Phone: [REDACTED] EMAIL: samanthasantoni@carleton.ca

Faculty Sponsor: Dr. Kim Matheson Phone: 613 520-2000 ext. 2684 EMAIL: kim_matheson@carleton.ca

Other personnel: Dr. Hymie Anisman Phone: 613 520-2000 ext. 2699 EMAIL: hanisman@ccs.carleton.ca

Location: The sessions will occur in one of the following rooms: Social Science Research Building Rooms 303, 305, 307, 308, 314, Visual Simulation Building 6211 depending on availability.

Appendix B

Informed Consent

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent has to provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Study Title: Biological underpinning to multiple social identities: The role of the oxytocin receptor gene

Contacts

The following people are involved in this research project and may be contacted at any time if you have further questions about this project, what it means, or concerns about how it was conducted:

Robbie Woods, Graduate Researcher, Department of Neuroscience
Phone: 613 520-2600 ext. 2683, Robbie.Woods@carleton.ca

Samantha Santoni, Graduate Researcher, Department of Neuroscience
Phone: [REDACTED], Samantha.Santoni@carleton.ca

Dr. Kim Matheson, Faculty Member, Department of Psychology
Phone: 613 520-2000 ext. 2684, kim_matheson@carleton.ca

Dr. Hymie Anisman, Faculty Member, Department of Neuroscience
Phone: 613 520-2000 ext. 2699, hanisman@ccs.carleton.ca

Location: The sessions will occur in one of the following rooms: Social Science Research Building Rooms 303, 305, 307, 308, Visual Simulation Building 6211 depending on availability

Compensation: For your participation in the following study, you will be compensated 2.0% course credit.

Should you have any ethical concerns about this research, please contact Dr. Shelley Brown, at: Shelley_Brown@carleton.ca (613-520-2600 ext. 1505). For any other concerns, please contact Dr. Joanna Pozzulo (Chair, Department of Psychology, 613-520-2600, ext. 1412, psychchair@carleton.ca).

Purpose and Task Requirements:

The purpose of this study is to assess how group membership is related to your mental health and well-being. For this study, you will be asked to fill out a series of questionnaires, followed by a saliva sample for genetic analysis (all of which will be destroyed three years following completion of the study). In total, there are 15 questionnaires, some of which will be sensitive in nature, such as assessing depressive symptoms, unsupportive social interactions, and social anxiety, and childhood maltreatment. Moreover, questionnaires assessing demographic characteristics and a brief medical history, identification of social groups, attitudes to various

identities, and measures related to mood, coping, cognitive flexibility, and attachment to pets.

We are also interested in looking at how genes may influence our social behaviours, which may relate to psychological well-being. To assess this, we are asking participants to provide a DNA sample through the simple act of spitting into a tube (this should take approximately 3-5 minutes). We will use the saliva sample (that you will provide today) to determine the presence or absence of particular genes. By understanding the genetic factors that contribute to one's social identity, we will better understand group formation and interpersonal ties.

At the end of this session you will be fully debriefed about this study and what the researchers are expecting to find. The session will take 2 hour.

Potential risks or causes of discomfort for participants

Some individuals might feel discomfort when asked to provide personal, sensitive information. Furthermore, responding to some of the questions in this study or thinking about various stressors or difficulties in your life can be anxiety provoking or make you unhappy. If this is the case, the debriefing form at the end of the study contains contact information for people who are available to help. You can also choose to withdraw from the study at any point without penalty (i.e., you will still receive your research participation credit).

We are looking to collect about a teaspoon of saliva. Some participants may therefore feel discomfort or embarrassment about spitting into a tube in the presence of the researcher, in which case he/she will be happy to leave the room to give you privacy. You will be asked to not drink, eat, smoke or chew gum for 30 minutes before providing this sample.

Anonymity and confidentiality

All information and samples collected from you for this study will be identified with a code number, as opposed to any personal identifiers such as your name or address. We will maintain a separate record that matches your personal identification details with this code number. This is necessary in order for us to re-contact you in the future (with your express permission to do so), and so that we can respect your right to withdraw from the study at a later date (to be described below). However, this record that allows your identity to be linked to your information will kept in a separate and secured location, and will only be accessible to the primary researchers who have key access, in order to maintain anonymity and confidentiality of your information. All personal identifying information will be destroyed within 3 years of collection. Furthermore, the saliva samples will be stored in a secured storage area that is only accessible by the researcher and research assistants. Once these samples have been genotyped, any remaining saliva will be disposed of and will not be used for any other purposes.

Genetic testing: Common questions and concerns

What is DNA?

DNA is a large molecule that contains information necessary for our bodies to build all the components needed for our development, growth and survival. This information is commonly referred to as the genetic code or the DNA sequence. Some rare diseases can be attributed entirely to simple errors in our DNA sequence. However, the majority of common diseases (including depression) are caused by a combination of many different genetic factors, together

with environmental factors (how we grew up, life events, etc.).

What will my DNA be used for?

If you compare any two people, their DNA will be about 99% identical. We are interested in the 1% of DNA that is different between people. Our current plan is to investigate these differences, focusing on just a small proportion of your genes (we are targeting less than 100 of the ~30,000 genes that humans have), which we anticipate may be involved in risk of either anxiety or depression. We are also planning future follow-up studies on your DNA, which will extend the analysis to substantially more genes – potentially all genes. These future studies will be limited to analyses of the DNA molecule and the genetic code, and will not involve any other use or manipulation of your DNA sample. However, in no case will your samples be kept for more than 3 years, at which time the samples and the sample container will be incinerated. Thus, the data derived from the sample will be kept (stripped of any information that could identify you), but the sample or remnants of the sample, including DNA, will be destroyed (incinerated). At the end of this form, you have the option to opt-out of any such future uses of your DNA sample.

How long will my DNA be stored, and potentially used in research?

By providing a DNA sample and signing this form, you are indicating that you are willing for us to preserve and analyze your DNA sample for an extended period of time (3 years or less). During this period, use of the sample is guaranteed to be limited to studies that read the DNA molecule. As indicated above, your saliva and any DNA samples will be destroyed, by incineration, no later than 3 years after saliva was collected.

Will I be told the results of my own genetic analysis?

No. Your DNA sample and genetic information will be identified by a code number, and not your name. This preserves confidentiality of this information. Returning your personal genetic information to you would require that confidentiality to be compromised, so will be avoided. Furthermore, as described above, genetic data collected in this study will not allow accurate prediction of whether or not you will develop any disease. Although in the future we hope to be able to use genetic materials to examine disease susceptibility, our research is in a preliminary phase and we are not currently able to provide this kind of information on an individual basis.

What if something unexpected and potentially dangerous is discovered in my DNA

None of the DNA sites that we plan to analyze are currently known to be predictive of disease with any real accuracy. However, future advances in genetic research could allow disease predictions to be possible based on information from these, or other genetic sites. In exceptional circumstances, if genetic research reveals information about a serious or life-threatening condition that can be prevented or treated through intervention, then we have an obligation to inform you of this information, and potentially also inform your biological relatives who may share similar risk of disease. This would therefore represent a potential breach of confidentiality. In this instance, only information directly relating to disease diagnosis, and participant identity, would be shared. We wish to make clear, however, that it is not our intent to systematically go through the samples after our initial analyses, and so even if it was subsequently discovered that certain genes might carry relevant information for you, it is unlikely that we would actually spot any vulnerabilities you might have.

Can my DNA ever be used to identify me?

This is a complicated question to answer. Unless you have an identical twin (whose DNA will be

identical to yours), your DNA is absolutely unique to you. It is this unique nature of genetic material that allows individuals to be identified based entirely on their DNA, through techniques such as DNA fingerprinting. It is therefore theoretically possible that in the future, your identity could be determined from simply analyzing your DNA sample. It is extremely unlikely, however, that you could be identified based on your DNA sample. In order to identify you based purely on your DNA sample, it would be necessary to compare your DNA sample that you provide today, with another DNA sample from you in a DNA database, which is linked to your identity. DNA databases do exist in countries including Canada, Australia, USA and UK, but are limited to samples from criminal offenders. Access to these databases is strictly limited to law enforcement agencies thus cannot be accessed by researchers. Access to DNA samples taken for this study will similarly be limited to the researchers, and will not be provided to any law enforcement agency unless we become legally obliged to do so (to our knowledge, this has never happened to any research group). Furthermore, these government DNA databases typically contain information about only 13 regions of human DNA, none of which are to be analyzed in the present study.

If you have any additional questions or concerns, please ask the researcher today, or contact any of the principal investigators at a later date.

Right to withdraw from this study

Participation in this study is entirely voluntary. At any point during the study you have the right to not complete certain questions or to withdraw with no penalty whatsoever. Furthermore, if at a later date you wish to withdraw from the study, you can contact the principal investigators and we will destroy all of your records (questionnaire answers, responses from the interview, plus DNA sample) from this study. The only exception is where data has already been published. In this instance, unpublished data plus your DNA sample will be destroyed.

Declaration of consent

Please indicate below how you would like your sample to be treated in the future. There are no obligations or penalties for you associated with your selection.

- Option 1: I grant use of my DNA/saliva sample, but this use will be strictly limited to the analysis of <100 genes, as described in the current research plan. This can only be done within a 3 year period of my saliva being collected.
- Option 2: I grant the researcher permission to re-contact me to seek consent to use my DNA/saliva samples in future research studies.
Email: _____ Phone Number: _____
- Option 3: I grant the researcher permission to use my DNA/saliva samples for any future research studies (for up to three years, after which all identifying personal information and genetic material will be destroyed) that involve analyses of the DNA molecule.

This study has been approved by the Carleton University Psychology Research Ethics Board (Ethics #14-131)

Signatures

I have read the above form and understand the conditions of my participation. My participation in this study is voluntary, and I understand that if at any time I wish to leave the experiment, I may do so without having to give an

explanation and with no penalty whatsoever. Furthermore, I am also aware that the data gathered in this study are confidential and anonymous with respect to my personal identity. My signature indicates that I agree to participate in this study.

Participant's Full Name: _____ Participant's Signature: _____

Researcher's Name: _____ Researcher's Signature: _____

Date _____

Appendix C

Written Debriefing for participants

What are we trying to learn in this research?

The goal of the present study is to examine the relationship between psychological well-being and the identification with social groups (social identity). In addition, whether having multiple social identities relates to one's mental health (decreased depression, anxiety, etc.). Recently, research has shown that having a strong identification to a social group is associated with greater psychological well-being. Sharing a social identity may lead to greater resources available, such as receiving social support (emotional comfort, financial assistance), therefore have positive psychological outcomes, whereas the lack of support or unsupportive interactions (anticipating support when sought out, but not receiving it) has been shown to be detrimental to one's mental health. Further, certain coping strategies may be more effective at dealing with the stressors at hand. A tendency towards certain coping profiles has been associated with the emergence of mental health issues (e.g. rumination). Certain factors may contribute to hinder one's ability to receive support from others, or identify with social groups. Feelings of loneliness, a disposition to be sensitive to rejection, or of being socially isolated, or an inherent distrust in others may prevent individuals from seeking social support. Moreover, these factors may prevent an individual from being able to identify with others, which may have negative psychological repercussions. Conversely, individuals who feel competent in their own abilities may demonstrate an ability to seek out support, or reorient their thoughts to other social groups that they share a sense of belonging to.

In addition, there are certain genes that may underlie our social behaviour. Recent research has focused on important genetic factors that may underlie increased susceptibility to develop depression and anxiety related illnesses. These genetic factors comprise small deviations of certain genes that control the functioning of neurochemical systems in the brain. The slight deviations are called polymorphisms, and are fairly common. You were asked to provide a saliva sample, so that genetic material (e.g., DNA) can be extracted. We are assessing whether people who have different polymorphisms relates to the development of social identities, which have been implicated in mental and physical well-being. The genes that regulate the hormone oxytocin and the growth factor brain-derived neurotrophic factor have been important in understanding behaviours associated with interpersonal trust. As populations vary with regards to the expression of particular genes, the goal of this study is to examine how such variations may influence both social interactions (social support and unsupport) and identifying with social groups (social identity). Furthermore, it has been seen repeatedly that early life traumatic events can lead to poor mental-health later in life. Where research is still unsure, however, is whether polymorphisms in the brain-derived neurotrophic factor (BDNF) gene will increase or decrease the likelihood that those traumatic experiences will lead to mental illness. Even less research has seen whether the same would be true for current life stress and experiences. In this study we hope to better understand how BDNF mutations can influence the relationship between stress and depression through. Moreover, we wish to understand how one's psychological well-being may be implicated in both genes and social interactions.

Lastly, companion animals play an important role in both the physical and psychological well-

being of individuals. They provide the emotional comfort, physical exercise, and help facilitate social interactions. Therefore they have the potential to provide a shared sense of belonging, decreased loneliness, and social support (particularly those who experience anxiety around other people). By examining the genes associated with social behaviours, this may explain the relationship between humans and companion animals, and how this may influence psychological well-being.

Why is this important to scientists or the general public?

Social relationships, whether they are human or non-human, are important contributors to our health and well-being, and recent research has shown that genes play an important role in our behaviours. Thus, some individuals may have a predisposition to developing social ties and identifying with others, whereas it may be more of an effort for others to seek support, and thus may compromise their mental health. Identifying a genetic contribution to social identity may benefit those who lack the predisposition in order to better target treatments that help improve the benefits of social identity.

What are our hypotheses and predictions?

Individuals possessing the more common OXTR (rs53576; which has been associated with greater trusting and affiliative behaviours) will possess a greater frequency of social identities and group belonging. As a consequence, this may serve as a protective factor against developing psychopathologies, such as depressive symptoms. Conversely, showing greater distrust or social anxiety may contribute to having fewer group memberships.

Where can I learn more?

If you would like to learn more about social identity and how genes play an important role in our mental health, please see these two papers as they provide a rationale for the basis of this study.

Krueger, F., Parasuraman, R., Iyengar, V., Thornburg, M., Weel, J., Lin, M., ... & Lipsky, R. H. (2012). Oxytocin receptor genetic variation promotes human trust behavior. *Frontiers in human neuroscience*, 6.

Cruwys, T., Haslam, S. A., Dingle, G. A., Haslam, C., & Jetten, J. (2014). Depression and Social Identity An Integrative Review. *Personality and Social Psychology Review*, 1088868314523839.

Is there anything that I can do if I found this experiment to be emotionally draining?

Thank you very much for your participation in this study. If you have experienced any distress while completing these measures, please consult the resources below:

Carleton University Health and Counseling Services at: 613-520-6674,

Ottawa Distress Centre: (613) 238 1089, Web Site: www.dcottawa.on.ca.

Mental Health Crisis Line: within Ottawa (613) 722-6914, outside Ottawa 1-866-996-0991, Web Site: <http://www.crisisline.ca/>

What if I have questions later?

Please contact:

Robbie Woods, Graduate Researcher
Phone: 613 520 2600 ext. 2683
Email: Robbie.Woods@carleton.ca

Samantha Santoni
Phone: [REDACTED]
Email: samantha.santoni@carleton.ca

Dr. Kim Matheson, Faculty Sponsor
Phone: 613 520-2000 ext. 2684
Email: kim_matheson@carleton.ca

Dr. Hymie Anisman, Other Personnel
Phone: 613 520-2000 ext. 2699,
Email: hanisman@ccs.carleton.ca

Ethical concerns: Dr. Shelley Brown, Chair of Carleton University Ethics Committee for Psychological Research, Shelley_Brown@carleton.ca (613-520-2600 ext. 1505).

Any other concerns: For any other concerns, please contact Dr. Joanna Pozzulo (Chair, Department of Psychology, 613-520-2600, ext. 8218, psychchair@carleton.ca); Dr. John Stead, Chair, Department of Neuroscience, 613 520-2600 ext. 8774, john_stead@carleton.ca.

Appendix D

General Information

The purpose of the following set of questions is to collect demographic information about various aspects of your life. Although some of the questions may seem unrelated to the present study (e.g. weight, height, religion, etc....) these factors may be important determinants of your health and well-being.

1. Sex: Female / Male (please select one)

2. Age: _____

3. What is your citizenship status?

_____	Canadian citizen	Since what year? _____	Country of origin _____
_____	Landed immigrant	Since what year? _____	Country of origin _____
_____	Student visa	Since what year? _____	Country of origin _____
_____	Temporary visa	Since what year? _____	Country of origin _____
_____	Refugee	Since what year? _____	Country of origin _____

4. What is your first language?

If your first language is not English, how long have you been **fluent** in reading, writing and comprehension of the English language?

5. What is your ethnic/racial background? *Please select the one that best applies to you.*
 - _____ Asian (e.g., Chinese, Japanese, Korean)
 - _____ South Asian (e.g., East Indian, Pakistani, Punjabi, Tamil, Sri Lankan)
 - _____ South East Asian (e.g., Cambodian, Indonesian, Laotian)
 - _____ Arab/West Asian (e.g., Armenian, Egyptian, Iranian, Lebanese, Moroccan)
 - _____ Black (e.g., African, Haitian, Jamaican, Somali)
 - _____ Latin American/Hispanic
 - _____ Aboriginal
 - _____ White/Euro-Caucasian
 - _____ Other (please specify): _____

6. What is your religious affiliation? *Please select the one that best applies to you.*
 - _____ None—Atheist (e.g., belief that there is NO God)
 - _____ None—Agnostic (e.g., belief that the existence of God cannot be known)
 - _____ Protestant (e.g., United, Anglican, Baptist, Presbyterian, Lutheran, Pentecostal, Mennonite, “Christian”)
 - _____ Catholic (e.g., Roman Catholic, Ukrainian Catholic)
 - _____ Jewish
 - _____ Muslim
 - _____ Buddhist
 - _____ Hindu
 - _____ Sikh
 - _____ Bahá’í
 - _____ Other (please specify): _____

7. How long have you been a resident of Ottawa, Ontario?

8. If you just moved to Ottawa to attend University, how far is your former place of residence?
- I was already living in Ottawa before attending Carleton University
 - Less than an hour from Ottawa
 - 1+ hour from Ottawa
 - 3+ hours from Ottawa
 - 5+ hours from Ottawa..... Please specify the city _____
 - Outside of Ontario/Quebec..... Please specify the province _____
 - Outside of Canada..... Please specify the country _____

9. If you just moved to Ottawa to attend University, how long did you live in your former place of residence?

10. If you just moved to Ottawa, how often do you do you go back to your former place of residence?

11. If you just moved to Ottawa, have you been away from home before?

YES NO

If YES, please indicate how often: _____ and for how long: _____

12. What is your current living arrangement? *Please select the one that best applies to you.*

- Living alone in residence (at Carleton University)
 - Living alone off-campus
 - Living with friends in residence (at Carleton University)
 - Living with friends off-campus
 - Living with roommates in residence (at Carleton University)
 - Living with roommates off-campus
 - Living with parents
 - Living with spouse/significant other
 - Living with spouse/significant other and young children (13 years and younger)
 - Living with spouse/significant other and older children (13 years and older)
 - Living alone with young children (13 years and younger)
 - Living alone with older children (13 years and older)
 - Other (please specify)
- _____

13. Do you have any relatives or close friends in Ottawa? YES NO

If YES, how many?

14. What is your current relationship status? *Please select the one that best applies to you.*

- Single, and not seeing anyone
- Going out with someone
- In a serious dating relationship
- Have recently broken up ... Please specify how many weeks ago you broke up _____
- Living with an intimate other
- Engaged
- Married
- Separated/Divorced Please specify how many months ago you separated _____
- Widowed

15. Is your current (or most recent) partner: Male _____ OR Female _____?

16. What level of education have you completed?

- _____ 8 years or less of elementary school
- _____ some high school but no diploma
- _____ a high school diploma or equivalent
- _____ 1 to 3 years of college/university (including study at a technical college or CEGEP)
- _____ an undergraduate university degree
- _____ a master's degree
- _____ a doctoral degree
- _____ a professional degree [medicine (M.D.), dentistry (D.D.S.), law, etc.]

17. Have you had or do you currently have any health related (i.e., medical) illnesses or physical conditions? *Please select the one that best applies to you.*

- _____ NO, I don't
- _____ YES, I did but I no longer do
- _____ YES, I do

If YES, please specify illness/condition you had/have

If YES, please specify any current treatment you are receiving

18. Do you currently have a psychological disorder/condition (e.g. depression, anxiety, etc.)?

- _____ NO, I don't
- _____ YES, I do

If YES, please specify disorder/condition

If YES, are you currently being treated for this disorder/condition?

- _____ NO, I'm not
- _____ YES I am

If YES, please specify treatment type (e.g. medications, therapy).

19. Have you ever in the past had a psychological disorder/condition (e.g. depression, anxiety, etc.) but no longer do?

- _____ NO, I haven't
- _____ YES, I have

If YES, please specify the disorder/condition you had _____

20. In your opinion, how would you describe your health?

- _____ Poor
- _____ Fair
- _____ Good
- _____ Very good
- _____ Excellent

21. Are you on any of the following medications (please check all that apply)?

Anti-inflammatories (please specify) _____
Anti-depressants (please specify) _____
Anti-anxieties (please specify) _____
Allergy medication (please specify) _____
Other prescription drugs (please specify) _____

22. What is your estimate of your family's gross income per year? *Please select the one that best applies to you.*

- | | |
|--|---|
| <input type="checkbox"/> under \$15,000 | <input type="checkbox"/> \$60,000 - \$74,999 |
| <input type="checkbox"/> \$15,000 - \$29,999 | <input type="checkbox"/> \$75,000 - \$89,999 |
| <input type="checkbox"/> \$30,000 - \$44,999 | <input type="checkbox"/> \$90,000 - \$104,999 |
| <input type="checkbox"/> \$45,000 - \$59,999 | <input type="checkbox"/> \$105,000 or more |

23. What is your employment status?

- Employed Part-time
 Employed Full-time
 Unemployed
 Retired
 Other: _____

Appendix E

Beck Depression Inventory

On this questionnaire are groups of statements. Please read the entire group of statements in each category. Then pick out ONE statement in that group which best describes the way you feel. Check off the number beside the statement you have chosen.

1. 0 = I do not feel sad
 1 = I feel sad or blue
 2a = I am blue or sad all of the time and I can't snap out of it
 2b = I am so sad or unhappy that it is very painful
 3 = I am so sad or unhappy that I can't stand it

2. 0 = I am not particularly pessimistic or discouraged about the future
 1 = I feel discouraged about the future
 2a = I feel I have nothing to look forward to
 2b = I feel I won't ever get over my troubles
 3 = I feel that the future is hopeless and things cannot improve

3. 0 = I do not feel like a failure
 1 = I feel I have failed more than the average person
 2a = I feel I have accomplished very little that is worthwhile or that means anything
 2b = As I look back on my life, all I can see is a lot of failures
 3 = I feel I am a complete failure as a person

4. 0 = I am not particularly dissatisfied
 1a = I feel bored most of the time
 1b = I don't enjoy things the way I used to
 2 = I don't get satisfaction out of anything anymore
 3 = I am dissatisfied with everything

5. 0 = I don't feel particularly guilty
 1 = I feel bad or unworthy a good part of the time
 2a = I feel quite guilty
 2b = I feel bad or unworthy practically all of the time now
 3 = I feel as though I am very bad or worthless

6. 0 = I don't feel I am being punished
 1 = I have a feeling that something bad may happen to me
 2 = I feel I am being punished or will be punished
 3a = I feel I deserve to be punished
 3b = I want to be punished

7. 0 = I don't feel disappointed in myself
 1a = I am disappointed in myself
 1b = I don't like myself
 2 = I am disgusted with myself
 3 = I hate myself

8. 0 = I do not feel I am any worse than anybody else
 1 = I am very critical of myself for my weaknesses or mistakes
 2a = I blame myself for everything that goes wrong
 2b = I feel I have many bad faults

9. ___ 0 = I don't have thoughts of harming myself
 ___ 1 = I have thoughts of harming myself but I would not carry them out
 ___ 2a = I feel I would be better off dead
 ___ 2b = I have definite plans about committing suicide
 ___ 2c = I feel my family would be better off if I were dead
 ___ 3 = I would kill myself if I could
10. ___ 0 = I don't cry any more than usual
 ___ 1 = I cry more now than I used to
 ___ 2 = I cry all the time now. I can't stop it
 ___ 3 = I used to be able to cry but now I can't cry at all even though I want to
11. ___ 0 = I am no more irritable than usual
 ___ 1 = I am more irritable than usual
 ___ 2 = I am much more irritable than usual
 ___ 3 = I am irritable all the time
12. ___ 0 = I have not lost interest in other people
 ___ 1 = I am less interested in other people than I used to be
 ___ 2 = I have lost most of my interest in other people and I have little feeling for them
 ___ 3 = I have lost all my interest in other people and don't care about them at all
13. ___ 0 = I make decisions about as well as ever
 ___ 1 = I am less sure of myself now and try to put off making decisions
 ___ 2 = I can't make decisions anymore without help
 ___ 3 = I can't make decisions at all anymore
14. ___ 0 = I don't feel I look any worse than I used to
 ___ 1 = I am worried that I am looking old or unattractive
 ___ 2 = I feel that there are permanent changes in my appearance and they make me look unattractive
 ___ 3 = I feel that I am ugly or repulsive looking
15. ___ 0 = I can work about as well as before
 ___ 1a = It takes extra effort to get started at doing something
 ___ 1b = I don't work as well as I used to
 ___ 2 = I have to push myself very hard to do anything
 ___ 3 = I can't do any work at all
16. ___ 0 = I can sleep as well as usual
 ___ 1 = I wake up more tired in the morning than I used to
 ___ 2 = I wake up 1-2 hours earlier than usual and find it hard to get back to sleep
 ___ 3 = I wake up early every day and can't get more than 5 hours of sleep
17. ___ 0 = I don't get any more tired than usual
 ___ 1 = I get tired more easily than I used to
 ___ 2 = I get tired from doing anything
 ___ 3 = I get too tired to do anything
- 18a. ___ 0 = My appetite is no worse than usual
 ___ 1 = My appetite is not as good as it used to be
 ___ 2 = My appetite is much worse now
 ___ 3 = I have no appetite at all any more
- 18b. ___ 0 = I am not eating more than usual
 ___ 1 = I am eating a little more than usual
 ___ 2 = I am eating somewhat more than usual

___ 3 = I am eating a lot more than usual

- 18c. ___ 0 = I have had no change in food preferences lately
___ 1 = I have been craving more carbohydrates (starches or sweets lately)
___ 2 = I have had irresistible craving for sweets and starches lately

- 19a. ___ 0 = I haven't lost much weight, if any, lately
___ 1 = I have lost more than 5 pounds
___ 2 = I have lost more than 10 pounds
___ 3 = I have lost more than 15 pounds

- 19b. ___ 0 = I have not gained any weight lately
___ 1 = I have gained more than 5 pounds
___ 2 = I have gained more than 10 pounds
___ 3 = I have gained more than 15 pounds

20. ___ 0 = I am no more concerned about my health than usual
___ 1 = I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body
___ 2 = I am so concerned with how I feel or what I feel that it's hard to think of much else
___ 3 = I am completely absorbed in what I feel

21. ___ 0 = I have not noticed any recent change in my interest in sex
___ 1 = I am less interested in sex than I used to be
___ 2 = I am much less interested in sex now
___ 3 = I have lost interest in sex completely

22. ___ 0 = I have not had any increase in nightly sleep length lately
___ 1 = I have had at least 1-hour increase in sleep length
___ 2 = I have had at least 2-hour increase in sleep length
___ 3 = I have had at least 3-hour increase in sleep length

My average nightly sleep length in the past week is: _____ hours (per night)

23. ___ 0 = I am not feeling more fatigued than usual
___ 1 = I feel more fatigued than usual lately, but it does not interfere with my daily functioning
___ 2 = I feel more fatigued than usual lately, and it interferes somewhat with my daily functioning
___ 3 = I feel more fatigued than usual lately, and it significantly interferes with my daily functioning

24. ___ 0 = I have not had any mood swings or slumps lately
___ 1 = I have had some mood swings or slumps lately but very minor
___ 2 = I have had more mood swings or slumps than usual
___ 3 = I have had severe mood swings or slumps lately

Appendix F

Additional debriefing form

(This debriefing will be provided to those who select 2a or higher on question 9 of the BDI)

Depression is a condition that can occur for many reasons, including workplace, school, or relationship stressors, traumatic life events, discrimination, as well as physical/biological imbalances. Approximately 10-15% of people will suffer some degree of depression during their lifetime. With advances in modern medicine, most people can readily be treated for this illness, which if unattended can be long lasting and affect many aspects of one's life. The symptoms of depression comprise:

- * Poor or depressed mood, or a reduction in the pleasure gained from otherwise positive experiences
- * Sleep disturbances
- * Eating disturbances (loss of appetite, or overeating despite not being hungry), which may be linked to weight changes
- * Lack of sexual interest
- * Fatigue and lethargy (you don't feel like doing anything)
- * An inability to focus (e.g., you have a hard time reading)
- * Reduced interactions with family and friends
- * Thoughts of suicide

Someone who is depressed may experience several (3-4), but not necessarily all of the above symptoms. It is likewise the case that 60% of individuals will encounter a severe traumatic event in their lives and of these people, a fair number will develop symptoms that cause severe anxiety. Illnesses of this nature, including posttraumatic stress disorder (PTSD) can be treated. Once again, if unattended, the repercussions can be severe.

Symptoms include:

- * Hyperarousal (e.g., feelings of anxiety and reactivity even to minor situations)
- * Intrusive thoughts (memories of the event come into your head frequently)
- * Avoiding thoughts or stimuli related to the event

These symptoms can persist for more than a month following the event, and influence your day-to-day functioning.

Your responses to this survey suggest that you may be experiencing one of the above disorders. If you are not already receiving attention for this problem, it is suggested that you contact your family physician. It is not a good idea to allow problems to fester, as ruminating over these problems will typically not make them go away. Your family physician or counselor will usually be able to help you or to refer you to someone who can. If you do not have a family physician, then you can contact either of the following:

Mental Health Crisis Line: within Ottawa (613) 722-6914, outside Ottawa 1-866-996-0991,
Web Site: <http://www.crisisline.ca/>

Ottawa Distress Centre: (613) 238 1089, Web Site: www.dcottawa.on.ca

Appendix G

Suicidal Ideation Protocol

IN-PERSON/TELEPHONE SITUATIONS

Item 9 (from 21-item version; Item 7 on 13-item version) on the Beck is checked immediately (e.g., while getting credit information and debriefing ready).

If the Beck 9 is 0 or 1, nothing is done except give credit and debriefing. The debriefing includes a summary of the goals of the study as well as a list of contact numbers (e.g., Health and Counseling Services).

If the Beck 9 is a 2a, the participant is reminded of counseling services available at Carleton, and in the community. Credit and debriefing are subsequently given. If there are many participants (group questionnaire setting) and it is not feasible to remind the participant privately in the study room of services, then the participant will be taken to a private room, with the researcher saying that they are being taken to be debriefed, and they will be reminded of services available there. Credit and debriefing sheet are provided.

If the Beck 9 is 2b, c or 3:

If possible, the participant is spoken to privately. If speaking with the participant privately in the study room is not feasible (group setting), then the participant will be taken to a private room, with the researcher saying they are being taken to be debriefed. The researcher will state that they have noticed the Beck item, and that they are concerned about their welfare. The summarized seven-step protocol (below) is then implemented. The summarized seven-step protocol (below) is then implemented.

The following will be assessed:

1. The length of time that participant has had suicidal thoughts.
2. Whether the participant has talked to anyone regarding these thoughts.
3. Whether the participant is currently seeing a therapist.
4. Whether the participant has a plan and the means to carry out their plan
5. Whether the thought to carry out their plan is imminent
6. If plan is imminent then the protocol outlined below will be followed.

NOTE: Keep a written record documenting the assessment.

ADDITIONAL DETAILS:

The plan and means. The participant is questioned about the plan and the means to carry out this plan. Examples of plans are such things as taking large amounts of painkillers, and means are having lots of painkillers available. You don't have to give examples of plans, just ask whether they have thought about how they would do it.

If there are no plans, or there are plans but no means (e.g., take painkillers but none around), remind the participant of counseling services available in the community and also the ER at the hospital. If the participant is also seeing a therapist, it is suggested that the participant speak with the therapist about this. Then the credit and debriefing are given.

If there are both plans and means, the participant is asked whether thoughts to carry out this plan are imminent (that is, are they thinking of doing this very soon? For example, within the next day).

If not imminent, OR have plans and means but don't think they would carry them out (e.g., yes,

I've thought about doing it occasionally and have the meds but realize I could not go through with it), the participant is reminded of counseling services available in the community, and also the ER at the hospital. If also seeing a therapist, it is suggested that the participant speak with their therapist about this. Then the credit and debriefing are given.

If means are available and plan is imminent, and there is good reason to believe that then individual may in fact carry out the suicidal thoughts soon, then the participant is informed that you will be calling 911 because you are very concerned that they will harm themselves. During the 911 call, the police are informed of the individual's imminent intent to commit suicide. The person's name and phone are given to the police. This step involves breaking confidentiality, but the welfare of the participant takes priority (APA and CPA and Tri-Council guideline 3.1) 911 will take it from there. The situation is documented, and your supervisor and ethics chair are contacted.

Things NOT TO DO in both in-person and telephone situations

Do not give out your lab number as a resource for somewhere to call for help.

Do not give out home phone numbers of research personnel.

Do not intervene directly with the participant. That is, do not escort the person to the hospital or health services. If a participant does call the lab for help, refer them again to the resources, such as Health Services or the Distress Centre or hospital. Assess for immediacy of suicidal intention, and follow the steps outlined above, such as finding out if there is someone else there, calling 911 directly if there is imminent suicidal intent, etc.

Do not engage in a helping relationship with the person. Provide the information about resources, but, for example, do not make follow-up calls to check up on the person and see how they are doing.

Do not do any of this assessment and suicidal screening if you do not feel confident about it. Refer it to your supervisor.

BDI item 9

9. ___ 0 = I don't have thoughts of harming myself

___ 1 = I have thoughts of harming myself but I would not carry them out

___ 2a = I feel I would be better off dead

___ 2b = I have definite plans about committing suicide

___ 2c = I feel my family would be better off if I were dead

___ 3 = I would kill myself if I could

Appendix H

Early Life Trauma Inventory (ELTI)

Most people have experienced a traumatic event at some point in their life. For this survey, we are interested in the types of traumatic events that you may have experienced, and how old you were when you experienced it. For each question, please indicate how many times you have experienced the event (if at all), and at what age range, by circling the appropriate number.

Part 1. General Traumas

1. Were you ever exposed to a life-threatening natural disaster?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
2. Were you involved in a serious accident?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
3. Did you ever suffer a serious personal injury or illness?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
4. Did you ever experience the death or serious illness of a parent or a primary caretaker?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
5. Did you experience the divorce or separation of your parents?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
6. Did you experience the death or serious injury of a sibling?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

7. Did you ever experience the death or serious injury of a friend?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

8. Did you ever witness violence towards others, including family members?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

9. Did anyone in your family ever suffer from mental or psychiatric illness or have a “breakdown”?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

10. Did your parents or primary caretaker have a problem with alcoholism or drug abuse?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

11. Did you ever see someone murdered?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

Part 2: Physical Punishment

1. Were you ever slapped in the face with an open hand?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

2. Were you ever burned with hot water, a cigarette or something else?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

3. Were you ever punched or kicked?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
4. Were you ever hit with an object that was thrown at you?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
5. Were you ever pushed or shoved?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

Part 3: Emotional Abuse

1. Were you often put down or ridiculed?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
2. Were you often ignored or made to feel that you didn't count?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
3. Were you often told you were no good?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
4. Most of the time were you treated in a cold, uncaring way or made to feel like you were not loved?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
5. Did your parents or caretakers often fail to understand you or your needs?						
Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

Part 4: Sexual Events

1. Were you ever touched in an intimate or private part of your body (e.g. breast, thighs, genitals) in a way that surprised you or made you feel uncomfortable?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

2. Did you ever experience someone rubbing their genitals against you?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

3. Were you ever forced or coerced to touch another person in an intimate or private part of their body?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

4. Did anyone ever have genital sex with you against your will?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

5. Were you ever forced or coerced to perform oral sex on someone against your will?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

6. Were you ever forced or coerced to kiss someone in a sexual rather than an affectionate way?

Age 0 to 5	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 6 to 12	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times
Age 13 to 18	0 Never	1 Once	2 2-3 times	3 4-5 times	4 6-10 times	5 More than 10 times

In the past year did the following occur? (Leave blank if the event did not occur in the past year.)	YES/NO	Positive Impact	Negative Impact	Extent of Impact
Change work situation (different work responsibility, major change in working conditions, hours, etc.).				
New job.				
Serious illness or injury of close family member.				
Marriage				
Sexual difficulties.				
Trouble with in-laws.				
Trouble with employer (in danger of losing job, being suspended, demoted, etc.).				
Major change in financial status (much better/worse off).				
Major change in closeness of family members (increased or decreased closeness).				
Gaining a new family member (through birth, adoption, family member moving in, etc.).				
Change of residence.				
Separation from partner (due to conflict).				
Major change in church activities (increased or decreased attendance).				
Reconciliation (making up) with partner.				

In the past year did the following occur? (Leave blank if the event did not occur in the past year.)	YES/NO	Positive Impact	Negative Impact	<i>Extent of Impact</i>
Major change in number of arguments with partner (a lot more or a lot fewer arguments)				
Change in spouse/partner's work (loss of job, beginning new job, retirement, etc.).				
Major change in usual type and/or amount of recreation.				
Borrowing more than \$10,000 (buying home, business, etc.)				
Borrowing less than \$10,000 (buying car, RV, getting school loan, etc.).				
Being fired from job.				
You or your spouse/partner had an abortion.				
Major personal illness or injury.				
Major change in social activities, such as parties, movies, visiting (increased or decreased participation).				
Major change in family living conditions (building new home, remodelling, deterioration of home, neighbourhood).				
Divorce.				
Serious injury or illness of a close friend.				
Separation from spouse (due to work, travel, etc.)				
Engagement.				

In the past year did the following occur? (Leave blank if the event did not occur in the past year.)	YES/NO	Positive Impact	Negative Impact	<i>Extent of Impact</i>
Breaking up with boyfriend/girlfriend.				
Leaving home for the first time.				
Reconciliation (making up) with boyfriend/girlfriend.				
Beginning a new school experience at a higher academic level (college, graduate school, professional school, etc...)				
Changing to a new school at same academic level (undergraduate, graduate, etc...)				
Academic probation				
Being dismissed from dormitory or other residence				
Failing an important exam				
Changing a major				
Failing a course				
Dropping a course				
Joining a fraternity/sorority				
Financial problems concerning school (in danger of not having sufficient money to continue)				

Appendix J

SCOPE

The purpose of this questionnaire is to find out how people deal with their problems or the stresses in their lives. The following are activities that you may have done. After each activity, please indicate the extent to which you would use this as a way of dealing with problems or stresses in recent weeks.

<i>Ordinarily, in recent weeks have you</i>	Never	Seldom	Sometimes	Often	Almost Always
1. accepted that there was nothing you could do to change your situation?	0	1	2	3	4
2. tried to just take whatever came your way?	0	1	2	3	4
3. talked with friends or relatives about your problems?	0	1	2	3	4
4. tried to do things which you typically enjoy?	0	1	2	3	4
5. sought out information that would help you resolve your problems?	0	1	2	3	4
6. blamed others for creating your problems or making them worse?	0	1	2	3	4
7. sought the advice of others to resolve your problems?	0	1	2	3	4
8. blamed yourself for your problems?	0	1	2	3	4
9. exercised?	0	1	2	3	4
10. fantasized or thought about unreal things (e.g., the perfect revenge, or winning a million dollars) to feel better?	0	1	2	3	4
11. been very emotional compared to your usual self?	0	1	2	3	4
12. gone over your problems in your mind over and over again?	0	1	2	3	4
13. asked others for help?	0	1	2	3	4

14. thought about your problems a lot?	0	1	2	3	4
15. became involved in recreation or pleasure activities?	0	1	2	3	4
16. worried about your problems a lot?	0	1	2	3	4
17. tried to keep your mind off things that are upsetting you?	0	1	2	3	4
18. tried to distract yourself from your troubles?	0	1	2	3	4
19. avoided thinking about your problems?	0	1	2	3	4
20. made plans to overcome your problems?	0	1	2	3	4
21. told jokes about your situation?	0	1	2	3	4
22. thought a lot about who is responsible for your problems (besides yourself)?	0	1	2	3	4
23. shared humorous stories etc. to cheer yourself and others up?	0	1	2	3	4
24. told yourself that other people have dealt with problems such as yours?	0	1	2	3	4
25. thought a lot about how you have brought your problems on yourself?	0	1	2	3	4
26. decided to wait and see how things turn out?	0	1	2	3	4
27. wished the situation would go away or be over with?	0	1	2	3	4
28. decided that your current problems are a result of your own past actions?	0	1	2	3	4
29. gone shopping?	0	1	2	3	4
30. asserted yourself and taken positive action on problems that are getting you down?	0	1	2	3	4

31. sought reassurance and moral support from others?	0	1	2	3	4
32. resigned yourself to your problems?	0	1	2	3	4
33. thought about how your problems have been caused by other people?	0	1	2	3	4
34. daydreamed about how things may turn out?	0	1	2	3	4
35. been very emotional in how you react, even to little things?	0	1	2	3	4
36. decided that you can grow and learn through your problems?	0	1	2	3	4
37. told yourself that other people have problems like your own?	0	1	2	3	4
38. wished I was a stronger person or better at dealing with problems?	0	1	2	3	4
39. looked for how you can learn something out of your bad situation?	0	1	2	3	4
40. asked for God's guidance?	0	1	2	3	4
41. kept your feelings bottled up inside?	0	1	2	3	4
42. found yourself crying more than usual?	0	1	2	3	4
43. tried to act as if you were not upset?	0	1	2	3	4
44. prayed for help?	0	1	2	3	4
45. gone out?	0	1	2	3	4
46. held in your feelings?	0	1	2	3	4
47. tried to act as if you weren't feeling bad?	0	1	2	3	4

48. taken steps to overcome your problems?	0	1	2	3	4
49. made humorous comments or wise cracks?	0	1	2	3	4
50. told others that you were depressed or emotionally upset?	0	1	2	3	4

Appendix K

Cognitive Flexibility Questionnaire

The purpose of this questionnaire is to determine what individuals generally think, feel, and do when experiencing stressful situations. Of course, you may respond differently depending on the situation, but think of what you usually do when you are under a lot of stress. Using the scale below, indicate the extent to which agree or disagree with the following statements. Please answer according to what you *actually* think/feel/do rather than what you think is the correct response.

Strongly Disagree	Disagree	Slightly Disagree	Neutral	Slightly Agree	Agree	Strongly Agree
1	2	3	4	5	6	7

Generally, in stressful situations...

1. I weigh out many options before choosing how to take action.	1	2	3	4	5	6	7
2. I can't focus on anything when I am upset.	1	2	3	4	5	6	7
3. It's hard to think of different ways of dealing with the situation	1	2	3	4	5	6	7
4. I control my thoughts and feelings by putting the situation in context.	1	2	3	4	5	6	7
5. I can remain in control over my thoughts and emotions.	1	2	3	4	5	6	7
6. It's difficult let go of intrusive thoughts or emotions.	1	2	3	4	5	6	7
7. It's hard for me to put things in perspective when I'm upset.	1	2	3	4	5	6	7
8. I have a hard time managing my emotions.	1	2	3	4	5	6	7
9. I take the time to see things from different perspectives before reacting.	1	2	3	4	5	6	7
10. I feel like I lose control over my thoughts and emotions.	1	2	3	4	5	6	7
11. It's hard for me to shift my attention away from negative thoughts or feelings.	1	2	3	4	5	6	7
12. I find it easy to look for something positive, even when I am stressed.	1	2	3	4	5	6	7
13. I control negative thoughts and emotions by modifying the way I think about the situation.	1	2	3	4	5	6	7
14. It is easy for me to ignore distracting thoughts.	1	2	3	4	5	6	7
15. It's hard for me to ignore negative emotions once they have been provoked.	1	2	3	4	5	6	7
16. I can think of multiple coping options before deciding how to respond.	1	2	3	4	5	6	7

17. I get easily distracted by upsetting thoughts or feelings.	1	2	3	4	5	6	7
18. I approach the situation from multiple angles.	1	2	3	4	5	6	7
19. My thoughts and emotions interfere with my ability to concentrate.	1	2	3	4	5	6	7
20. I take the time to think of more than one way to resolve the problem.	1	2	3	4	5	6	7
21. It is easy for me to shift my attention to other things if I am upset.	1	2	3	4	5	6	7
22. I manage my thoughts or feelings by reframing the situation.	1	2	3	4	5	6	7
23. I find it difficult to think of many options for resolving the situation.	1	2	3	4	5	6	7
24. Putting a positive spin on a bad experience comes fairly easy to me.	1	2	3	4	5	6	7
25. I find it easy to set-aside unpleasant thought or emotions.	1	2	3	4	5	6	7
26. It is easy for me to reassess a negative experience into a positive one.	1	2	3	4	5	6	7
27. I can easily suppress upsetting memories.	1	2	3	4	5	6	7
28. I take the time to think of several ways to best cope with the situation before acting.	1	2	3	4	5	6	7

Appendix L

Connor-Davidson Resilience Scale (CD – RISC)

Considering your experiences over the last month, please rate how true the following statements are.

	0	1	2	3	4			
	Not true at all	Rarely true	Sometimes true	Often true	True nearly all of the time			
1. I am able to adapt to change				0	1	2	3	4
2. I have close and secure relationships				0	1	2	3	4
3. Sometimes fate or God can help				0	1	2	3	4
4. I can deal with whatever comes				0	1	2	3	4
5. Past success gives me confidence for new challenges				0	1	2	3	4
6. I see the humorous side of things				0	1	2	3	4
7. Coping with stress strengthens me				0	1	2	3	4
8. I tend to bounce back after illness or hardship				0	1	2	3	4
9. Things happen for a reason				0	1	2	3	4
10. I put in the best effort no matter what				0	1	2	3	4
11. I can achieve my goals				0	1	2	3	4
12. When things look hopeless, I don't give up				0	1	2	3	4
13. I know where to turn for help				0	1	2	3	4
14. Under pressure, I focus and think clearly				0	1	2	3	4
15. I prefer to take the lead in problem solving				0	1	2	3	4
16. I am not easily discouraged by failure				0	1	2	3	4
17. I think of myself as a strong person				0	1	2	3	4
18. I can make unpopular or difficult decisions				0	1	2	3	4
19. I can handle unpleasant feelings				0	1	2	3	4
20. I have to act on a hunch				0	1	2	3	4
21. I have a strong sense of purpose				0	1	2	3	4
22. I am in control of my life				0	1	2	3	4
23. I like challenges				0	1	2	3	4
24. I work to attain my goals				0	1	2	3	4
25. I take pride in my achievements				0	1	2	3	4

