ANXIETY, DEPRESSION AND HOPELESSNESS IN ADOLESCENTS:
A STRUCTURAL EQUATION MODEL

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Dedication

To my stunning and brilliant mother who never lets me down. Her strength and wisdom inspires my potential, pushing me to be half of the woman she is. To my devoted father, who constantly shows me how to see the big picture, never forgetting to instil his strength and compassion. To my brother Nathan whose tireless perseverance motivates me in ways I can’t describe. To my sister Kelsey whose vibrant charisma and honesty never ceases to amaze me. To my brother Garret whose humour and support proved to be invaluable to me at times. To Neil, who showed me how to love without boundaries and reminded me how wonderful life in love can be. To all my wonderful friends who believed in me, constantly encouraging and supporting me throughout this process.

In loving memory to two extraordinary individuals who were integral in shaping my success. My grandmother’s unwavering love and compassion for learning spurred me on when I was too exhausted to keep going. Jordan, your passion for life reminded me to live and savour every moment. Despite your physical absence, I am reminded with every step of my journey that you are watching and inspiring me to be a stronger and better person every day of my life.
Abstract

This study tested a structural model, examining the relationship between a latent variable termed demoralization and measured variables (anxiety, depression and hopelessness) in a community sample of Canadian youth. The combined sample consisted of data collected from four independent studies from 2001 to 2005. Nine hundred and seventy one (n=971) participants in each of the previous four studies were high school students (grades 10-12) from three geographic locations: Calgary, Saskatchewan and Lethbridge. Participants completed a battery of self-report questionnaires including the Beck Anxiety Inventory (BAI), Beck Depression Inventory-Revised (BDI-II), Beck Hopelessness Scale (BHS), and demographic survey. Structural equation modeling was used for statistical analysis. The analysis revealed that the final model, including depression, anxiety and hopelessness and one latent variable demoralization, fit the data (chi-square value, \(X^2\) (2) =7.24, \(p<.001\), goodness of fit indices (CFI=0.99, NFI=0.98) and standardized error (0.05). Overall, the findings suggest that close relationships exist among depression, anxiety, hopelessness and demoralization. In addition, the model was stable across demographic variables: sex, grade, and location. Further, the model explains the relationship between sub-clinical anxiety, depression and hopelessness. These findings contribute to a theoretical framework, which has implications with educational and clinical interventions. The present findings will help guide further preventative research in examining demoralization as a precursor to sub-clinical anxiety and depression.
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Chapter One: Introduction

The relationship between the development of depression and anxiety within an adolescent population is a promising area of inquiry. The increase in sub-clinical levels of anxiety and depression, where symptoms are present but do not meet criteria for a depressive or anxiety disorder, has been linked to developmental changes in adolescence. Although research has increased over the last two decades, findings lag far behind the adult literature. Moreover, a gap remains in understanding the relationship among sub-clinical levels, etiology, measurement, and the nature of development of emotional disorders. This lack of focus has had implications for early modes of intervention and subsequent prognosis of adolescent anxiety and depression. The present study attempts to further explore the relationship between sub-clinical anxiety and depression in a large sample of adolescent students.

Defining what is normal and abnormal adolescent behaviour is not a simple task for researchers. Most would agree that there is an array of factors that contribute to normal and abnormal behaviour (Santrok, 2001; Ingram & Price, 2001). The field of developmental psychopathology has largely focused on identifying, exploring, and confirming developmental pathways of adolescent problems and disorders. Conceptualizing the relationship between risk factors of particular disorders in adolescents holds implications for ways in which these individuals may be aided in adapting and coping with adulthood. Depression and anxiety are the two most common reasons for adolescent referrals for treatment. It has become clear within the literature that depression and anxiety are major, pervasive, and debilitating aspects of adolescent development (Santrok, 2001).
Often referred to as the common cold of mental disorders, both anxiety and depression are debilitating conditions that greatly impair psychological, social and emotional well-being. Moreover, such consequences place significant strain on interpersonal relationships and present an economic cost to society (Dozois & Dobson, 2004; Barlow, 2002; Gotlib & Hammen, 2002). Given the fact that anxiety and depression tax the resources of both individuals and society, it is important for researchers to develop models to aid in understanding the development of these disorders; especially in terms of the relationship between sub-clinical anxiety and depression in normal populations. Understanding sub-clinical anxiety and depression can facilitate the development of early intervention strategies.

For millennia, humans have experienced and sought to understand anxiety and depression (e.g., Plato and Aristotle). Researchers continue to develop and test theory-based constructs to explain the disorders. Anxiety and depression possess aspects that are both normative and debilitating to one’s life. Sigmund Freud aptly summarized this viewpoint in the following words “One thing is certain, that the problem of anxiety is a nodal point, linking up all kinds of the most important questions; a riddle of which the solution must cast a flood of light upon our whole mental life” (Barlow & Durand, 2005, p. 250).

Several psychological theories have examined anxiety, depression and the relationship between both constructs. For example, psychodynamic, behavioural, cognitive and, more recently, integrated theories, have all attempted to explain the distinct and overlapping features of anxiety and depression.
Psychodynamic theories have focused mainly on underlying unresolved conflicts that individuals express through the symptomologies of anxiety and depression. Sigmund Freud viewed anxiety as a defence mechanism for repressed and unconscious impulses (Davison, Neale, Blankstein, & Flett, 2005). Similarly, many psychoanalysts have pondered the point that depressed individuals harbour unconscious negative feelings towards those they love, causing anger to turn inward (Davison et al., 2005). Freud posited that this is a defence mechanism to cope with socially unacceptable feelings. Unfortunately, there is little empirical evidence to support these psychodynamic assumptions (Barlow & Durand, 2005; Davison, et al., 2005; Kandel, 1999).

Behavioural theorists view anxiety and depression as a product of learning, where learned behavioural patterns contribute to the development of symptoms through processes such as modeling and classical conditioning. Furthermore, anxiety has been linked to a sense of little to no control over perceived future events within the environment (Chorpita & Barlow, 1998). Similarly, stressful events are identified in the etiology of depression (Barlow & Durand, 2005). The behavioural model more or less assumes that psychopathology is environmentally determined.

Cognitive theorists posit that assessing unique cognitive content would enable clinicians and researchers to distinguish between anxiety and depression (Beck & Perkins, 2001). More specifically, depressive cognitive content would reflect related cognitions conceptualized in Beck’s Negative Cognitive Triad consisting of negative assessment of self, the world and future (Beck, 1976). It was hypothesized that depression reflects a cognitive content that encompassed themes of negative self-evaluation, hopelessness and a general pessimistic assessment of the world (Clark, Beck
& Stewart, 1990). By contrast, cognitive theorists have proposed that anxiety-based cognitive content is related to concerns of physical or psychological threat (Beck & Perkins, 2001). Yet, understanding anxiety and depression as unique constructs has been further clouded by the consistent finding of high correlations of scores on self-report scales assessing symptoms of depression and anxiety (Dobson, 1985).

Behavioural and cognitive theories of depression and anxiety empirically support and fundamentally inform clinical practice. Over time, behavioural and cognitive models for depression and anxiety have become more complex. For example, the hopelessness and negative cognitive triad models of depression have evolved into the stress-diathesis model of depression. Similarly, integrating numerous factors, Barlow (2000; 2002) has developed the triple vulnerability theory that includes anxiety and related disorders. This theory speculates that the development of anxiety and related disorders, most commonly depression, are a result of numerous vulnerabilities that interact with environmental stressors (Barlow, 2000; 2002). This interaction encompasses aspects of both biological contributions and psychological contributions that underlie individual vulnerability.

During the 1980s, cognitive theorists investigated the overlap of anxiety and depression partly in response to diagnostic and assessment issues regarding the relationship of each unique construct (Beck & Perkins, 2001). Diagnostically, anxiety and depression are two distinct constructs, both encompassing multiple forms. Recent findings, however, have demonstrated that anxiety and depression overlap. For example, Barlow (2002) stated that the genetics of anxiety and depression are closely related, as is the neurobiology and the nature of individual vulnerability to each. Barlow (2002) points out that a temporal relationship may exist in that some people with vulnerability react
with anxiety to life stressors and others react further to become depressed. While conceptually distinct, anxiety and depression are psychological phenomena more likely to have a complex etiological relationship. The need to understand etiology highlights the importance of studying the relationship between sub-clinical anxiety and depression in normal populations.

In sum, a current theory involving anxiety and depression recognizes the importance of drawing attention to the overlap of symptoms. Examining the relationship between anxiety and depression and attempts to discriminate one syndrome from the other seem to be the current theoretical and clinical focus among researchers and clinicians.

In recent years, there has been an increase in empirical support and research in the domains of anxiety and depression. Models of anxiety and depression have been developed from different theoretical backgrounds. Furthermore, models of anxiety and depression have guided the development of effective treatments. For years, a controversy has existed between the nature and nurture debate as it relates to psychopathology. In recognition of the importance of the gene-environment interaction, the diatheses-stress model was developed to explain why individuals who inherit the tendency to express a specific trait or behaviour might not exhibit the behaviour unless an environment stressor triggers activation of the trait (Barlow & Durand, 2005).

In treatment, precise diagnosis is paramount when formulating appropriate care plans for individuals dealing with the crippling effects of anxiety and depression. Anxiety and depression describe distinct phenomena, at least theoretically: anxiety is based upon fear while depression is based upon loss (Rutter & Rutter, 1992). What then makes a
person vulnerable to developing anxiety, depression, or both? An environmental stressor enters the diathesis to activate the expression of a particular trait or behaviour. Multiple paths to a given outcome of anxiety or depression must exist because not all individuals with an inherited vulnerability express or terminate a particular trait or behaviour, or encounter a threshold of environmental stressors. Complex interactions between psychological and biological factors may alter various stages of development leading to different pathways and developmental outcomes. Researchers not only examine what makes people exhibit particular disorders, but also how those who are at risk or who are vulnerable might be protected (Barlow & Durand, 2005).

While a substantial effort has been invested in the treatment of acute phases of anxiety and depression, few studies examine risk and vulnerability factors, or factors that may prevent sub-clinical anxiety and depression from reaching clinical levels. There is a current need for research on models that conceptualize sub-clinical states of anxiety and depression. For example, epidemiological studies that have examined adolescent populations have focused mainly on the prevalence of anxiety and depressive disorders as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) or International Classification of Diseases (ICD) criteria. By placing emphasis on sub-clinical symptoms of anxiety and depression, researchers may begin to understand the etiology that underlies the development of anxiety and depression as opposed to normative development.

This study proposes a theoretical framework that delineates a model of sub-clinical anxiety, depression and hopelessness examined in a sample of Canadian youth. The proposed study will extend all previous integrated models by including the
examination of model stability across age, sex and geographic location. This model focuses on a younger population (adolescents) to examine whether developmental similarities exist between anxiety, depression and hopelessness. In addition, similarities between urban and rural populations will be examined to confirm model stability. Finally, the present model will examine sex and age effects.

This study will examine a model that explains the relationship between anxiety and depression and hopelessness in development terms, focusing on self-reported emotional distress such as anxiety, depression, hopelessness, as well as demographic factors such as sex, age and geographic location.

Past studies have described the relationship between anxiety and depression. In this study, it is hypothesized that the typical relationship between anxiety and depression will remain stable across age, sex and geographic location. For example, anxiety and depression arise separately in development (time), with one common factor accounting for the relationship across grades ten to twelve (using grade as a proxy to age). Similarly, the effects of sex and geographic location (urban / rural) on the relationship between anxiety and depression are examined.

A variety of factors contribute to our understanding of these disorders. It is, therefore, important to describe each construct. Parts A and B of chapter II contain literature reviews providing accounts of anxiety and depression, respectively. These sections also include a description of current diagnostic criteria and issues related to measurement. Additionally, variables important to the research questions are described. These include age, sex and geographic location. Part C of chapter II reviews the issue of
comorbidity. These sections conclude by illustrating the importance of detecting sub-clinical symptomology in youth.

Part D of chapter II provides a testable model that describes the relationship between anxiety, depression and hopelessness. In addition, the model provides the basis to test whether its path constructs are stable across sex, geographic location and age. Part E of chapter II outlines the study’s design, the relevance of structural path analysis, and details specific research questions.

Chapter III, the methods section, presents a description of the self-report inventories used to measure anxiety, depression and hopelessness. Descriptions of the psychometric properties of the Beck Anxiety Scale, Beck Depression Scale and Beck Hopelessness Scale are included. Sections of chapter III also present descriptions of the sample, data collection procedures and data analysis. Chapter IV presents the results of the present study. Chapter V discusses findings in relation to previous research, and implications of models for anxiety and depression prevention in youth. In conclusion, study limitations are reviewed. In order to help the readers become familiar with the various concepts used in the thesis, a glossary of terms is provided in Appendix A.
Chapter Two: Literature Review

Adolescent Development

What is Adolescence?

For many, adolescence is a time in life that coincides with many milestones of development (e.g., the onset of puberty or high school graduation). However, these celebratory events do not define the transition between childhood and adulthood. Throughout the literature, the term adolescence has been difficult to concretely define (Hine, 1999; Santrok, 2001). This may be due to the fact that adolescence requires consideration of multiple factors including age and contextual influences. In addition, adolescence is a cultural and social phenomenon causing difficulty in defining the beginning and endpoints of this development stage (Santrok, 2001). For example, within the Jewish community, adolescent males are considered an adult at the age of thirteen, and is directly associated with the Bar Mitzvah ceremony (a formal age of maturity). In contrast, many Western societies define adolescence within a much broader time period. For example, the United States recognizes adolescence between the ages of thirteen to twenty four years of age (Hine, 1999). The literature has suggested that adolescence is a relatively new phenomenon within Western societies and is viewed as a by-product of social pressures specific to culture (Hine, 1999). Compared to many other cultures, western societies have not defined adolescence as distinct phase of life. This causes uncertainty as to when adulthood begins, and how an individual makes the transition from childhood to adulthood.
Developmental Issues

Given that adolescence is a developmental period that constitutes potential for conflict as well as opportunities for growth, understanding the conflicts that arise for individuals attempting to overcome barriers and obstacles along their developmental path may provide insight into the development and expression of emotional difficulties. Numerous developmental theorists have attempted to explain the changes that occur in the period between childhood and adulthood. Many theories have been developed in an attempt to explain development in terms of stages, focusing on specific changes that occur at each stage, and progressing to the next stage in order to reach a functional level as an adult. For instance, Piaget’s theory emphasized that a critical stage in adolescence was developing abstract thinking (Piaget, 1972). Another stage theorist, Eric Erikson (1968), focused on psycho-social stages within adolescent development proposing that this is a period where each adolescent formulates their own identity. The risk of identity confusion is included in Erikson’s psychosocial stages identified as occurring in the transition between childhood and adulthood. Erikson (1968) describes this risk as feelings of isolation, doubt, anxiety and indecision. Furthermore, these traits may interfere with the individual’s ability to enter the next stage of development.

Understanding Adolescence: What Matters?

Adolescent Processes

Development has typically been described in the literature in terms of time periods. Three processes commonly determine adolescent development: biological, cognitive and socio-emotional (Santrok, 2001).
Biological processes consist of individuals’ unique biological make-up that genetically influences behaviour and development. Biologically determined processes of development were dominant viewpoints in the early twentieth century. Stanley Hall often thought of as the father of scientific study of adolescence, applied the scientific and biological dimensions of Darwin’s Theory of Evolution in the study of adolescence. Hall believed that development was predominantly biologically determined, and environmental factors played a minimal role. It is important to note that although he viewed development as predominantly biological, Hall did incorporate environmental accounts for change in development during adolescence (Hall, 1904). Current thought emphasizes the fact that biology does play a critical role in development, although theories are now attempting to identify how biological processes interact with environmental processes in adolescent development.

Cognitive processes incorporate that which changes in individual thinking throughout the course of development. This view emphasizes that adolescents have sophisticated thinking abilities and are motivated to understand and construct their own cognitive worlds. Theorists focus on individual differences involving complex cognitive abilities including problem solving abilities, memory capacity, decision making. Current strides within this perspective include examining how individuals within this age group apply critical thinking and are able to adapt competently within their environment. Cognitive processes have implications for creating educational programs during the process of development.

Socio-emotional processes involve the development of individual emotions in relation to social context and other people. Research suggests that important contexts in
adolescent development include family, peer, school and cultural contexts. In addition to particular contexts in which individuals are encompassed, social and personality development (e.g., gender roles, identity, sexuality and achievement) interact with environmental situations to further complicate this process. Research in this field has focused on developmental and contextual factors involved in healthy or unhealthy development.

In summary, biological, cognitive and socio-emotional processes are intricately related and constantly interacting. Changes in adolescent development are an outcome of all processes involved.

*Normative Versus Atypical Development*

One of the greatest challenges in studying adolescence is reaching an understanding of the nature of normal versus abnormal development, and the relationship between the two. Given that the literature on adolescent psychopathology lags behind that of adult psychopathology, it is not surprising that there is a gap in the literature in understanding adolescent psychopathology. Researchers are still unclear on several issues pertaining to the development of psychopathology in adolescence. Mash and Dozois (1996) identified issues typically debated in the literature when defining normal versus abnormal development.

First, many theorists have questioned whether the development of psychopathology in adolescence is a result endogenous to the child, an outcome due to environmental circumstance, or a reaction to both. Second, many have pondered how to conceptualize the development of psychopathology in terms of being either categorically distinct from normal functioning or, being an extreme point on a normally occurring
continuum of development. Third, psychopathology can be viewed as a static or dynamic process influenced by both developmental individual changes and changes within environmental context. Despite differences in viewpoints, most theorists agree that adolescents are faced with numerous developmental demands, from hormonal changes to emotional regulation, in an attempt to successfully adapt and orient themselves towards a normative path. Moreover, psychopathology lends itself to a form of maladaptation resulting from difficulties in adapting to developmental demands. It becomes clear in the literature that understanding atypical development is only possible when looking at the whole picture of normative development.

Rationale for the Study of Vulnerability Processes

Many benefits have been identified in studying the vulnerability processes underlying atypical development in adolescence. Research within the area of developmental psychopathology has demonstrated that adolescence is a critical period for understanding etiology and course of emotional disorders (Price & Lento, 2001). In addition, the study of atypical development may also enhance our understanding of normative processes. Chorpita and Barlow (1998) developed a model to test the affects of early life experiences and perceived lack of control, on the development of specific cognitions that specific events may be out of one’s control. Findings demonstrated that the specific cognitions tested represent a psychological vulnerability for the development of both anxiety and depression. Chorpita and Barlow (1998) inferred that if a lack of perceived control over a given situation makes one vulnerable to maladjustment, then perceived control might contribute to normative development.
Unfortunately, anxiety, depression, and hopelessness are often dismissed as developmentally normal components of a teenager’s life (Price & Ingram, 2001). This perception may explain the lack of existing research about sub-clinical emotional difficulties in adolescence. For example, if an adolescent reports four out of five targeted symptoms they would not be diagnosed with clinical anxiety or depression. Rather, they may represent an intermediate anxiety or depressed mood state in which symptoms are present but do not meet clinical criteria. Therefore, a conceptualization of sub-clinical maladjustment and psychopathology is also necessary to understand the difference between natural emotional variation in adolescents and the role of sub-clinical symptoms in the etiology of clinical anxiety and depression (Price & Ingram, 2001).

A number of theorists have attempted to explain the complexities of adolescent development and broaden current understanding. Erikson (Erikson, 1968, 1970) and Piaget (Piaget, 1954, 1972) have added to the knowledge base of normative development and have made important contributions. In addition, theorists have also examined abnormal development in adolescence by focusing on individuals who are generally at risk. However, no single theory or model has been able to capture the whole picture of normative or abnormal development. The inability of a single theory to explain all aspects should not be seen as a shortcoming. Rather, continuing to study more specific aspects of abnormal development as opposed to a general model would be advantageous in preventative approaches.

The remainder of this literature review will attempt to provide theoretical and empirical rationale for specifically examining anxiety, depression and hopelessness symptoms amongst male and female adolescents.
Anxiety in Adolescence: A Historical Perspective

Anxiety Versus Fear

Diverse explanations of anxiety may in part be due to the fact that anxiety is not only a common experience, but it can also be a functional component of everyday life. Adolescents often experience anxiety during this transitional phase related to social identification and interpersonal issues (Castellanos & Hunter, 1999). Given that anxiety is such a complex construct, it is not surprising that it has been linked with performance levels, intelligence, survival and pathology. Problems arise when anxiety levels increase in intensity causing many adolescents to become developmentally dysfunctional. Furthermore, severe levels of anxiety have been associated with negative outcomes including peer relationship difficulties, academic issues, and future onset of comorbid disorders including major depressive disorder (Bernstein & Borchardt, 1999).

Also associated with a feeling of impending danger, anxiety is a normal response that is both appropriate and necessary to avoid dangerous situations. Moreover, anxiety symptoms mirror those of fear, which is similarly produced by present or threatening danger. For example, when a dangerous situation is readily apparent (i.e., when a barking dog is charging at you) then a fear emotion is evoked (Marks, 1977). Conversely, when a situation is ambiguous and slightly incomprehensible (i.e., walking down a street and not knowing where the loud barking is coming from) an anxious emotion may be induced. It is not surprising then that fear and anxiety often overlap.

Fear and anxiety are commonly experienced by people at minimal levels and are considered normal and adaptive in many situations. Low levels of anxiety and fear seldom elicit an avoidance reaction and tend to decrease in intensity with time and
explanation (Marks, 1977). At this level, these emotions do not typically require professional attention. Furthermore, anxiety and fear are normative emotional reactions that increase physiological arousal to better equip individuals in preparing for anticipated danger.

Physiological arousal is often associated with anxiety and includes skin turning pale, sweating, pupil dilation, increased breathing, heart rate, blood pressure and blood flow to major organs. Parallel to physiological changes is a change in biochemical levels including secretion of epinephrine and norepinephrine. When this physiological-biochemical reaction is prolonged in healthy individuals, they become higher risk of becoming depressed (Marks, 1977).

Theories of Anxiety

Keeping in mind that explanations of anxiety have for centuries been derived from theories and models popular at the current time. Humans have always accepted that behaviours or traits can be caused by outside entities. The supernatural model was the accepted theory of understanding psychopathology for much of our recorded history (Barlow & Durand, 2005). Through the lens of this model, unexplained and irrational behaviour was often seen as a sign of evil. During the 14th and 15th century, insanity was believed to be a natural phenomenon caused by mental or emotional stress (Barlow & Durand, 2005). Mental anxiety and depression were recognized as an illness caused by demonic influence, but they were seen as curable. Treatments at this time included exorcism, bloodletting and some more humane treatments like ointments or baths (Barlow & Durand, 2005). From a historical perspective, anxiety and depression have shared symptoms and treatments dating back to the 14th century.
To date, varying theories account for the etiology of anxiety. Consistent with the diathesis-stress model of psychopathology, anxiety has been attributed to neurochemical (Gray & McNaughton, 1995) and genetic factors (Turner, Beidal, & Costello, 1987), which in combination with life stressors significantly increase vulnerability to the disorder. Turner et al. (1987) found that children who had parents with anxiety disorders were more likely to be anxious, report school difficulties, worry about family members and themselves, exhibit somatic complaints, and spend time alone compared to the control groups. Barret (2000) also outlines important developmental factors that are especially salient to the study of adolescent anxiety. First, infant temperament can predict later anxiety, which seems to support the presence of a biological vulnerability, and when combined with insecure attachment, the risks are heightened. Early experiences with uncontrollability, which can be due, in part, to overly controlling parenting, can also lead to increased anxiety (Barlow et al., 1996). In adolescence, when abstract reasoning increases and peers become more influential than parents, fear of negative evaluation, fear about future, and social fears develop (Barret, 2000).

There is mounting evidence that supports an integrated model for anxiety, which includes a diversity of factors (Barlow, 2002). Placing these factors together has become the scope of research used to examine the etiology and prevention of anxiety disorders. One of the first integrated models developed was the Diathesis-Stress model. Barlow (2000, 2002) describes a theory of anxiety and related disorders coined the “Triple Vulnerability Theory”. Utilizing aspects of the diatheses-stress model, Barlow (1988, 2000, 2002) describes three vulnerabilities (or diatheses) that contribute to anxiety,
anxiety disorders and related emotional disorders. Finally, the tripartite model has been a focus for many researchers because of the integration of anxiety and depression.

**Diagnostic Criteria for Anxiety**

According to the DSM-IV-TR, anxiety disorders can be broken down into the following types: panic disorders, agoraphobia, specific phobia, social phobia, obsessive-compulsive disorder, posttraumatic stress disorder, acute distress disorder, generalized anxiety disorder, anxiety disorder due to a general medical condition, substance-induced anxiety, and anxiety disorder not otherwise specified (American Psychological Association, 2000, p.429). Several symptoms are common to the range of anxiety disorders, such as “a sense of uncontrollability focused on possible future threat, danger, or other anticipated, potentially negative events,” (American Psychological Association, 2000, p.429) a shift towards self-centeredness, physiological symptoms (e.g., sweating, heart palpitations, trembling, etc.) (Barlow, Chorpita & Turovsky, 1995).

Common symptoms become problematic when using existing classification systems, raising the issue of discriminate validity (Dozois & Dobson, 2004). Researchers speculate that overlapping symptoms are significant in understanding prevention, treatment and etiology of emotional disorders (Andrews, 1996; Brown & Barlow, 2002). Moreover, given criteria listed by classification systems such as the DSM-IV, identification of sub-clinical symptoms may be highly compromised (Eisen & Kearney, 1995). It is, therefore, crucial to understand the extent of sub-clinical symptoms and the overlapping relationship with that of established criteria as set in the DSM-IV.

Research conducted by Breton, Bergeton, Valla, Berthiaume, and Gaudet (1999) demonstrated that when the criteria for clinical significance is accounted for, prevalence
rates of anxiety in children and adolescence increased to 70%. These findings emphasize the importance of early detection of sub-clinical symptoms that may otherwise go undetected using classification systems that focus primarily on clinical states. Identifying sub-clinical signs and symptoms is of considerable importance for early detection and intervention.

**Prevalence of Anxiety**

Anxiety disorders are among the most prevalent diagnosis within the United States and they are the most common type of mental disorder found in adolescents (Kasahni & Orvaschel, 1988). Prevalence rates for anxiety in a community sample of adolescents vary considerably. Depending on the specifics of methods, stringency of diagnostic criteria, and other particularities of a study, clinical anxiety disorders have been estimated to occur in 5.7% to 28.8% of community adolescents (Costello & Angold, 1995; Essau, Conradt & Petermann, 2000; Kashani & Orvascal, 1988; Lewinsohn et al. 1993; Verhulst, van der Ende, Ferninand & Kasius, 1997; Woodward & Fergusson, 2001). According to the Canadian and Community Health Survey, 2.3% of adolescents and young adults (age 15 to 24) have had panic disorder, 0.8% agoraphobia, and 4.8% social phobia (Statistics Canada, 2002). Other studies report lifetime prevalence rates for anxiety disorders in community samples of adolescents ranging from 9.15 to 18.60% (Lewinsohn et al., 1993; Essau et al., 2000). More specifically, panic disorder ranged from 0.50 to 1.19%, agoraphobia from 0.60 to 4.10%, social phobia from 1.46 to 1.60%, specific phobia from 2.12 to 3.50%, and obsessive-compulsive disorder from 0.6 to 1.3%.

One possible source of discrepancy between studies is the diagnostic criteria used. While it is important to know the estimated number of adolescents who do meet criteria
for a clinically diagnosable disorder, it also seems that this information does not provide a complete picture of the anxiety experienced by this population. Few studies have examined the prevalence of sub-clinical anxiety symptoms in adolescents, despite findings suggesting that sub-clinical levels of anxiety can also cause considerable levels of distress.

The onset of some types of anxiety disorder tends to be in early adulthood, whereas others tend to emerge in childhood or adolescence. Parallel with the literature on the prevalence of anxiety disorders, onset typically occurs in adolescence (Dozois & Dobson, 2004). This finding highlights the importance of identifying early indicators of anxiety within this population due to the fact that many adolescents experience mild to moderate (sub-clinical) levels of anxiety. Consequently, sub-clinical levels of anxiety in adolescents may have adverse affects on development (Ohannessian et al., 1996). Unfortunately, compared to the empirically-based knowledge about depression, knowledge about adolescent anxiety is less available, perhaps because anxiety, specifically as a research focus, has been largely neglected (Ohannessian et al., 1999).

Models of Anxiety

Two major streams of models of anxiety have evolved and highlight biological and psychological bases: animal models of anxiety and human models of anxiety.

Animal Models of Anxiety

Animal models of anxiety have studied environmental factors that contribute to anxiety and are typically developed to induce a fear or anxious response to stimuli (Craig, Brown, & Baum, 2000). Anxiety and fear have similar behavioural and physiological symptoms but have different etiological roots. For example, anxiety is associated with
non-specific stimuli, commonly referred to as generalized unfocused response (Craig et al., 2000), while fear is often a result of an experienced danger in the immediate environment.

Due to ethical issues inherent in studying disruptive human emotion, animals have been frequently used to study conditions linked to anxiety, fear and phobias. Comparisons are then made between proposed animal models and underlying mechanisms encompassing human anxiety, assuming that the cause of the emotional response in the animal would be similar to human emotional response and therefore, assumptions are made in terms of treatment and etiology (Craig et al., 2000). Anxiety-evoking agents such as unpredictability, uncontrollability and negative experiences are used in both human and animal studies. The following animal models were developed to closely parallel theories that guide clinical practice and include conditioned emotional response, fear-potentiated startle and punishment-conflict and separation and abandonment models of anxiety.

*Conditioned emotional response.* The conditioned emotional response model was developed to produce an emotional response in animals using a series of pairings and non-pairings of an unconditioned stimulus (US), such as a shock, with a conditioned stimulus (CS), such as a tone (Craig et al., 2000). The unconditioned stimulus (US) and conditioned stimulus (CS) relationship has allowed researchers to ascertain if an emotional conditioning is developed, inhibited, or retarded. In time, the conditioned baseline behaviour response of the shock-tone pairing decreases and is known as a conditioned emotional response (CER). Consequently, when the shock and the tone are
not paired together, the tone represents a safe period to the animal and is able to inhibit fear and anxiety.

Within this paradigm, the presence or absence of the emotional response is predictable and expected. Therefore, this produced a conditioning procedure more closely resembling human fear experiences and is not easily generalized to anxiety. Given that anxiety is linked with unknown events, animal models that produce uncertainty in the environment by neither pairing nor not pairing the tone or shock would more likely give an anxiety-based reaction. The tone provides no information about the likelihood of the impending negative event. Research on this model of anxiety has demonstrated that unpredictable and uncontrollable experiences in an animal’s environment appear to produce a negative emotional state and often leave these animals susceptible to developing ulcers and tumours (Visintainer, Volpicelli, & Seligman, 1982).

*Fear-potentiated startle.* The fear-potentiated startle animal model enhances an acoustic startle reflex by presenting an auditory startle stimulus in the presence of a conditioned stimulus that previously was paired with a shock (Craig et al., 2000). A potential startle response only occurs following paired presentation conditioned stimulus and stressor. This model is unique in that it equates enhanced emotional response with the desired effect rather then using suppression of ongoing behaviours as an indicator of an emotional state. More importantly, this allows researchers to measure a distinct symptom of anxiety, compared with other animal models of affective disorders that are characterized by behavioural inhibition, such as depression and learned helplessness (Craig et al., 2000).
The fear-potentiated startle animal model has provided parallel similarities in increased sympathetic arousal often present in human cases of PTSD (Craig et. al., 2000). In addition, drugs that induce anxiety in normal people and enhance it in anxious people also increase the fear-potentiated startle in rats. Similarly, drugs that decrease anxiety in people also decrease the fear-potentiated startle in rats. These findings are important to this model because the demonstrated effects of these drugs cannot be attributed to a decrease in general performance.

Punishment-conflict. The punishment-conflict model uses operant techniques to elicit well-established behaviours, and then punishes the behaviours using aversive stimuli causing the behaviours to subside. The suppression in behaviour is thought to parallel the passive-avoidant component of anxiety.

The development of this model arose from the works of B.F. Skinner (1938, 1991, 1999) who discovered that a conditioned stimulus (e.g., shock) would suppress an animal’s response to eat. Furthermore, the environment elicits cues that repeated exposure is paired with punishment.

Separation and abandonment. The separation and abandonment model is based on the manipulation of social interactions (e.g., if you were to separate an animal from an object of attachment). This model has been utilized in studies using Rhesus monkeys. Rhesus infants that have been separated from attached objects have demonstrated fear responses in the absence of dangerous situations, comparatively linked to panic attacks in humans. Like anxiety, intensity and frequency of these behaviours are dependent upon type and amount of stress encountered.
**Human Models of Anxiety**

*State versus trait anxiety.* Based on a psychological approach to anxiety as an acute emotion and personality construct, Spielberger (1972, 1980, 1983) has focused on psychometric tools and individual differences when studying anxiety. Spielberger (1972, 1983) introduced a distinction between two types of anxiety: state and trait. This distinction has now become commonplace in literature and has been further studied. Lazarus (1991) defined state anxiety as “an unpleasant emotional arousal in face of threatening demands or dangers”. Conversely, trait anxiety has been defined as reflecting stable individual differences regarding the tendency to respond with state anxiety in anticipation of a threatening situation (Lazarus, 1991).

*Self-efficacy and anxiety.* This model proposes that self-efficacy is a key construct of anxiety. The basis of this assumption is that individual’s mood and linked behaviour are determined by their ability to control the environment (Craig et al., 2000). Self-efficacy can be defined as an individual’s expectations in achieving set goals, sense of control, personal agency and a focus on how one perceives their own success.

Individuals not only evaluate their own self-efficacy, but also take into account vicarious experiences such as observational learning and modeling (Marks, 1977). Many identified state variables such as arousal, appraisal, and social influence have been shown to affect ones evaluation on self-efficacy (Craig et al., 2000).

In addition, self-efficacy has demonstrated positive correlations with self-esteem and individual self-concept. Consequently, ones ability to cope with anxiety arousing events has been linked to low self-efficacy. It is not surprising then that avoidance behaviour of feared objects may not exclusively be controlled by the fearful stimulus, but
also by evaluations of self-efficacy. Findings have demonstrated that fear and self-efficacy are distinct predictors of anxiety and affiliated behaviour.

*Depression in Adolescence*

**The Nature of Depression**

The first episode of depression frequently occurs in mid- to late adolescence (Hammen, 2001) and has been shown to predict future adjustment problems in many areas including academic functioning (Puig-Antich et al., 1985), substance abuse (Rohde et al., 1996; Paton, Kessler, & Kandal, 1977) school dropout (Kandel & Davies, 1986), marriage (Kandel & Davies, 1986), unemployment status (Chiles, Miller, & Cox, 1980), and delinquent behaviour (Chiles et al. 1980; Kandel & Davies, 1986).

Fergusson and Woodward (2002) studied 1,265 children for a 21-year period and found significant adjustment issues in the cohort that developed depression during adolescence. These individuals were more at risk for developing anxiety disorders, substance abuse problems, nicotine dependence, decreased academic functioning, and higher rates of recurrent unemployment and recurrent depressive episodes than the non-depressed adolescents.

It has been suggested by Joiner (2000) that numerous variables such as stress, seeking negative feedback, seeking assurance, and avoidance of interpersonal conflict are involved with adolescent depression. Results demonstrated that interaction of variables within the model maintained the depressive process and increased individual vulnerability and likelihood of recurrence of future depressive episodes. The results demonstrate the importance of developing models that inform and test preventative efforts directed at the reduction of onset and relapse.
Diagnostic Criteria for Depression

Depression is a type of mood disorder described in the DSM-IV-TR (American Psychiatric Association, 2000). Major depressive disorder is most commonly referred to as “depression” and is characterized by depressed mood, loss of interest, sadness, change of appetite, somatic complaints (e.g., aches and pains), psychomotor changes (e.g., agitation), decreased energy and fatigue, a sense of worthlessness or guilt, impaired concentration, and suicidal ideation. In addition, a hallmark characteristic specific to depression is anhedonia: the inability to feel pleasure. In some instances, sadness is often replaced with irritability in children and adolescents. Melancholia, psychosis, and suicide attempts are often found less in children and adolescents (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, Perel, & Nelson, 1996). Conversely, psychomotor retardation, delusions and hypersomnia are more commonly found in adolescents and adults than in children (Dozois & Westra, 2004).

Prevalence of Depression

Lifetime prevalence rates for MDD have been estimated to range between 15 to 22%, while point prevalence rates range from 0.4 to 8.3% (Birmaher et al., 1996). In the Oregon Adolescent Depression Project (OADP) (which used a community sample of American high school students), Lewinsohn et al. (1993) found point prevalence rates for MDD (according to the DSM-III-R) at 2.57% (time 1) and 3.12% (time 2) and a lifetime prevalence rates of 18.48% (time 1) and 24.01% (time 2). In a Canadian sample, 6.3% of a sample of adolescents and young adults (age 14 to 24) met the criteria for MDD (Statistics Canada, 2002). Prevalence rates of self-reported depression in adolescents as
measured by the Beck Depression Inventory have ranged from 22% to 33% (Roberts, Andrews, Lewinsohn, and Hops, 1990).

Social Impact

It is apparent that devastating consequences are placed on depressed individuals impairing their ability to function and ultimately lowering quality of life. The impact of depression also has vast costs on society given that depression is the most common presenting concern in the mental health community (Dozois & Westra, 2004). Sub-clinical levels of depression are often dismissed as normal parts of an individual’s life. Unfortunately, sub-clinical levels of depression have a devastating impact on adolescents, increasing the risk for severe psychiatric condition (Gotlib & Hammond, 2002).

Judd (1994) also described subsyndromal symptomatic depression (SSD), in which adults did not meet the criteria for minor depression, major depression, and/or dysthymia, but experienced social dysfunction and disability. These dysfunctions included decreased functioning in social and occupational roles, increased days spent in bed, and health issues (hypertension, diabetes, gastrointestinal disease). SSD was also correlated with lifetime depressive symptoms (Johnson, Weissman, & Klerman, 1992 as cited in Judd, 1994), and was the second most important risk factor predicting MDD one year later (Howarth, Johson, Kerman & Weissman, 1992 as cited in Judd, 1994).

In a related study by Lewinsohn, Soloman, Seeley and Zeiss (2000), subsyndromal depressive symptoms were investigated in adults, older adults and adolescents. They found that clinical depression could be understood on a continuum with other degrees of depressive symptoms. Furthermore, they found that adolescents did
not need to exhibit clinical levels of depression to be at risk for substance abuse disorder, and the subsyndromal depressive symptoms predicted future MDD.

Given that research has been conducted to identify risk factors, it is unfortunate that more research is not available to explain how these risk factors interact in a population with sub-clinical symptoms that leave individuals vulnerable to depression. The present study explores the relationship between depressive and anxious symptoms within one structural model.

Past depression predicts future depression (Lewinsohn, Hoberman, & Rosenbaum, 1988). In fact, higher rates of reoccurrence have been linked to having a history of depressive episodes, double depression (a diagnosis that incorporates both major depressive disorder and dysthymia), increased duration of episodes, family history of mood disorders, and comorbidity (the co-occurrence of two disorders) of anxiety or substance abuse (Dozois & Westra, 2004). A question that consistently surfaces is: do the mechanisms associated with the initial onset of depression different from recurrent episodes? Further research is needed to understand the etiologic structural path mechanisms associated with the initial onset of depression (most commonly found in adolescence), but it may be possible that high sub-clinical levels may predict this early onset.

In support of this hypothesis, research indicates that elevated depressive symptoms appear to be a strong risk factor for diagnosis of depression. As a result, sub-clinical symptoms are not insignificant. Distinguishing sub-clinical depression from normative transient sadness is important (Lewinsohn, Soloman, Seeley, & Zeiss, 2000). Studies have neither focused on the occurrence of depressive symptoms in non-referred
adolescents nor examined the risk factors that play a role in how these symptoms develop. The present study investigates depressive symptoms as a risk factor in a high school aged sample.

Models of Depression

Historically, three major theoretical models of depression have dominated throughout the 20th century: biological, intrapsychic and environmental approaches. Biological approaches posit that vulnerability to depression is a result of chemical imbalances. Specifically, biological models emphasis disequilibrium of neurotransmitters alluding to depressive symptoms being a brain-related dysfunction. While these reductionist models are still currently present within the medical model, in part because pharmacological treatment for depression can be successful, these models co-exist with diathesis-stress approaches.

Intrapsychic models first gained popularity when Sigmund Freud founded the psychoanalytic approach to understanding psychopathology. Freidians assume that emotional disturbances and disorders originate due to unresolved internal conflicts from ones childhood (Hammen, 2001). Modern versions of the intrapsychic wave followed with Beck’s cognitive model and Blatt’s model of depressive personality styles. These models changed the scope of the intrapsychic perspective and focused largely on how individuals perceive themselves and the world. An individual’s perspectives were, therefore, viewed as a product of experience and learning (Hammen, 2001). Modern versions of intrapsychic models currently exist and are continually tested and refined.

Alloy and Abramson (1979) found that individuals with depressed mood, even when experimentally induced, demonstrated a clear and realistic ability to control an
outcome compared to the non-depressed controls, which demonstrated an illusion of control in their environments. It appears that depressed individuals have the ability to view their lives through a realistic lens. Large empirical bases from social psychological studies have found that retaining an illusion of control is an adaptive function of both anxiety and depression (Barlow, 2002).

Also associated with depression is suicidal behaviour. Lewinsohn, Rohde, and Seeley (1993b) found that 10% of depressed boys and 4% of depressed girls had a past suicide attempt. Depression in adolescence has also been found to predict subsequent depression in adulthood (Harrington, Fudge, Rutter, Pickles, & Hill, 1990). Kovacs et al. (1984) in a longitudinal study found that as many as 40% of those with depression may suffer a reoccurrence within five years.

*Biological Theories of Depression*

For decades theorists have pondered genetic predisposition to mood disorders. Research on mood disorders having a heritable component has offered some support for this predisposition. Success rates of pharmacological treatments of depression, such as selective serotonin reuptake inhibitors, suggest the importance of biological factors (Davison et al., 2005).

Evidence in the neurochemistry of mood disorders has come from research focusing on two neurotransmitters: norepinephrine and serotonin. The theory suggests that low levels of both norepinephrine and serotonin leads to depression. It has been suggested that serotonin plays an important role in neurotransmitter regulation. Unfortunately, the relationship between serotonin specifically and depression is not clear.
Current theorists in this area suggest that the balance of these two neurotransmitters is important for mood regularity (Barlow & Durand, 2005).

Genetic studies have offered some support for the biological theory of depression. Twin and adoption studies in an adult population have accounted for at least 50% of the variance in the transfer of mood disorders between parents and their offspring (Birmaher et al., 1996). Furthermore, adolescents are more likely to have depression when closely related relatives suffer from the same disorder (Cicchetti & Toth, 1998; Nurcomb, 1994; Rice, Harold & Tharper, 2002). Moreover, in monozygote twin studies, if one twin is diagnosed with depression, the other is more likely to have depression. Conversely, concordance rates are lower in dizygotic twins (Cicetti & Toth, 1998; Nurcombe, 1994; Rice et al., 2002).

Cognitive Theories of Depression

Cognitive approaches to depression have largely focused on how thoughts and personal beliefs play a role in depression. A variety of cognitive models have been have been proposed to explain the development of depression.

Aaron Beck (1967, 1987) developed a cognitive model of depression emphasizing that negative interpretations underlie the development of depression. Beck (1967, 1987) rationalized three levels of cognitive activity in the etiology of depression. First, beginning in childhood or adolescence, depressed individuals develop a negative schema, which Beck (1967, 1987) describes as a negative view of the world. Beck (1967, 1987) believed that schemata are perceptual sets that are largely acquired and learned. Furthermore, if an individual has a negative experience a negative schema may develop. Moreover, negative schemata are reactivated when similar situations arise.
Beck posited that depressed individuals would develop cognitive biases from past experiences and misperceive present reality. Negative schemata combined with cognitive biases leads to what Beck (1967, 1987) coined the negative triad: negative view of self, the world and the future.

Seligman (1974) influenced the cognitive perspective by developing the learned helplessness theory of depression, which posits that individuals who endure repeated traumas or stressful experiences and perceive their situation as uncontrollable develop a sense of helplessness that then leads to depression. More specifically, Seligman (1974) believed that humans learn to be helpless. Initially studying classical conditioning in Pavlovian dogs, Seligman paired shocks with a conditioned stimulus to dogs that were kept in a shuttle box where they had to learn to escape the shock (1965). Seligman (1965) found that previous experience failing to escape the shock interfered in the dogs’ ability to learn to escape. Seligman (1974) later applied this theory to human depression with the central assumption that all animals learn that consequences are uncontrollable.

Limitations applying the learned helplessness theory of depression to humans and the inability to explain all aspects of depression lead to much need for revision. Critics of the helplessness theory (Comer, 2004) found that some depressed individuals hold themselves accountable and often blame themselves for the way they feel. This contradicted the learned hopelessness theory in that it did not explain how helpless individuals could hold themselves accountable. In response to inadequacies of the learned helplessness theory, Abramson, Seligman and Teasdale (1978) proposed the attributional reformation. The addition of the concept attribution to the learned helplessness theory added the component of the depressed individual’s explanation of behaviour (Abramson,
Seligman & Teasdale, 1978). The reformulated theory posits that individuals attribute negative life events to global, stable and internal factors (Abramson et al., 1978). Furthermore, these individuals are said to have a negative attributional style that leads to depression.

The reformulated theory belongs to the cognitive diathesis-stress family emphasizing an interaction between multiple factors leading to a subtype of depression termed learned helplessness (Abramson et al., 1978). The question that arises is whether helplessness is a cause of depression or a side-effect. Another question speculated upon is whether this psychological vulnerability is specific to depression. Research has found that anxiety and depression are triggered by the uncontrollability of stressful life events indicating that the specific psychological vulnerability may underlie many disorders (Barlow, 2002). It is because specific attributions could not account for all aspects of depression that Abramson, Metalski, and Alloy (1989) revised the learned helplessness theory emphasizing hopelessness as a core aspect of depression.

Hopelessness Theory of Depression

Abramson et al. (1989) proposed the hopelessness theory of depression to describe a sub-type of depression known as hopelessness depression. Abramson et al. (1989) de-emphasized specific attributions contributing to depression and highlight hopelessness as a symptom and cause to hopelessness depression. An important difference between helplessness and hopelessness is that research has found that helplessness is present in both anxious and depressed individuals. Conversely, only depressed individuals appear to become hopeless or give up trying to regain control
Hopelessness is an important construct developed by Abramson and colleagues, and has been associated with internalizing disorders. Although there is no specific mental disorder that is comparable to this construct, it is important to note that this construct has been linked to the core characteristics of depression. Previous research has indicated that hopelessness is a powerful predictor of adolescent suicidal ideation (Beck, Steer, Kovacs & Garrison, 1985). Previous studies (Beck et al., 1985; Swedo, Rettew, Kuppenheimer, Lum, Dolan & Goldberger, 1991) have indicated that hopelessness may be a stronger predictor of suicidal ideation than depression. Using the Beck Depression Inventory (BDI) and the Hopelessness Scale, Beck et al. (1985) found that depression severity did not discriminate between those who actually committed suicide compared to the rest of suicidal ideators, although higher Hopelessness Scale scores did. Surprisingly, hopelessness was also a better predictor of suicidal risk than direct measures of suicidal intent. Similarly, Swedo et al. (1991) found that hopelessness discriminated between those at risk for suicide and suicide attempters. Hopelessness Scale scores for the suicide attempters were almost twice as high as those for risk adolescents, and four times higher than those for the control group. Suicidal ideation and suicidal behaviour have increased in the adolescent population at an alarming rate over the past 50 years, the importance of understanding hopelessness and its prediction of suicidal attempt is evident (Statistics Canada, 2002).
These findings suggest the need for accurate evaluation of adolescents at risk for suicide and how the developmental process of suicide ideation plays a role in the relationship between anxiety and depression.

**Correlated Conditions**

The occurrence of comorbid diagnosis within the mental health domain is common. Consequently, it is not surprising, given the high prevalence rates of anxiety and depression, that they exhibit high comorbidity (see Appendix A) rates with Axis I, II and III disorders (Barlow, 2002; Dozois & Dobson, 2002; Hammen, 2001). Interestingly, the commonalities found within anxiety and depression are astounding to the point where it has been questioned whether these are actually two distinct disorders (Barlow, 2002).

Consistent within the literature in both community and clinical samples, the average comorbid rate of anxiety disorders with major depressive disorder is over 50% (Brown, Campbell, Lehman, Grishem, & Mancil, 2001). Dobson (1985) found that the correlation between anxiety and depression using self-report measures at the symptom level is higher than .61. Although anxiety and depression are often queried in the distinctness, two is not better than one. Correlated conditions of the anxiety and depression is also associated with increased severity of symptoms, psychological distress and overall impairment (Angst, 1997a).

Investigating the rates and developmental pathways of co-occurrence between symptoms of psychological distress in adolescents is important because comorbidity has implications for clinical and functional issues (Lewinsohn, Rohde, & Seely, 1995). More specifically, psychological comorbid disorders have been associated with greater severity of symptoms, more persistent difficulties, and an increased likelihood of seeking
treatment (McGee et al., 1990). Lewinsohn et al. (1995) suggest that psychiatric comorbidity in senior high school students was positively correlated to students’ academic problems, mental health treatment utilization, and history of suicide attempts.

The co-occurrence of anxiety and depression increases the difficulties faced by adolescents. Adolescents with correlated conditions may experience increased symptom severity and duration (Kendall et al., 2001). Furthermore, studies have linked comorbid anxiety and depression to increased suicidal tendency, increased risk for substance abuse, poorer response to psychotherapy, and more psychosocial problems (Birmaher, Williamson, Brent, & Kaufman, 1996). Stein and colleagues (2001) found that a comorbid diagnosis of social anxiety disorder in depressed adolescents was associated with a more malignant course and character of subsequent depressive illness. Moreover, relative to individuals who have one disorder, comorbid anxiety and depression are associated with increased suicidal ideation (Lecrubier, 1998b).

Recent evidence supports a temporal relationship between anxiety and depression in clinical, community and college settings (Brown, Campbell, et al., 2001; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Wetherell, Gatz, & Pederson, 2001). Anxiety disorders appear to precede the onset of depression by approximately two years. This temporal relationship indicates that anxiety disorders predispose adolescents to development of depression (Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998). Additionally mixed anxiety-depressive disorders predict more serious subsequent episodes (Angst, 1997b).

Dozois and Westra (2004) have speculated several reasons for the temporal relationship commonly found between anxiety and depression:
1. The relationship may reflect the effects of chronic anxiety on self-esteem
2. Anxious avoidance leading to a lack of reinforcements contributes to depression
3. The progression of closely related cognitive styles such as helplessness and hopelessness

Aside from the observation that anxiety is a risk factor for depression, there is no explanation of the specific reasons why correlations between anxiety and depression are so high. Perhaps these two disorders share similar etiologic origins including several overlapping mechanisms (Mineka, Pury, & Luten, 1995). Further exploration is needed to identify and understand the relationship between anxiety and depression to clarify etiologically concerns.

Given that the mechanisms underpinning the development of anxiety and depression, together with their high correlations, are not fully articulated, it remains crucial to further examine the identified hypothesis in sub-clinical populations.

Understanding the association between sub-clinical anxiety and depression in adolescents is important for several reasons. First, to confirm whether or not associations between anxiety and depression are reported in sub-clinical populations. Second, to provide information about sub-clinical populations that may help formulate and evaluate prevention strategies. Third, to identify the overlapping symptoms within the DSM IV so as to examine how measurement tools may be confounding.

The present study examines the co-occurrence of these symptoms of emotional distress. Correlated conditions of mental disorders in adolescents is common in both clinical and community samples (Brady & Kendall, 1992; Caron & Rutter, 1991;
Lewinsohn et al. 1993a). Moreover, correlated conditions is suggested to be even more common in adolescents than in adults (Rhode, Lewinsohn, & Seeley, 1991).

**Sex Differences**

Sex effects of anxiety and depressive symptoms in adolescence are important given that sex-specific pathways may guide preventative approaches (Cicchetti et al., 1998). Previous research has suggested that, beginning in adolescence, females show a marked increase in anxiety and depressive disorders, whereas males do not (Lewinsohn et al., 1993). Therefore, being female can be regarded as a high-risk factor for developing internalizing disorders during adolescence. Further research has reported that even by age six, girls are twice as likely to have experienced anxiety symptoms as compared to boys (Lewinsohn et al., 1997a). Females have been found to exhibit higher levels of depression (Peterson, Sarigiani, & Kennedy, 1991) and hopelessness (Mazza & Reynolds, 1998) consistently within the literature. Given that emotional problems often unfold during adolescence and continue into adulthood, it is not surprising that this trend continues where women are twice as likely as men to experience emotional difficulties (Nolan-Hoeksema, 1990).

Conversely, Andrews and Lewinsohn (1992) found a significant association between anxiety disorders and suicide attempts in males; a difference not found in females. More empirical support is needed to understand how anxiety mediates suicidal behaviour in young males.

Several explanations have been offered for this notable sex difference between anxiety and depressive disorders. Some researchers theorize that females may have a genetic or biological predisposition in developing internalizing disorders. Others propose
that sex differences in anxiety and depressive disorders are linked differences in the experiences and social roles that males and females endure.

Lewinsohn, Gotlib, Lewinsohn, Seeley, and Allen (1998) explored sex differences in anxiety disorders in a community sample of adolescents. After psychosocial variables were controlled for, sex differences continued to have a significant influence on both anxiety symptoms and an anxiety disorder diagnosis. These results suggest that females’ vulnerability to anxiety is linked to genetic factors rather than purely environmentally determined sex differences.

Conversely, other studies have found conflicting results suggesting that the sex ratio for anxiety disorders in children and adolescents to be approximately equal (Osman et al., 2002). Costello and Angold (1995) found that with the exception of OCD, which is found to be more common in females, there is little evidence to suggest sex differences for anxiety disorders in adolescents. Correspondingly, Masi, Mucci, Favilla, Romano, and Poli (1999) examined anxiety symptoms in children and adolescents and found no significant sex differences between symptomatic profiles of males and females.

Given inconsistent findings within the literature and the lack of research examining predictors of sex differences in adolescent anxiety and depression, it is of importance in preventative research to gain an understanding of mechanisms that interact to cause sex effects.

Unfortunately, there are a lack of empirical studies that have tested the complexity of this interaction (Dozois & Westra, 2004). Given that etiologic sex differences are poorly understood, the ability to identify and modify the developmental
process that occurs has vast implications in preventative research (Dozois & Westra, 2004).

It is hypothesized in the present study, and consistent with previous research, that adolescent females will have significantly higher levels of depressive symptoms and hopelessness, and that sex will be a strong predictor of depression via anxiety within the model being tested.

*Age Differences*

Studies have demonstrated that age differences in developing anxiety during adolescence coincide with pubertal changes (Malcarne & Hansdottir, 2001). Moreover, the onset of puberty may increase an adolescent’s risk for developing anxious symptomatology. Hayward et al. (1992) found an increase in panic attacks with sexual maturity in a sample of 754 sixth and seventh grade girls. Although males were not representative in the sample, it is hypothesized that structural and neuroendocrine changes in the brain during puberty may increase risk for developing anxiety related disorders (Bernstein & Borchardt, 1991). Unfortunately, the developmental paths of the nature of these changes and associated mechanisms (e.g. sex) have not been identified.

Age differences are diagnostically important because mood disorders may manifest in adolescence as irritation rather than the common perception of sadness (Dozois & Westra, 2004). More importantly, when a clinical criterion for diagnosis is not met, the consequences of sub-clinical depressive symptoms can be very devastating. Consistent within the literature, sub-clinical depression is correlated with significant functional impairment and is shown to be a risk factor for the development of Major
Depressive Disorder (MDD) (Brent, Birmaher, Kolko, Baugher, & Bridge, 2001; Horwath et al., 1992).

Geographic Location Differences

Few studies have examined the relationship between anxiety, depression and hopelessness in an adolescent population in both rural and urban populations (Burns et al., 2004). Studies that focused on rural populations have examined psychopathology in general and yielded inconsistent findings. Additionally, studies of rural adolescents have not been compared to corresponding urban studies (e.g., Puskar, Sereika & Haller, 2003). Studies investigating the relationship between anxiety and depression in urban and rural samples have failed to examine the effect of geographic location (e.g., Boyd & Gullone, 1997).

It is difficult to gather accurate prevalence rates of mental illness in rural populations for several reasons. First, rural families are less likely to access resources for emotional disturbances compared with urban families (Rost, Smith, & Taylor, 1993). In addition, rural families are more likely to attach stigma to mental health concerns compared to their urban counterparts (Rost, Smith, & Taylor, 1993). The latter may in part be a factor in rural populations seeking less treatment for emotional disturbances.

Further investigation in rural populations is required with regard to specific mental health concerns. This highlights the importance of examining location effects of anxiety, depression and hopelessness in urban and rural populations of adolescents.
Integrated Models of Anxiety and Depression

Diathesis Stress Model

The Diathesis-Stress model paradigm arose due to the failure of existing models to integrate biological, psychological and environmental factors within one model for explaining the complexity of abnormal behaviour (Davison, Neale, Blankstein, & Flett, 2005). The Diathesis-Stress model attempted to integrate genetic and environmental factors and focused on the interaction between individual predisposition to illness (referred to as the diathesis) and the environment (referred to as stress).

The Diathesis-Stress model posits that if an individual inherits a diathesis making them susceptible to developing a disorder, an environmental event or stressor is trigger for the development of the disorder (Barlow & Durand, 2005). Possessing a specific genetic vulnerability does not cause a disorder to develop. Rather, the larger the vulnerability, the less amount of stress is needed from the environment to trigger the disorder. The smaller the vulnerability to the disorder, the greater amount of stress needed to trigger its development (Barlow & Durand, 2005).

From a theoretical stance, the Diathesis-Stress model highlights two important points regarding psychopathology. First, this model assumes that both the diathesis and the stress are necessary factors in the development of a particular disorder. Second, the development of a clinical ailment is unlikely to occur from a single factor. Conversely, clinical disorders are complex and multifaceted, suggesting that a network of factors is more likely to lead to the development of a disorder (Davison et al., 2001).

Caspi et al. (1995) investigated the Diathesis-Stress model in a longitudinal study of 847 individuals over the period of approximately two decades. Beginning at age three,
the participants completed a battery of assessments. These focused on a particular gene demonstrated to have resilient effects on animals’ ability to cope with stress (Caspi et al., 1995). This particular gene comes in two versions: that with short alleles and that with long. Individuals who possess both copies of the long allele are speculated to cope with stress more efficiently.

Stressful life events were recorded for all participants in the study. Caspi et al. (1995) found that depression was related to stressful events in the recent past among individuals who carried both long alleles. Conversely, depression was related to stressful childhood experiences among individuals who carried both short alleles (Caspi et al., 1995).

Caspi et al. (1995) demonstrated that neither diathesis nor stress can account for the onset of depressive disorders. Rather, a network of interaction between multiple factors is more likely. The study provided support for the Diathesis-Stress model in that it demonstrated that multiple factors are involved in the development of depression.

The Diathesis-Stress model has been useful in identifying possible risk factors for a variety of disorders. The temporal aspect has revealed factors contributing to the onset of disorders. Primarily, this model has served as a building block for research linking identified risk factors to the relationship between multiple factors. In addition, the Diathesis-Stress model provides a general framework for the development of multiple disorders, yet is not specific to any particular one. This generality does not address sex and age effects of specific disorders. In addition, studies using the Diathesis-Stress model have focused on clinical populations. The present study proposes a model specific to
anxiety, depression and hopelessness that focuses on sub-clinical levels in addition to examining age and sex effects.

*Tripartite Model*

The tripartite model of emotion was developed by Watson and Clark (1991) in an attempt to explain the high comorbidity rates between anxiety and depressive disorders. The tripartite model proposes there is a common general factor that both anxiety and depression share: negative affect (NA). In addition, Watson and Clark (1991) identified two specific factors in which physiological hyperarousal (PH) is specific to anxiety and low positive affect (PA) specific to depression.

This model has gained empirical support on child and adult populations (Brown, Chorpita, & Barlow, 1998; Chorpita, Albano, & Barlow, 1998; Joiner, Catanzaro, & Laurent, 1996; Lonigan, Hooe, David, & Kistner, 1999; Watson et al., 1995). Within the populations mentioned above, the tripartite model has demonstrated that NA and PA are temporally stable inherited risk factors. In addition, the tripartite model focuses on the comorbidity of emotional disorders and adds a theoretical underpinning for the overlap in symptomology for children and adults. The tripartite theory is important in preventative research for anxiety and depression because it highlights the importance of understanding pathology along a continuum – including how symptom expression changes over the course of development (Chorpita, 2002).

Support for the tripartite model has been demonstrated in non-clinical child populations (Jacques & Mash, 2004). Unfortunately, generalization across age is uncertain due to the lack of empirical research on adolescent populations (Jacques & Mash, 2004). This limitation points to the importance of including age as a variable when
studying the relationship between anxiety and depression. In addition, studies need to focus on adolescent populations when examining the development of anxiety and depression. However, the effects of age and sex have not been fully explored in the tripartite model, despite differences noted in the literature (Jacques & Mash, 2004). The tripartite model provides a basis from which to explore the shared aspects of anxiety and depression symptoms (e.g., NA). As well, their specific symptoms (e.g., PH and PA, respectively) may help explain age and sex differences in anxiety and depression. In this study, the effects of age and sex are examined in a sample of adolescents.

*Triple Vulnerability Model*

Barlow (2000; 2002) developed the triple vulnerability model integrating biological, psychological and social factors that contribute to the development and maintenance of anxiety.

The generalized heritable vulnerability was the first diathesis described within the model. The triple vulnerability model assumes that some people might inherit anxious traits. However, the theory does not assume that a generalized genetic vulnerability to develop anxiety is not anxiety itself (Barlow & Durand, 2005). Rather, there are complex interactions between closely related traits, temperaments, negative affect, behavioural inhibition (to name just a few) that give way to variations beneath a heritable vulnerability to emotional disorders (Barlow, 2000, 2002). Researchers have demonstrated that negative affect is positively correlated to anxiety disorders (Brown, Chorpita, & Barlow, 1998; Clark, Watson, & Mineka, 1994; Zinbarg & Barlow, 1996). Research has focused on the development of anxiety and the relationship with negative affect. Gersahuny and Sher (1998) found that, for anxiety and depression, an interaction
between high negative affect and low extroversion were variables predicting the
development of anxiety and depression.

The second diathesis is the generalized psychological vulnerability. This
vulnerability is based on the relationship between anxiety and a sense of unpredictability
and uncontrollability (Barlow, 2000, 2002). More specifically, this theory suggests that
based on early experiences, when an individual develops a cognitive template that
incorporates uncontrollability and unpredictability, it later mediates the initial emergence
of anxiety and depression early in development (Chorpita & Barlow, 1998). When
cognitions are robust there is a generalized psychological vulnerability to anxiety.
Findings suggest that the cognitive vulnerability differentially affects female adolescents
compared to male adolescents, accounting, in part, for sex differences in the development
of anxiety or mood disorders (Barlow, 1988, 1991; Nolan-Hoeksema & Girdus, 1994).

Finally, the third vulnerability is the specific psychological vulnerability,
described as the factor in which individuals learn from early experience (Barlow, 2000,
2002). For example, if a child is taught when they are very young that dogs may be
dangerous (i.e., because the child’s mother is afraid of dogs), then this may specifically
cause the child to have an anxious reaction to dogs.

Barlow (2002) suggests that possessing one vulnerability does not imply that an
individual will develop an anxiety disorder. Similarly, if an individual encompasses all
three vulnerabilities it does not mean they will develop an anxiety disorder. Rather, this
theory suggests that if an individual possesses all three vulnerabilities, then the risk of
developing an anxiety disorder is greatly increased.
In addition, the triple vulnerability model is applicable to mood disorders where individuals who develop them also experience psychological vulnerability coping with difficult life experiences – leading to feelings of inadequacy (Barlow & Durand, 2005).

**Demoralization**

Demoralization is a phenomenological term coined by Jerome Frank (1974) in an attempt to capture the essence of an individual’s inability to cope and the relationship between feelings of helplessness, hopelessness, meaninglessness, incompetence and low self-esteem. Frank (1974) saw demoralization as a non-specific emotional distress that was not linked to any one disorder. In recent years, researchers have examined demoralization and demonstrated this concept is not simply a non-specific emotion. In a narrative review, Clarke and Kissane (2002) examined demoralization and related concepts, drawing on a range of empirically-based literature. One finding is that the phenomenon of demoralization may play an important role in its relationship to specific disorders (Clarke & Kissane, 2002).

Central to the concept of demoralization is an individual’s inability to cope with current stressors. More specifically, Frank (1974) described an individual’s experience of stress from both internal and external stimuli that in turn cause the subjective perception of meaninglessness of life.

The phenomenon of demoralization has been largely applied to clinical populations to explain a developmental spectrum of psychopathology. Frank (1974) observed both anxiety and depressive symptomatology as direct expressions of demoralization. Research demonstrates that if an individual endures internal or external stressors that are perceived as severe, then anxiety levels increase (Clarke & Kissane,
2002). When anxiety levels increase, an individual may feel the situation is uncontrollable, leading to helplessness. If the feeling of helplessness is not attended to, then hopelessness and the inability to cope will develop (Clarke & Kissane, 2002).

Clarke and Kissane (2002) demonstrated the prevalence of the demoralized individual within psychiatric and medical populations. In addition, clarification was provided to define the difference between the constructs of demoralization and depression. Although the two share symptoms, Clarke and Kissane (2002) state that demoralization is characterized by incompetence and depression by anhedonia (the inability to feel pleasure). Interestingly, as with depression, the narrative review reported hopelessness (including suicidal ideation) as the hallmark of demoralization.

The model of demoralization posits a temporal relationship between anxiety, depression and hopelessness where demoralization is the outcome of depression and hopelessness that has not been treated (Clarke & Kissane, 2002). Conversely, Rickelman (2002) suggest that demoralization may a precursor to anxiety, substance abuse, depression and suicide. Unfortunately, few studies have examined the construct of demoralization in sub-clinical populations. In addition, literature examining demoralization is sparse and definitive criteria unclear (Rickelman, 2002). This study proposes a model that utilizes the construct of demoralization as a common factor of sub-clinical anxiety, depression and hopelessness in an adolescent population. Additionally, because few researchers have examined sex or age effects in sub-clinical populations, age and sex issues will also be addressed.
**Structural Model of Anxiety and Depression**

A fundamental assumption of the triple vulnerability theory is that anxiety and related mood disorders develop as a result of complex interaction between three vulnerabilities or diatheses: generalized biological vulnerability, generalized psychological vulnerability and specific psychological vulnerability (Barlow, 1988, 2000, 2002). Models of depression have incorporated multiple factors and pathways to describe development and etiology. Demoralization (see Appendix A) is a term used to describe a common factor of the above process that presents over time. In terms of the present study model, demoralization is a defined latent variable common in anxiety, depression and hopelessness, measured in a normal population of adolescents. This study focuses on subclinical levels of anxiety, depression and hopelessness. The model represents important groundwork from which a prospective examination of vulnerabilities to the later development of clinical anxiety, depression and related disorders can be drawn.

Numerous studies have found that anxiety disorders typically precede depressive disorders (Lewinsohn et al., 1997; Alloy, Kelly, Mineka, & Clements, 1990; Rohde et al, 1991; Brelau, Schultz, & Peterson, 1995). This temporal pattern is a factor in the development of depression and holds true at clinical levels. The present model hypothesizes that this is also true at sub-clinical levels. Some theories (Alloy et al, 1990) have addressed the temporal pattern relating anxiety to depression (e.g., the integrated theories of anxiety and depression). Most theoretical frameworks, however, have not included the possibility that anxiety symptoms precede depression.

The proposed model is most closely related to Barlow’s triple vulnerability model (see Appendix A) of anxiety and related disorders and Frank’s model of demoralization
(see Appendix A) for several reasons. First, all three models are specifically concerned with anxiety and related mood disorders. Second, all three models include a temporal pattern in the relationship between anxiety and mood disorders. More specifically, this proposed model examines the temporal (age-related) pattern between anxiety, depression, hopelessness and demoralization.

This study will also extend the model from the triple vulnerability theory (see Appendix A) and model of demoralization (see Appendix A) by focusing on sub-clinical levels of anxiety, depression and hopelessness. Given the triple vulnerability model and model of demoralization are models that focus on clinical populations, it is important to extend existing models by testing sub-clinical populations.

According to the present model, hopelessness is considered a sub-component of depression. The latent variable, demoralization, is defined in this model by two measured variables: depression and hopelessness. The present model attempts to account for maximum amount of variance for the temporal relationship between anxiety, depression and hopelessness. The latent factor, which is not directly measured, is hypothesized to have a stable relationship in the development of the measured variables anxiety, depression and hopelessness.

The literature suggests that anxiety precedes depression in clinical populations (Barlow, 2000, 2002; Brown, Campbell, et al., 2001; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Dozois & Westra, 2004; Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998; Wetherell, Gatz, & Pederson, 2001). As mentioned earlier, adolescence is changing within Western societies where the age range is viewed within a broader lens (e.g., 14 to 24; Hine, 1999). It is hypothesized here that, in the present
model, there will be an increase in depression with age and a decrease in anxiety levels where grade nine and ten participants will have scores more highly correlated with anxiety and grade eleven and twelve participants will have scores more highly correlated with depression. In addition, it is expected that this effect will be greater in females as compared to males. Finally, given the gap in the literature exploring urban and rural differences, in this study, geographic location will be further examined. In sum, it is suspected that one model will account for the relationship between anxiety, depression, hopelessness and latent variable demoralization across age, sex and location.

*Structural Equation Modeling*

Structural equation modeling (SEM) is a multivariate statistical technique; an extension of the general linear model that multiple regression analysis is part of SEM belongs to a family of statistical analysis that includes path analysis and factor analysis. SEM functions on the basis of the process that integrates many techniques from numerous statistical analyses. First, SEM focuses on validating measurement models, typically using confirmatory factor analysis. Second, SEM attempts to fit structural models, typically through path analysis with latent variables (see Appendix A).

Structural equation modeling is usually used as a confirmatory approach as opposed to exploratory, though a common approach found in the literature combines exploratory and confirmatory analysis to strengthen model development. A limitation to the approach is that the confirmed model is post-hoc in nature. This indicates the confirmed model was built with the same data set used for the exploratory analysis, in turn having implications for model-fit of any new data.
To overcome the above dilemma, the present study uses a cross-validation strategy in which the model is developed with a small sub-sample of the data and confirmatory analysis is tested with the remainder and independent sample. This approach is utilized in the present study because repeatability with separate independent samples adds strength and statistical power to the results. In addition, this technique is a measure of validity and reliability allowing results to be more generalized to the population under study.

Model specificity begins with a firm theoretical basis of insight and judgement to guide model development.

*History of Structural Equation Modeling*

Multivariate analysis is recognized by researchers as an acceptable tool to examine the complexities of our society. Structural equation modeling was developed as a unified model by amalgamating methods from econometrics, psychometrics, sociometrics and multivariate statistics (Bentler, 1994).

Unlike other statistical techniques, structural equation modeling is a family of related procedures classified together under one name. Due to the diversity of techniques, more than one origin is likely. Structural equation modeling developed out of exploratory factor analysis in 1904 by Charles Spearman (Kline, 1998). Sewell Wright coined the term “path analysis” in 1921 and introduced this innovative technique to variety of disciplines (Kline, 1998). In later years, measurement and structural approaches were integrated to form what we know today as structural equation modeling.
Present Study

As mentioned, there is escalated concern within the mental health profession regarding the lack of research on adolescent psychopathology, given adolescents are the ones who suffer most from psychological problems and are those least likely to seek treatment. Moreover, initial onset of mood disorders consistently occurs in childhood and adolescence and is thought to be one of the strongest predictors of mental health problems in adulthood (van Os & Jones, 1999). As with many other debilitating disorders, the literature demonstrates that early onset is much more devastating than later onset (Hoehn-Saric, Hazlett, & Mcleod, 1993). Individuals with early onset of anxiety and depression are at much higher risk for correlated conditions, marital instability, academic difficulties, teenage childbearing, early marriage and low socio-economic status (Kessler, Olfson & Berglund, 1998). This data is of paramount importance when developing preventative intervention programs to alleviate emotional difficulties within the adolescent population before clinical levels are reached. Early detection of sub-clinical symptoms becomes crucial in preventative research.

Given that anxiety is shown within the literature to precede depression, it is speculated here that more attention needs to be placed on the mechanisms enhancing anxiety in adolescence and the dynamic interplay present. Restricting the progression of mood disorders in adolescence must encompass the identification of how risk factors for anxiety play a role in the structural path to depression (Dozois & Westra, 2004).

Comorbidity (see Appendix A) of depression with a number of mental illnesses is not only common, but also associated with a more severe and chronic course (Dozois & Westra, 2004). Specifically, anxiety and depression share many early risk factors that
contribute to the manifestation of the course of the illness. A large base of evidence is beginning to query how these shared factors interact to manifest a specific mood disorder. This is essential to preventative interventions at multiple levels along the structural paths that specific mood disorders take.

This study will attempt to extend knowledge of anxiety/depression disorder correlated conditions by investigating rates and patterns of concurrent anxious, depressive and hopeless symptoms in high school students. It is speculated that the relationship between anxious and depressive symptomatology will be consistent with the relationship between clinical levels of anxiety and depression. As previously discussed, in regards to the relationship between depression and hopelessness, there is an abundance of research examining the correlation between feelings of depression, suicide and hopelessness (e.g., Cochrane-Brink et al., 2000). Therefore, it is hypothesized that a similar strong relationship between symptoms of depression and hopelessness will exist. Little research has investigated the relationship between hopelessness and symptoms of anxiety, particularly within a community adolescent sample.

Theoretically, both anxiety and hopelessness stem from perceptions of negativity surrounding the future in combination with a lack of control surrounding such events (Barlow, 2000; 2002). It is theorized that a significant relationship exists between anxious symptoms and hopelessness. Likewise, because a strong association is hypothesized to exist among symptoms of depression and both anxiety (Barlow, 2000; 2002) and hopelessness (Beck et al., 1974), the present study will investigate the possible relationship between hopelessness and anxiety and how these variables interact within a structural model related to one latent factor demoralization.
Furthermore, there is insufficient research discussing the prevalence of hopeless feelings in non-clinical samples. In addition, no research was found on the occurrence of anxious, depressive and hopeless symptomology in community adolescents. Existing prevalence estimates give a broader picture of symptom occurrence in high school students and has allowed for the identification of individuals at risk for developing clinical-level internalizing disorders. The approach of the present study is to conceptualize the natural occurrence of the relationship between anxiety, depression and hopelessness represented as a model (Figure 1) containing one latent factor, termed for the purposes of this study, demoralization (see Appendix A), in a non-clinical community sample of adolescents.

The majority of epidemiological research has focused on adolescents living in urban centers (Burns et al., 2004; Puskar et al., 2003; Rost et al., 1993). The present study examines the proposed model not only an urban city center located in Alberta, Canada, but also in samples representing rural community populations in Alberta and Saskatchewan, Canada. It is important to note that when discussing geographic location in the present study, the sample specifically represents a Western Canadian sample of urban and rural locales that includes a restricted age range of grades 10-12.

Empirical research has consistently demonstrated females are more susceptible to mood disorders than males. Many hypotheses speculate as to why this sex discrepancy is present (Cicchetti et al., 1998, Lewinsohn et al., 1993, Lewinsohn et al., 1997a, Peterson, Sarigiani, & Kennedy, 1991, Mazza & Reynolds, 1998, Nolan-Hoeckema, 1990), although the specific cause has remained largely unknown. The present study considers the effect of sex on the model.
This study is an extension of unpublished research at the University of Calgary conducted by Slatts and Alladin (2002), Nosen and Alladin (2003), Chong and Alladin (2004), and West and Alladin (2005) and ultimately aims to develop and test a parsimonious model that describes the relationship between sub-clinical anxiety, depression and hopelessness. Another primary aim of this study is to examine the model in terms of a sample that represents a range of demographic variables that are important to the relationship between anxiety, depression and hopelessness in a non-clinical community sample of adolescents.

In this study a specific method of analysis is used to develop and test the model. As previously mentioned, much of the existing literature has focused on identifying risk factors rather than examining developmental pathways. It is, therefore, necessary to go beyond the identification of risk factors to study the developmental pathways leading to emotional disorders in adolescents. Using structural equation modeling (see Appendix A), the current study incorporates data from the point-prevalence studies mentioned above, to examine one latent factor as a precursor to anxiety and depression within a preventative context. On the basis of previous research, a model will be constructed to describe the relationship among a set of measured variables using confirmatory factor analysis (Barlow, 2002; Cicchetti et al., 1998).

A cross-validation method (see Appendix A) using exploratory and confirmatory factor analysis will be used to develop and test the model (Figure 1) relating anxiety, depression, hopelessness and demoralization across age, sex and location. Another primary goal of the study is to examine the hypothesized relationship between anxiety,
depression, hopelessness and demoralization in one model across the strata of age, sex and location.
Figure 1. Proposed latent variable model relating anxiety, depression and hopelessness.
The specific research questions (RQ) related to this model (Figure 1) are as follows:

RQ1: Does the proposed model account for the relationship between anxiety, depression, hopelessness and the postulated latent variable demoralization across age, sex and geographic location?

RQ2: Will the proposed model remain stable across sub-clinical levels of anxiety, depression and hopelessness?
Chapter 3: Methods

Participants

The combined sample consists of data collected from four independent studies from 2001 to 2005. All studies used identical sampling methods, procedure and measures. Nine hundred and seventy one (n=971) participants in each of the previous four studies were high school students (grades 10-12) from three geographic locations: Calgary, Saskatchewan and Lethbridge. Participants were distinguished between urban and rural locations in Western Canada.

Nine hundred and seventy one participants comprised the sample. Males and females were equally represented in samples developed for the exploratory and confirmatory analyses. Grade is utilized in the present study as a proxy to age including grades 10 (n=301), 11 (n=296) and 12 (n=374) representing ages ranging from 14, 16 and 18 years of age. Four hundred and sixty three participants (47.68%) came from rural dwellings, with the remainder coming from urban dwellings. All participants attended school in Canada. The demographic data for the total sample are presented in Table 1.

Procedure

Parent and student consent forms were collected from all participants prior to conducting the study and only students who handed in both parent and student consent forms were able to participate. In each past study, on the testing day, informed consent from participants was obtained once again. Each participant was reminded that their participation was completely voluntary, and that their responses would remain anonymous and reported only in aggregate form.
Participants completed a battery of self-report questionnaires including the Beck Anxiety Inventory (BAI), Beck Depression Inventory-Revised (BDI-II), Beck Hopelessness Scale (BHS), and demographic survey during one of their regular school classes (see appendices for complete set of study materials given to participants). After completing the questionnaires, participants were debriefed (see appendices) and were thanked for their participation. The time required to complete the test battery was approximately 45 minutes for each participant in the original studies.

*Materials*

The test materials consisted of three self-rating questionnaires that have been used with adolescent community samples in other studies: the BAI (Beck & Steer, 1993a), the BDI-II (Beck, Steer, & Brown, 1996), and the BHS (Beck & Steer, 1993b). These questionnaires are used as screening instruments and are not used independently to diagnose anxiety or depression. The three scales were thought to be suitable for the present study because they are continuous measures that allow for the quantitative assessment of emotional difficulties. Furthermore, the BAI, BDI-II, and BHS provide four categories of severity scores labelled as “minimal”, “mild”, “moderate”, or “severe”, and these ratings are useful for the estimation of symptom severity and risk in the development of the present model. In addition, given that these instruments are used as screening tools and not for diagnostic purposes, they were appropriate in measuring sub-clinical levels of anxiety, depression and hopelessness. Mild to moderate ranges were used as criteria to identify sub-clinical levels within the current sample population.

Several statistics provide information about the reliability and validity of the measures employed in this study. The field studies of interrater reliability and test-retest
reliability used the Pearson correlation (r) or the intraclass correlation coefficient (Shrout & Fleiss, 1979) as the indices of agreement for continuous variables, and Cronbach’s alpha (α: Cronbach, 1951) to examine the internal consistency of items making up the various scales.

*Beck Anxiety Inventory*

The BAI was used to measure the severity of anxiety symptoms. It is a 21-item questionnaire that takes 5-10 minutes to complete. Each of the 21 items (anxiety symptoms) is represented by four statements reflecting increasing levels of anxiety. Using a 4-point scale ranging from 0 (not at all) to 3 (severely; I could barely stand it), participants rate the severity of each of the symptoms by indicating how much they have been bothered by the symptoms during the preceding week, including the test day. Severity scores for each question are summed, deriving a score ranging from 0-63. A minimal overall severity rating ranges from 0 to 7, mild from 8 to 15, moderate from 16 to 25, and severe from 26 to 63. Anxiety reaches a “clinical” level at a score of 16 or greater.

The BAI has been found to be a reliable and valid measure. Test-retest reliability ranges from .62 (1 week) (Beck, Steer, & Foran, 1985 as cited in Waller, 1993) to .75 (7 weeks) (Creamer, Foran, & Bell, 1995). In addition, Creamer et al. (1995) found moderate concurrent validity with the State Trait Anxiety Inventory (state .64, trait .68), and determined that the BAI adequately discriminates between anxiety and depression. The BAI was originally designed for the use of adult populations, however has been found to be valid in adolescent populations. Jolly, Aruffo, Wherry, and Livingston (1993) administered the BAI to inpatient adolescents (aged 12 to 19) diagnosed with anxiety
according to the DSM-III-R criteria and found high internal consistency (.94). Furthermore, the concurrent validity was moderate to high, where the BAI significantly correlated with clinician ratings (r=.40) and self-report anxiety measures (r=.58).

*Beck Depression Inventory-Revised*

Depressive symptoms were measured using the BDI-II, which takes 10 to 15 minutes to complete and consists of 21 items. The severity scale ranges from 0 (low depressive indicator) to 3 (high depressive indicator) for each item. For example, on the “sadness” item, participants can choose a rating of 0 (“I do not feel sad”), 1 (“I feel sad much of the time”), 2 (I am sad all the time”), or 3 (“I am so sad or unhappy that I can’t stand it”).

Ratings are made based on symptoms experienced “during the past 2 weeks, including today”. Similar to the BAI, the sums of these scores can range from 0 to 63, with severity groupings as follows: minimal (0-13), mild (14-19), moderate (20-28), severe (29-63). The cut off for clinical depression is a score of 20. This, therefore, includes participants falling into the moderate and severe groups.

The BDI-II is strong in terms of psychometric properties. In one study, reliability was estimated at .92 with outpatients and .93 for a non-clinical sample (Santer, Ramsey, & Zuroff, 1994 as cited in Arbisi, 1996). Test-retest reliability was also excellent at .93. The BDI demonstrates good concurrent validity and has correlations with the Hamilton Psychiatric Rating Scale for Depression-Revised at .71 in outpatients, and discriminate validity had good correlation with the Hamilton Rating Scale for Anxiety-Revised at .47. Furthermore, individuals with mood disorders scored higher on the BDI than those who were diagnosed with anxiety or adjustment disorder.
Beck Hopelessness Scale

The BHS was used to assess hopelessness, or the extent of negative expectancies about the future. It is a 20-item inventory that consists of true-or-false statements, and it takes 5 to 10 minutes to complete. Participants indicate whether a statement describes their attitude for the preceding week, including the test day. The BHS is scored by summing the keyed responses of hopelessness for each of the 20 statements; scores can range from 0 to 20. There are four severity groupings for this inventory: minimal (0-3); mild (4-8); moderate (9-14); and severe (15-20). The cut-off score related to clinical hopelessness is 9 (moderate and severe groups).

The BHS is the best known and most widely used scale for evaluation of hopelessness in adolescents (Orbach & Bar-Joseph, 1993). It has shown a high internal consistency (r=.93), and concurrent validity with clinicians ratings of hopelessness of r=.74 (Mazza & Reynolds, 1998). High scores (over 9) on the BHS generally predict about 90% of suicide completers, while about 6% of such scores turn out to be false positives (Beck et al., 1985). Low scores on the BHS merely suggest a lack of hopelessness rather than an abundance of hope (Beck et al., 1985).

Statistical Analysis

The present study utilized quantitative techniques that included descriptive statistics, such as means and standard deviations. A one-way analysis of variance (ANOVA) comparing means was implemented to examine mean differences between independent variables to facilitate post hoc analysis examining differences between the variables.
In developing the proposed multivariable model, a description of each study variable was necessary for theoretical formation and conceptualization. Means with 90% confidence intervals and frequencies were used in generating descriptive statistics for continuous and categorical variables respectively.

**Structural Equation Modeling**

Structural equation modeling (SEM) (see Appendix A) was selected as the statistical technique for data analysis of this study because it is effective in reducing data and testing models in behavioural and social sciences (Hox & Bechger, 1998). Structural equation modeling provides researchers with a general and convenient foundation for data analysis that incorporates several components derived from the result of multivariate analyses (Hox & Bechger, 1998). The combination of factor analysis (exploratory and confirmatory), correlation matrices, and graphical path diagrams to depict the model are used and tested in this study. The analytic strength behind structural equation modeling resides in the theoretical underpinnings that explain the relationship between the observed or measured variables and the latent variables (factors) (see Appendix A). The following section describes the SEM method used to analyse data in the present study, introduce the estimation technique (Maximum-likelihood) and the fit index used to estimate the utility of the model.

**Factor Analysis**

Factor analysis (FA) involves the study of order and structure in multivariate data. FA including theory and underlying constructs important in structuring observed data. One important assumption that factor analysis highlights is that covariances between observed variables can be described by a reduced set of latent variables. Exploratory
factor analysis explores the relationship between the latent factors and observed variables with no specific hypothesis present regarding this relationship (Hox & Bechger, 1998). Consequently, the model is arbitrary with the emphasis placed on estimating the parameters of the model. Conversely, confirmatory factor analysis represents a clear hypothesis of factor structure within the model, which aims to confirm the model’s goodness of fit to the data (Hox & Bechger, 1998).

In sum, using exploratory and confirmatory analysis, structural equation modeling serves two purposes in the present study. First, using exploratory analysis, the parameters of the model including factor loading, variances and correlations of the latent variable and residual error of the observed variables were estimated. Second, using confirmatory analysis, the hypothesis that one model consisting of the latent variable will describe the relationship between the observed or measured variables across several conditions.

It is important to estimate parameters of the proposed model because factor loadings are specified to be equal for each class of the grouping variable. Setting the factor loading equally across each indicator variable determines if the indicators are valid across groups. In the proposed model factor loadings is used to induce the meaning of the latent variable (demoralization). This approach is taken because if the loadings differ substantially across groups or across time, then the induced meanings of the factor will differ substantially and not necessarily be grounded by the theory intended when building the model. Each statistical property utilized in the present study will be further explained in the results section.
Estimation and Model Fit

Maximum Likelihood Estimation (ML). Maximum likelihood estimation is a statistical means used to estimate all model parameters simultaneously (Kline, 1998). ML estimation is the most commonly used algorithm in model fitting programs (Kline, 1998). Similar to multiple regression, ML yields path co-efficients and variances of the sample population. ML assumes that the estimated parameter values that maximize the likelihood (probability) that the observed covariances were drawn from this population (Kline, 1998). In SEM, a typical assumption is that the sample data follows a multivariate normal distribution such that means and covariances contain the same information and represent the whole sample. ML follows the normal distribution assumption at reasonably large sample sizes (>200). The basic statistical model often used is: DATA=MODEL + ERROR, where complex algorithms maximize the fit of the model. Maximum Likelihood estimation is appropriate when data meets the multivariate normal distribution; is continuous data; and, is a reasonable sample size (Hox & Bechger, 1998).

Bentler-Bonet Fit Index. As with estimation techniques, in order to have statistical power in model fitness to the data set, the sample size must be large. Three fit indices associated with SEM software (EQS) used for data analysis in the present study are as follows: the Bentler-Bonett Normed Fit Index (NFI), the Bentler Comparative Fit Index (CFI), and the Bentler-Bonett Non-Normed Fit Index (NNFI; Bentler, 1990; Bentler & Bonett, 1980).

The NFI specifies the models overall fit compared with the null model. This value is between zero and one, the closer the NFI value is to one, and the higher is the overall fitness of the model. For instance, if a NFI value was .80, the overall goodness of fit to
the data of the model being tested would be estimated at 80% than that of the null model estimation (Kline, 1998). In other words, the closer the fit value is to one, the better the goodness of fit of the model to the data.

Bentler (1990) developed a similar fit index called the Comparative fit index (CFI). The CFI is similarly scaled to the NFI, but is less sensitive to sample size (Kline, 1998). The Non-Normed fit Index was developed for testing complex models (includes a correction for complexity) and allows values to fall out of the zero to one range. This is advantageous because it is possible for fit values to be much lower than with other fit indexes so those smaller samples may be used. Due to the large sample size of the present study and the simplicity of the model being tested, it was most appropriate to use the Bentler-Bonett Normed Fit Index and Comparative Fit Index for the goodness of fit value.

The first set of analyses conducted were preliminary factor analysis to reduce data in explaining relationships among and between variables. Factor analyses were the first step in exploring how many factors account for the variance and explain the relation among the measured variables. The results of factor analysis were utilized in identifying which variables would be included in the structural models for further analyses based on factor loading on one latent factor. The following exploratory factor analyses were run on three independent sub-samples: phase I, phase II, and phase III. Demographic variables were considered in this model development phase.

One hypothesized model is then built on the basis of previous literature and results of factor analysis. This model is further tested using structural equation modeling.
Structural equation analysis was conducted using parallel procedure to exploratory factor analysis where three independent sub-samples were conducted.

Maximum-likelihood estimation was utilized using covariance matrices extracted from EQS outputs (Bentler, 1993). The model was analyzed based on its goodness of fit, residual error and chi-square values within each sub-sample. Goodness of fit is measured on the basis of the Bentler-Bonet normed fit index, Comparative Fit Index and the standardized residual error estimate (Bentler & Bonet, 1980). Fit index values range from zero to one with values greater than .90, standardized residual error equal to or less than 0.05 and a non-significant chi-square, being acceptable fit to the data (Bentler & Bonet, 1980). Confirmatory analysis was then conducted on the remainder of the sample (excluding phase I and II sub-sample) using the same coefficient values as the exploratory analysis. Exploratory and confirmatory standardized solutions were compared to each other to examine model convergence. Differences less than .02 were considered statistically significant.

The overall goal of this analysis was to examine the relationship between the continuous outcome dependent variable (demoralization), the measured independent variables (anxiety, depression and hopelessness) across three dichotomous strata (sex, age, location).

*Ethical Considerations*

In utilizing the data that had been gathered in the four previous studies, it is noteworthy that all participants who volunteered did so based on informed consent prior to, and on, testing day. The previous studies involved neither invasive procedures nor purposes hidden from the participants. Participants in each study were debriefed upon
completion of the test battery. Distress associated with the completion of the questionnaires was not anticipated or observed by the researchers involved. To ensure confidentiality, participants’ names did not appear on the data records, rather, each data record was assigned a consecutive number and, therefore, responses remained anonymous. All four studies received ethical approval from the University of Calgary research ethics board.
Chapter 4: Results

Demographic Characteristics

The sample was comprised of 971 participants, of whom 51.29% were female. Males and females were equally represented in samples developed for the exploratory and confirmatory analyses. Grade is utilized in the present study as a proxy to age including grades 10 (n=301), 11 (n=296) and 12 (n=374) representing ages ranging from 14, 16 and 18 years of age. Four hundred and sixty three participants (47.68%) came from rural dwellings, with the remainder coming from urban dwellings. All participants attended school in Canada. The demographic data for the total sample are presented in Table 1.

Table 1. Demographic Data for Measured Variables of Entire Sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>971</td>
<td>12.80</td>
<td>10.25</td>
<td>0</td>
<td>63</td>
</tr>
<tr>
<td>BDI</td>
<td>971</td>
<td>13.29</td>
<td>10.33</td>
<td>0</td>
<td>63</td>
</tr>
<tr>
<td>BHS</td>
<td>971</td>
<td>4.91</td>
<td>4.27</td>
<td>0</td>
<td>20</td>
</tr>
</tbody>
</table>

It was important to consider age, sex and grade as possible confounds in the relationship between the measured variables and the latent factor. This was examined in the developmental factor analysis. In developing the initial model, the categorical variables were considered in model developmental factor analysis to examine if they contributed to the model in preparation for the subsequent exploratory and confirmatory analyses.

From the total sample of 971 participants, mean scores on the three self-report symptom questionnaires (BAI, BDI-II, and BHS) were as follows: BAI, $M = 12.80$, $SD =$
10.25; BDI-II, M = 13.29, SD = 10.33; BHS, M = 4.91, SD = 4.27. The mean scores met criterion for sub-clinical levels of anxiety, depression and hopelessness because scores were all within the minimal-moderate range and did not exceed clinical cutoffs.

All analyses were conducted at an alpha level of .05. Pearson correlations were conducted between scores on the BAI, BDI-II, and BHS. Correlations below .35 are considered to be low strength, those between .35 and .65 are considered moderate strength and those above .65 are considered high strength. The correlations found among the measured variables for the entire sample are presented in Table 2. Scores on the BAI positively correlated with scores on the BDI-II (r(971)= .66, p<.001) and BHS (r(971)= .42, p<.001). Moreover, scores on the BDI-II were positively correlated with those on the BHS (r(971)= .58, p< .001).

Table 2. Correlation Matrix of Measured Variables

<table>
<thead>
<tr>
<th></th>
<th>BAI</th>
<th>BDI</th>
<th>BHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>0.66</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>BHS</td>
<td>0.42</td>
<td>0.58</td>
<td>1</td>
</tr>
</tbody>
</table>

*Factor Analysis*

The main purpose of factor analysis is to reduce data in explaining relationships among and between variables. Factor analyses were the first step in exploring how many factors account for the variance and explain the relation among the measured variables. The results were utilized in identifying which variables would be included in the structural models for further analyses based on factor loading on one latent factor. The
following exploratory factor analyses were run on three independent sub-samples: phase I, phase II and phase III.

*Phase I Exploratory Factor Analysis*

From the phase I sample (n=314), mean scores on the three self-report symptom questionnaires (BAI, BDI-II, and BHS) were as follows: BAI, $M = 12.85$, $SD = 10.65$; BDI-II, $M = 13.11$, $SD = 10.19$; BHS, $M = 4.73$, $SD = 4.08$.

The first phase I exploratory factor analysis, used to identify latent structure, included the categorical demographic variables to assess whether or not they were to be included within the model. Analysis of sex, location and grade as main effects or interaction terms revealed that these variables were not associated with the one latent factor that emerged from this analysis. The first independent sample (n=314), used in the phase I of exploratory factor analysis, revealed one latent factor (eigenvalue=1.54) related to the three measured variables. As a result of this finding, the categorical demographic variables were not included in subsequent analyses.

Reanalyzing the first sample without the demographic variables once again revealed one latent factor with a similar eigenvalue and loadings for the three measured variables as described below.

The correlations among the measured variables are presented in Table 3. Scores on the BAI positively correlated with scores on the BDI-II ($r(314) = .67$, $p<.001$) and BHS ($r(314) = .40$, $p<.001$). Moreover, scores on the BDI-II were positively correlated with those on the BHS ($r(314) = .55$, $p<.001$).
Table 3. Correlation Matrix Phase I Exploratory Factor Analysis

<table>
<thead>
<tr>
<th></th>
<th>BAI</th>
<th>BDI</th>
<th>BHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>0.67</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>BHS</td>
<td>0.40</td>
<td>0.55</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Having an eigenvalue greater than 1 was the specific criterion used for choosing the appropriate indicator(s) for latent factors. Only one emerged, which accounted most of the total variance. A coefficient level of 0.30 or greater was used as criteria for including a measured variable as significant in relationship to its factor loading (Karimova & Martin, 2003). This level is consistent with previous studies that have used exploratory factor analysis (e.g., Karimova & Martin, 2003). In the phase I factor analysis (n=314), the eigenvalue for the latent factor met the study criteria at 1.54 with factor loadings for the BAI, BDI and BHS of 0.718, 0.814, and 0.602, respectively.

*Phase II Exploratory Factor Analysis*

From the exploratory sample (n=338), mean scores on the three self-report symptom questionnaires (BAI, BDI-II, and BHS) were as follows: BAI, M = 12.99, SD = 10.42; BDI-II, M = 13.87, SD = 10.53; BHS, M = 5.07, SD = 4.41.

The correlations among the measured variables are presented in Table 4. Scores on the BAI positively correlated with scores on the BDI-II (r(338)= .65, p<.001) and BHS (r(338)= .42, p<.001). Moreover, scores on the BDI-II were positively correlated with those on the BHS (r(338)= .56, p< .001).
Table 4. Correlation Matrix Phase II Exploratory Factor Analysis

<table>
<thead>
<tr>
<th></th>
<th>BAI</th>
<th>BDI</th>
<th>BHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 338</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BAI</td>
<td></td>
<td>0.65</td>
<td>1.00</td>
</tr>
<tr>
<td>BDI</td>
<td>0.42</td>
<td></td>
<td>1.00</td>
</tr>
<tr>
<td>BHS</td>
<td>0.56</td>
<td>0.64</td>
<td></td>
</tr>
</tbody>
</table>

In phase II of exploratory factor analysis (n=338), the eigenvalue for the latent factor met the study criteria at 1.52 with factor loadings for the BAI, BDI and BHS of 0.61, 0.81, and 0.61, respectively.

*Phase III Exploratory Factor Analysis*

From the phase II exploratory sample (n=319), mean scores on the three self-report symptom questionnaires (BAI, BDI-II, and BHS) were as follows: BAI, M = 12.54, SD = 9.65; BDI-II, M = 12.84, SD = 10.26; BHS, M = 4.91, SD = 4.32.

The correlations among the measured variables are presented in Table 5. Scores on the BAI positively correlated with scores on the BDI-II (r(319) = .68, p<.001) and BHS (r(319) = .46, p<.001). Moreover, scores on the BDI-II were positively correlated with those on the BHS (r(319) = .64, p<.001).

Table 5. Correlation Matrix Phase III Exploratory Factor Analysis

<table>
<thead>
<tr>
<th></th>
<th>BAI</th>
<th>BDI</th>
<th>BHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 319</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BAI</td>
<td></td>
<td>0.68</td>
<td>1.00</td>
</tr>
<tr>
<td>BDI</td>
<td>0.46</td>
<td></td>
<td>1.00</td>
</tr>
<tr>
<td>BHS</td>
<td>0.64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In phase III of the exploratory factor analysis (n=319), the eigenvalue for the latent factor met the study criteria at 1.70 with factor loadings for the BAI, BDI and BHS
of 0.68, 0.84, and 0.68, respectively. Factor loadings and eigenvalues for all three factor analyses are summarized in Table 6.

Table 6. Factor Loadings of all 3 Variables and Eigenvalue of the Latent Factor

<table>
<thead>
<tr>
<th></th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>0.72</td>
<td>0.61</td>
<td>0.68</td>
</tr>
<tr>
<td>BDI</td>
<td>0.81</td>
<td>0.81</td>
<td>0.84</td>
</tr>
<tr>
<td>BHS</td>
<td>0.60</td>
<td>0.61</td>
<td>0.68</td>
</tr>
<tr>
<td>Factor Eigenvalue</td>
<td>1.53</td>
<td>1.52</td>
<td>1.70</td>
</tr>
</tbody>
</table>

Summary of Exploratory Factor Analyses

From the results above, particularly, the correlations, sample sizes, and standard deviations from each independent sample were employed to construct three parallel structural equation models. Each independent sample and set of results was used to develop, explore and confirm the main hypothesis of this study that a final confirmatory model consisting of one latent variable explained the relationship between measured variables representing depression, anxiety and hopelessness.

Analysis of the Structural Model

Structural equation modeling (SEM) was used to estimate the posited relationship between the indicator variables (BAI, BDI, BHS) and the latent variable that emerged from the exploratory factor analysis. Demographic variables were left out of the final model as including them did not improve the fit of the model. More specifically, the demographic variables did not meet criteria to significantly load on the latent factor and subsequently did not increase the fit index value of the model. The following criteria were used to build the model. First, the model can be theoretically explained. Second, the
model is simple in structure. Finally, the model fits the data that is evaluated using comparative goodness of fit index (CFI) of 0.9 or greater (not exceeding 1.0) and standardized residual error equal to or less than 0.05 and a non-significant chi-square, indicating a good fit. In addition, standardized solution values were compared between exploratory and confirmatory analysis having a difference of less than 0.2.

Correlation matrices, rather than covariance matrices, were chosen for two reasons as the values used to interpret findings resulting from employing the factor analysis results in the EQS algorithm. First, the objective of this study is to understand the interrelationship between constructs and not to explain the total variance of each construct. Second, comparisons made across different variables are only appropriate with correlations because the scale of the measure affects covariances (Hair, Anderson, Tatham, & Black, 1992).

Model I: Confirmatory Factor Analysis Structural Equation Model

For this first set of analyses, the coefficients and correlated errors between anxiety, depression, and hopelessness were freed in both analyses including and excluding demographic variables, meaning that these were not numerically defined in advance as they were in subsequent models.

For Model I (see Table 7), the coefficients between anxiety, depression, hopelessness and latent variable ran freely for the analysis. The chi-square value, $X^2$ (2) = 27.14, p < .001, and the goodness of fit indices (CFI=0.92, NFI=0.91) and residual error at 0.11.

Table 7. Model I Standardized Solution

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>0.76</td>
<td>0.65</td>
</tr>
<tr>
<td>BDI</td>
<td>0.81</td>
<td>0.58</td>
</tr>
<tr>
<td>BHS</td>
<td>0.70</td>
<td>0.72</td>
</tr>
</tbody>
</table>
Model II: Confirmatory Factor Analysis Structural Equation Model

Using exploratory factor analysis, the examination of the relations between a set of measures can be explained by a common factors, without specifying a particular factor structure. This technique was chosen to examine whether the factor model would provide acceptable fit to the chosen sample. If the proposed model of measured and latent variables could not fit the data sample, then there is no point in testing more constrained CFA in testing the proposed model.

In building the equations to run in EQS for model II, the coefficients were fixed to using the standardized solution values simulated from Model I analysis in building and modifying the model.

Model II, as shown in Table 8, produced a non-significant chi-square value, $X^2 (2) = 9.32, p < .001$, with goodness of fit indices (CFI=0.976, NFI=0.970) and lowered standardized error (0.057) indicating that the aforementioned modifications significantly decreased the chi-square value, suggesting that the model’s fit was improving. The correlation coefficients then from Model II will be used as fixed values in the Model III analysis. The factor and error coefficients for the BAI, BDI and BHS were used in constructing the final confirmatory model in order to assess the residual error and goodness of fit.

<table>
<thead>
<tr>
<th>Table 8. Model II Standardized Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 338</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>BAI</td>
</tr>
<tr>
<td>BDI</td>
</tr>
<tr>
<td>BHS</td>
</tr>
</tbody>
</table>
Model III: Confirmatory Factor Analysis Structural Equation Model

Confirmatory analysis was used to examine Model III (shown in Table 9). This was the final model being tested as a result to modifications resulting from the previous analyses. The coefficients were fixed to using the standardized solution values simulated from Model II analysis in building and modifying the model.

The final model produced a non-significant chi-square value, $X^2 (2) = 7.24, p<.001$, with goodness of fit indices (CFI=0.99, NFI=0.98) and lowered standardized error (0.05). This indicates that modifications that were made significantly decreased the chi-square value and suggests that the final model was the best fit to the data.

Table 9. Model III Standardized Solution

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAI</td>
<td>0.76</td>
<td>0.65</td>
</tr>
<tr>
<td>BDI</td>
<td>0.91</td>
<td>0.42</td>
</tr>
<tr>
<td>BHS</td>
<td>0.70</td>
<td>0.71</td>
</tr>
</tbody>
</table>

Comparison of the exploratory and confirmatory findings indicated convergence, having a difference of less than 0.2. There was very little difference between the standardized solution and fit indices. All difference values were below 0.2 indicating that the model is a good fit to both independent samples demonstrating statistical power and reliability.
Figure 2. Confirmatory structural equation model.
Summary

The preliminary analysis found that the demographic variables did not contribute to the factor emerging from the exploratory factor analysis (see Appendix A), hence, were not further considered. The three standardized solutions emerging from the structural equation models, based on confirmatory factor analysis (see Appendix A), revealed one latent variable to explain the relationship between the three measured variables (BAI, BDI and BHS), representing respectively, anxiety, depression and hopelessness. For the purposes of subsequent discussion, the latent variable will be labelled as “demoralization” (see Appendix A) (Frank, 1974; Clarke & Kissane, 2002).
Chapter 5: Discussion

The nature of the relationship between anxiety, depression and hopelessness has been a central focus in the literature of the recent decades (Barlow & Durand, 2005; Price & Ingram, 2001). Previous models of clinical anxiety and depression (Barlow, 2000, 2002; Watson & Clark, 1991) have identified numerous risk factors and researchers have debated whether each construct is unique in structure or is an overlap of common etiologic development.

This study tested a structural model, examining the relationship between a latent variable termed demoralization and measured variables (anxiety, depression and hopelessness) in a community sample of Canadian youth. The model was stable across demographic variables: sex, grade, and location. Further, the model explains the relationship between sub-clinical anxiety, depression and hopelessness.

Demographic Variables

Previous studies, have demonstrated a distinction between anxiety and depression, with many studies focusing on differences (e.g., Lewinsohn et al., 1993). The results of this study provide preliminary evidence to suggest that it is also valuable to focus on the alternative theoretical perspective. The results of this study indicate that there are coherent statistical commonalities between anxiety and depression that are here labelled “demoralization”.

Differences between sex, age, and location in relation to anxiety, depression and hopelessness have been documented in the literature (Cicchetti et al., 1998, Lewinsohn et al., 1993, Lewinsohn et al., 1997a, Peterson, Sarigiani, & Kennedy, 1991, Mazza & Reynolds, 1998, Nolan-Hoeksema, 1990). The differences were considered in this
analysis and although small differences were noted, they did not contribute to the final model. While differences between anxiety, depression and hopelessness are generally related to sex, grade, and location (e.g., Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998), these variables were excluded from the model because results indicated their inclusion would not improve the model. Hence, the hypothesis that sex would be a strong predictor in the model was not supported. It is not fully understood why sex differences did not manifest in this study, however, the following reasons are speculated. Firstly, all participants were geographically situated within Western Canada which may have confounded results. Secondly, compared to previous studies, social influences may be changing and affecting this population differently. Given that previous studies have been unable to lead to a full understanding of the complexity of this interaction, when focusing on differences (Dozois & Westra, 2004); results of this study indicate that this interaction can be explained by examining similarities within one model.

The nature of age differences or similarities could not be fully examined in the present study due to methodological limitations discussed below. From the present findings, similar to sex, the model was stable across the grades 10, 11 and 12 in the sample. Similarly, the model was stable across location, indicating that location also has no effect.

Relationship Between Anxiety, Depression, Hopelessness and Latent Factor

Demoralization

The correlation between anxiety and depression (.66) was quite consistent with prior findings (e.g., Dobson, 1985; Price & Ingram, 2001; Barlow, 2000), indicating that the relationship between these constructs is relatively stable across samples, instruments
and the statistical techniques used. Given that there is no current explanation for such high correlations between anxiety and depression, our results support the suggestion posed by Mineka, Purdy and Luten (1995), that these two disorders share one similar etiologic origin: demoralization.

All models tested were parsimonious, and met the criteria to explain the relationship between one latent factor and anxiety, depression and hopelessness. The reasons for this conclusion are as follows. First, there was little difference between the estimated coefficients across all three models in the standardized SEM solutions and the standard error values for the three measured variables were small in each model (Table 7, 8, & 9). Second, the overall fit of the final model was assessed. Each model was found to be statistically significant in terms of the relationship between the factor and the measured variables. In addition, goodness of fit indices for each model were within the range of recommended acceptance level greater than 0.9. The final model had an excellent goodness of fit index. Finally, when the coefficients, error values and fit indices were compared across each model, the models were found to converge. Taken together, the results indicate that the final model is robust, adequate and parsimonious.

The findings presented above, in combination with previous research (Barlow, 2000, 2002; Chorpita, 2002, Clarke & Kissane, 2002; Rickelson, 2002; Lewinsohn et al., 2000, Chorpita & Barlow, 1998; Brown et al., 1998) provide strong support for the hypothesized relationship between sub-clinical anxiety, depression, hopelessness and the latent variable (i.e., demoralization).

Demoralization has been identified in the literature as being a construct from which to understand the etiologic origins of anxiety and depression (Frank, 1974; Clarke
& Kissane, 2002; Rickelson, 2002). Indeed, the finding from this study that one factor efficiently explains the relationship between the measured variables in the present study supports a construct such as demoralization. The idea that demoralization is an etiological precursor of anxiety and depression is further supported by the fact that the sample was community-based. Therefore, demoralization may be considered sub-clinical in nature, hence closer to etiologic origin. While some subjects may have met clinical criteria, the majority did not. The latent factor demoralization may accurately represent the relationship of anxiety, depression and hopelessness in a sub-clinical population. As a result, it is reasonable to conclude that the main hypothesis of this study that one model can explain the relationship between anxiety, depression and hopelessness is supported from a statistical and theoretical perspective.

The current model adds support to the triple vulnerability theory (Barlow, 2000, 2002) as well as the model of demoralization (Frank, 1974, Clarke & Kissane, 2002; Rickelson, 2002). For example, the current results support one model that specifically focuses on anxiety, depression and hopelessness explaining their relationship with one latent factor. This model extends support to these theoretical models, especially because it was stable across sex, age, and geographic location. Unfortunately, it was difficult to further explore temporal relations due to the cross-sectional design, which is further discussed below.

Implications

The following will illustrate the importance of the present findings to the preventative literature. Preventative research may help alleviate the interpersonal and societal costs of anxiety and depression that have been noted in the literature (Dozois &
Dobson, 2004; Barlow, 2002; Gotlib & Hammen, 2002). These findings contribute to a theoretical framework, which has implications with educational and clinical interventions. The present findings will help guide further preventative research. The theoretical framework, specifying this model is simple and derived on the basis of the reduction of factors that have explained the relationship between variables and hypothesized one precursor of anxiety and depression.

Viewing demoralization as a precursor of anxiety and depression brings forward the idea that we can examine which disorder will emerge as a function of development or environmental factors (Rickelson, 2002). Past research has focused on identifying risk factors rather than fully understanding the development of structures over time (Price & Ingram, 2001; Cicchetti & Toth, 1998; Rickelson, 2002). This study moves beyond identifying isolated risk factors in specific domains (e.g., cognitive) to examining the relationship between constructs. That is, and how they are integrated within a common etiologic origin.

This approach, theoretically, conceptualizes the development of anxiety, depression and hopelessness and assumes the integration of developmental processes at multiple levels. Hence, this study adds preliminary results to understanding the etiological nature of anxiety and depression through the concept of demoralization (see Appendix A). This method of data reduction is unifying and also may give rise to unification within various discipline-based perspectives in understanding anxiety and depression. Integrating multidisciplinary approaches to treating anxiety and depression may structure our understanding in developmental patterns central to both normative and abnormal development. Training professionals from different disciplines (e.g., teachers,
guidance counsellors, social workers, psychologists, etc.) to identify the “demoralized” adolescent and being aware of appropriate interventions will have early implications on prevention.

One of the core differences between demoralization and depression noted in the literature (Clarke & Kissane, 2002) is that demoralization is based on feelings of incompetence while depression is based on “anhedonia” (see Appendix A). Furthermore, when focusing on preventative programs, attention to focusing on developmental incompetencies (e.g., peer relationships, academic achievement) is likely to benefit a demoralized adolescent and decrease risk of further development of sub-clinical anxiety and depression (Cicchetti & Toth, 1998, Rickelson, 2002).

The finding that the model supports the idea of a precursor has important implications for promotion, prevention and early identification programs. Intervention strategies build on healthy beliefs regarding personal control, central to preventing anxiety (Clarke & Kissane, 2002), which in turn affect success and mitigate against stressful life events. This may allow adolescents to have a sense of self-efficacy and competency, central to preventing demoralization (Clarke & Kissane, 2002). Viewing interventions and preventative programs from a developmental standpoint may contribute to further success in developmental tasks and increase personal control and competence. Therefore, the risk of subsequent demoralization, anxiety, depression and hopelessness may be reduced. Skill-building programs may aid adolescents in effectively meeting challenges and provide experience that reinforces competency, hence increasing resiliency against demoralization.
Based on information from this study, it is now possible to develop strategies to measure the impact of programs on the sensitivity and specificity of early identification instruments. Such development would necessarily depend upon longitudinal study. Longitudinal study could, in this instance, not only identify anxiety as a precursor to depression in sub-clinical populations, but if successful also be able to identify the specific content of prevention and promotion programs that are age appropriate.

Finally, social stigma continues to affect individuals in seeking treatment for sub-clinical anxiety and depression. Further, psycho-education is needed within the community and education system to increase awareness of the effects of not treating sub-clinical symptoms of anxiety and depression; identifying precursor and risk factors; and educating others regarding appropriate resources available. In addition, screening mechanisms need to be put in place in the school system to detect demoralization.

Suggestions for Future Research

Several recommendations can be made to direct future investigations of the relationship between sub-clinical anxiety, depression and hopelessness in Canadian youth. A number of measures should be taken in order to add greater credibility to the instruments used in this study as well as results and conclusions drawn and mentioned above.

Further research is needed in examining the psychometric properties in measuring highly comorbid constructs. In addition, identifying specific measures of demoralization to screen for anxiety, depression and hopelessness would be beneficial. In addition, further interventions need to be developed and tested on sub-clinical anxiety, depression and hopelessness.
Future research could focus more precisely on examining the strength of the observed relations between the particular variables and factors described in this study. Study replication is important in order to validate and support findings. Using item analysis to more specifically pinpoint the common items on the measures that load onto the factor demoralization would strengthen future analysis. Further factor analyses and structural equation modeling should be conducted to support or refute that one latent variable can explain the relationship between anxiety, depression and hopelessness.

As mentioned above, this study was unable to fully explore the relationship between one latent variable and anxiety, depression and hopelessness because item scores were not available to examine. Rather, summed scores were utilized to understand a more general relationship. Research should be conducted to examine possible causal relationships between the measured and latent variables using more specific methods and measures.

It would be of further interest to examine this model in other samples (e.g., clinical samples) to note if equivalent structures and parameters are estimated. In addition, it would be of interest to test the model on adult and child populations to determine if this model applies.

Finally, as noted earlier, structural research of longitudinal data would be of considerable value in verification of demoralization as a common etiologic factor exerting strong influence on the pathogenesis, course, and treatment of anxiety, depression and hopelessness.
Limitations

Caution should be taken when interpreting some results from the present study. First, there are necessary limitations of a cross-sectional, brief quantitative pen-paper survey that each study utilized to collect data. This data represents a “snapshot” of each student’s life on the particular day of testing, and explored associations between variables (such as depression) and identified potential risk factors (such as increased anxiety). There is no evidence from previous studies that indicates causation. Rather, the data suggests that at this point, when the battery was completed, anxiety and depression were observed. The interpretation of the present study is limited by the cross-sectional nature of the previous studies utilized to test the current model. Using longitudinal methodology would help in further understanding developmental implications. That being said, the present findings add to the body of literature examining risk factors the development of anxiety, depression and hopelessness.

Second, although the sample size was large overall, there were several points at which only limited data was available for meaningful analysis. For example, only one of the previous studies included grade nine participants, causing a smaller subgroup of this data for the model that was not generalizable to all strata. For this reason, the grade nine participants were dropped from analysis. In addition, some participants did not indicate sex, causing these subjects to be dropped from sex analysis.

Third, mental health is a multifaceted arena and one must take into account that factors contributing to anxiety and depression include a range of individual, familial, social, community and cultural factors. Identifying risk factors and structural paths are useful in preventative research and early intervention strategies. Research has suggested
that identifying oversimplified pathways to poor mental health outcomes in adolescents can increase their distress. Moreover, it is important to highlight that for every adolescent that does experience a particular risk factor and a subsequent negative mental health effect, there are also many adolescents that experience a risk factor that will not have a negative outcome.

Finally, this data set did not include inventory items. Having all the items within this data set may have identified a more refined and precisely described latent variable as a function of the sub-clinical sample study. Item analysis would also identify more precisely the contents required to ameliorate sub-clinical symptoms related to demoralization in prevention and promotion programs. Furthermore, item analysis would further permit development of much more precise instruments for identifying in sub-clinical populations, those who are at risk for developing clinical anxiety and depression.

Conclusion

Although feelings of anxiety, depression, and hopelessness are prevalent in high school students, not much is known yet about the etiological origin of internalizing disorders in this population. There is a particular lack of research on demoralization as a precursor to sub-clinical anxiety and depression. In the past, research in the area of adolescent anxiety and depression has tended to focus on psychological sequelae and not the mechanisms, which underlie their development. It is argued, from the results of this study, that in order to begin to address long-term effects of anxiety and depression, more focus is needed on underlying mechanisms. Literature focusing on sub-clinical anxiety and depression in adolescence must move away from identifying risk factors and take a look at the mechanisms which may cause the symptomology (e.g., demoralization). More
epidemiological studies are needed to further advance our knowledge in this area, focusing on one common origin to promote better prevention and intervention programs specifically designed for the adolescent population.
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Appendix A. Glossary

Adolescence: A transitional period of development between youth and maturity involving biological, cognitive and socioemotional changes.

Anhedonia: A hallmark characteristic of depression where individuals are unable to feel pleasure.

Anxiety: A state of apprehension, uncertainty, and fear resulting from the anticipation of a realistic or fantasized threatening event or situation, often impairing physical and psychological functioning.

Anxious cognitive content: Within the cognitive model, anxiety-based cognitive content is related to concerns of physical or psychological threat.

Beck Anxiety Inventory (BAI): Developed by Aaron Beck this 21-item questionnaire is used to measure and screen the severity of anxiety symptoms.

Beck Depression Inventory (BDI): Developed by Aaron Beck this 21-item questionnaire is used to measure and screen the severity of depressive symptoms.

Beck Hopelessness Scale (BHS): Developed by Aaron Beck to assess hopelessness, or the extent of negative expectancies about the future.

Behavioral theory: A school of psychology that confines itself to the study of observable and quantifiable aspects of behavior and excludes subjective phenomena, such as emotions or motives.

Bentler-Bonnet normed fit index (NFI): A fit index that specifies the models overall fit compared with the null model.

Bentler comparative fit index (CFI): A fit index that specifies the models overall fit compared with the null model and is also sensitive to size.
Bentler-Bonet non-normed fit index (NNFI): A fit index developed for testing complex models (includes a correction for complexity) and allows values to fall out of the zero to one ranges.

Biological models: A broad theoretical view that holds that mental disorders are caused by some aberrant somatic process or defect.

Classical conditioning: A basic form of learning, also known as Pavlovian conditioning, in which a neutral stimulus is repeatedly paired with another stimulus (called the unconditioned stimulus, UCS) that naturally elicits a certain desired response (called the unconditioned response, UCR). After repeated trials the neutral stimulus becomes a conditioned stimulus (CS) and evokes the same or a similar response, now called the conditioned response (CR).

Cognitive Theory: An approach to depression developed by Aaron Beck that focuses on how thoughts and personal beliefs play a role in the development of depression including negative schemata and interpretations of oneself, the world and the future.

Comorbidity: The co-occurrence of two disorders, as when a person is both depressed and anxious.

Conditioned emotional response: An animal model of anxiety that produced an emotional response in animals using a series of pairings and non-pairings of an unconditioned stimulus (US), such as a shock, with a conditioned stimulus (CS), such as a tone to ascertain if an emotional conditioning is developed, inhibited, or retarded.
Confirmatory factor analysis (CFA): Seeks to determine if the number of factors and the loadings of measured (indicator) variables on them conform to what is expected on the basis of pre-established theory.

Cross-validation strategy: A strategy under which a model is developed using a sub-sample of the data and then confirmed using an independent validation sample.

Defence mechanism: In psychoanalytic theory, reality-distorting strategies unconsciously adopted to protect the ego from anxiety.

Depression: A mood often characterized by an inability to concentrate, insomnia, loss of appetite, anhedonia, feelings of extreme sadness, guilt, helplessness and hopelessness, and thoughts of death.

Depressive cognitive content: Within the cognitive model, depressive cognitive content reflects related cognitions conceptualized in Beck’s Negative Cognitive Triad consisting of negative assessment of self, the world and future.

Demoralization: A phenomenological term coined by Jerome Frank (1974) in an attempt to capture the essence of an individual’s inability to cope and the relationship between feelings of helplessness, hopelessness, meaninglessness, incompetence and low self-esteem.

Diagnostic and Statistical Manual of Mental Disorders (DSM-IV): A publication of the American Psychiatric Association that is an attempt to delineate specific and discrete syndromes or mental disorders. It has been through several revisions and the current one is the Forth Edition (IV).
Diathesis-stress model: As applied in psychopathology, a view that assumes that individuals predisposed toward a particular mental disorder will be particularly affected by stress and will then manifest abnormal behavior.

Endogenous variable: A variable that is caused by one or more variable in the model.

Exogenous variable: A variable that is not caused by another variable in the model.

Usually this variable causes one or more variables in the model.

Exploratory factor analysis (EFA): Seeks to uncover the underlying structure of a relatively large set of variables. The researcher's à priori assumption is that any indicator may be associated with any factor. This is the most common form of factor analysis.

Factor analysis: Is a multivariate technique that is used in this study to reduce a large number of variables to a smaller number of factors for modeling purposes, where the large number of variables precludes modeling all the measures individually. As such, factor analysis is integrated in structural equation modeling (SEM), helping create the latent variables modeled by SEM.

Fear: An emotion produced by present or threatening danger.

Fear-potentiated startle: An animal model of anxiety that equates enhanced emotional response with the desired effect rather then using suppression of ongoing behaviours as an indicator of an emotional state to provide parallel similarities in increased sympathetic arousal often present in human cases.

Generalized heritable vulnerability: The first diathesis described within the triple vulnerability model that assumes that some people might inherit anxious traits.
Generalized psychological vulnerability: The second diathesis described within the triple vulnerability model that assumes vulnerability is based on the relationship between anxiety and a sense of unpredictability and uncontrollability.

Hopelessness: Having no hope; a symptom of depression and a predictor of suicide attempters and completers.

Hopelessness theory of depression: A theory that highlights hopelessness as a symptom and cause to depression and emphasizes the difference between helplessness and hopelessness. Helplessness is present in both anxious and depressed individuals whereas only depressed individuals appear to become hopeless or give up regaining control.

International Classification of Diseases (ICD): Used to classify diseases and other health problems recorded on many types of health and vital records including death certificates and hospital records. The ICD enables the storage and retrieval of diagnostic information for clinical and epidemiological purposes.

Intrapsychic models: Models that focus largely on how individuals perceive themselves and the world. An individual’s perspective is therefore viewed as a product of experience and learning under this perspective.

Latent variable: A variable in the model that is not measured.

Learned helplessness theory: This theory posits that individuals acquire passivity and a sense of being unable to act and to control their lives; happening through unpleasant experiences and traumas against which their efforts were ineffective; according to Seligman, this brings on depression.
Learned hopelessness theory: This theory assumes that people, in face of negative life events, become passive and depressed when they attribute negative life events to stable and global causes.

Maximum likelihood estimation: Is a statistical means used to estimate all model parameters simultaneously.

Measured variable: Also referred to as indicator variables, is a variable that can be observed directly and is measurable.

Modeling: Learning by observing and imitating the behaviour of others.

Multivariate analysis: A branch of statistics concerned with the analysis of multiple measurements, made on one or several samples of individuals.

Negative affect (NA): A common general factor that both anxiety and depression share as assumed by the tripartite theory.

Negative cognitive triad: In Beck’s theory of depression, a person’s baleful views of the self, the world, and the future; the triad is in a reciprocal causal relationship with pessimistic assumption and cognitive biases.

Physiological hyperarousal (PH): Identified in the tripartite theory as a specific factor related to anxiety.

Positive affect (PA): Identified in the tripartite theory as a specific factor related to depression.

Psychodynamic theory: Also referred to as psychoanalytic theory of personality developed by Sigmund Freud that focuses on repression and unconscious forces and includes the concepts of infantile sexuality, resistance, transference, and division of the psyche into the id, ego, and superego.
Punishment-conflict model: An animal model that uses operant techniques to elicit well-established behaviours, and then punishes the behaviours using aversive stimuli causing the behaviours to subside. The suppression in behaviour is thought to parallel the passive-avoidant component of anxiety.

Reformulated theory: Developed in response inadequacies of the learned helplessness theory, this theory posits that individuals attribute negative life events to global, stable and internal factors.

Self-efficacy: An individual’s expectations in achieving set goals, sense of control, personal agency and a focus on how one perceives their own success.

Separation and abandonment model: An animal model of anxiety model that is based on the manipulation of social interactions to demonstrate fear responses in the absence of dangerous situations.

Specific psychological vulnerability: The third diathesis described within the triple vulnerability model described as the factor in which individuals learn from early experience.

State anxiety: An unpleasant emotional arousal in face of threatening demands or dangers.

Structural equation modeling: A statistical technique for building and testing models, which are often causal in nature. It is a hybrid technique that often encompasses aspects of exploratory and confirmatory factor analysis.

Trait anxiety: Reflecting stable individual differences regarding the tendency to respond with state anxiety in anticipation of a threatening situation.
Tripartite model: A model of emotion developed by Watson and Clark (1991) in an attempt to explain the high comorbidity rates between anxiety and depressive disorders, proposing that there is a common general factor that both anxiety and depression share: negative affect (NA).

Triple vulnerability theory: A model integrating biological, psychological and social factors that contribute to the development and maintenance of anxiety and related mood disorders.

Risk factor: A condition or variable that, if present, increases the likelihood of developing a disorder.
Appendix B. Certification of Calgary Board of Education
Appendix C. Parent/ Guardian Informed Consent Form
Appendix D. Participant Informed Consent Form
Appendix E. Study Materials
Appendix F. Debriefing Form
Appendix G. Certification of University of Calgary Conjoint Faculties Research Ethics Board