

2012

# Latent Classes of Self-Reported Adolescent Depression in a Clinical In-Patient Population

Jonathan F. Doti  
*Utah State University*

Follow this and additional works at: <http://digitalcommons.usu.edu/etd>

 Part of the [Psychology Commons](#)

---

## Recommended Citation

Doti, Jonathan F., "Latent Classes of Self-Reported Adolescent Depression in a Clinical In-Patient Population" (2012). *All Graduate Theses and Dissertations*. Paper 1157.

This Dissertation is brought to you for free and open access by the Graduate Studies at DigitalCommons@USU. It has been accepted for inclusion in All Graduate Theses and Dissertations by an authorized administrator of DigitalCommons@USU. For more information, please contact [dylan.burns@usu.edu](mailto:dylan.burns@usu.edu).



LATENT CLASSES OF SELF-REPORTED ADOLESCENT DEPRESSION  
IN A CLINICAL IN-PATIENT POPULATION

by

Jonathan F. Doti

A dissertation submitted in partial fulfillment  
of the requirement for the degree

of

DOCTOR OF PHILOSOPHY

in

Psychology

Approved:

---

Susan L. Crowley, Ph.D.  
Major Professor

---

Gretchen G. Peacock, Ph.D.  
Committee Member

---

Jamison D. Fargo, Ph.D.  
Committee Member

---

Michael P. Carey, Ph.D.  
Committee Member

---

Maria G. Norton, Ph.D.  
Committee Member

---

M. Scott DeBerard, Ph.D.  
Committee Member

---

Mark R. McLellan, Ph.D.  
Vice President for Research and  
Dean of the School of Graduate Studies

UTAH STATE UNIVERSITY  
Logan, Utah

2012

Copyright © Jonathan F. Doti 2012

All Rights Reserved

## ABSTRACT

Latent Classes of Self-Reported Adolescent Depression in a  
Clinical In-Patient Population

by

Jonathan F. Doti, Doctor of Philosophy

Utah State University, 2012

Major Professor: Susan Crowley, Ph.D.  
Department: Psychology

The depressive disorders are among the most common mental health problems with substantial financial and quality-of-life costs. Depression has generated considerable debate as to the underlying structure and the taxonomy continues to be frequently debated. Adolescents who meet diagnostic criteria for major depressive disorder often experience anxiety (and vice versa). Emerging statistical approaches such as latent class analysis (LCA) have utility for understanding the underlying structure of depression as well as the co-occurrence of depression and anxiety. An LCA of adolescents with depression would add to our conceptual understanding of the disorder(s) and facilitate treatments of adolescents with depression and potentially those with co-occurring anxiety symptoms. The current study adds to the body of literature on the latent structure of depression and co-occurring anxiety of a juvenile in-patient sample. LCA was conducted on an in-patient sample of juveniles ( $N = 722$ ). Analyses yielded six distinct classes or

subtypes of depression that were different from each other on overall symptom severity as well as the presence or absence of anhedonia. Results may have implications regarding subtypes of adolescent depression, comorbidity of anxiety, and our understanding of the taxonomic structure of categorical versus dimensional aspects of depression diagnosis. Results suggest subclinical features of anxiety commonly co-occur with depression among juveniles, suggesting a common construct of adolescent distress made up of both depression and anxiety.

(119 pages)

## PUBLIC ABSTRACT

Latent Classes of Self-Reported Adolescent Depression in a  
Clinical In-Patient Population

by

Jonathan F. Doti, Doctor of Philosophy

Utah State University, 2012

The depressive disorders are among the most common mental health problems with substantial financial and quality-of-life costs. Depression has generated considerable debate as to the underlying structure/taxonomy and continues to be frequently debated. Adolescents who meet diagnostic criteria for major depressive disorder often experience anxiety (and vice versa). Therefore, understanding the underlying structure of depression as well as the co-occurrence of anxiety in a population of adolescents adds to our conceptual understanding of these disorders and facilitates treatment clarity.

This investigation sought to investigate the following research questions for adolescents' self-reported symptoms of depression, and self-reported symptoms of depression and anxiety in combination.

1. Are there latent subtypes or classes that can be identified from an in-patient sample?
2. How do the latent subtypes of depression and anxiety relate to clinical diagnoses?
3. How do participants in each latent class differ on age, gender, and symptom severity?

Results have implications regarding subtypes of adolescent depression and the comorbidity of anxiety among adolescents. Results contribute to our understanding of the taxonomic structure of categorical versus dimensional aspects of a mood diagnosis. Additionally, the benefit of our findings adds to our understanding of the subclinical features of anxiety that commonly co-occur with depression among juveniles. Results suggest a common construct of adolescent distress made up of features both depression and anxiety that fosters greater treatment clarity.

## ACKNOWLEDGMENTS

I thank my dissertation chair, Dr. Susan Crowley, for her help throughout this long and at times difficult process. She was always cheerful and encouraging throughout the development of this dissertation. Among her best qualities was her reliability in answering my many questions while I was away from campus. Her prompt replies reduced my anxiety greatly and let me know she was always on my side. She also facilitated untold piles of official paperwork and deadlines while I was away on military assignments.

My good friend, Leo, also deserves thanks; he visited me often and sat quietly as I wrote many of these pages. I looked forward to his silent encouragement.

However, among all the people I would like to thank there is one who stands above all others. She was first a fellow student, a friend, a girlfriend, and then my wife. She knows me better than anyone else; sometime better than I know myself. She has been a steady companion who has made all of this possible and I am certain without her help I would have lost the energy and focus to complete this project. I could list all the things she has done but that would distract from the greater good she has been for me. She has rekindled my courage for the future and brought laughter simply through the goodness of a person's love. Therefore, I thank my wife, Rachel Duchoslav, for her constancy, commitment, and hope for positive outcomes in almost all things. She is the most intelligent and kind person I know—and I feel an immense debt of gratitude to her.

Jonathan F. Doti

## CONTENTS

	Page
ABSTRACT.....	iii
PUBLIC ABSTRACT .....	v
ACKNOWLEDGMENTS .....	vi
LIST OF TABLES.....	ix
LIST OF FIGURES .....	xi
CHAPTER	
I. INTRODUCTION .....	1
II. REVIEW OF LITERATURE .....	5
Introduction to Adolescent Depression.....	5
Etiology.....	8
Child and Adolescent Depression Research .....	13
Comorbidity .....	16
Subclinical Symptoms .....	17
Significance of Taxonomy.....	22
Categorical Versus Dimensional Diagnoses.....	22
Latent Class Analysis.....	26
Summary and Conclusions .....	28
III. METHODS .....	32
Participants.....	32
Measures .....	33
Procedures.....	37
Analyses.....	38
IV. RESULTS .....	42
Preliminary Analyses.....	42
Initial Latent Cluster Analyses.....	43
Second Latent Cluster Analyses .....	54



VI. DISCUSSION.....	70
Description of Clusters .....	71
Categorical Versus Dimensional Diagnostic Models .....	83
Limitations .....	87
Future Directions for Research.....	88
REFERENCES .....	90
CURRICULUM VITAE.....	102

## LIST OF TABLES

Table	Page
1. Demographic Variables for Participant Sample .....	34
2. Diagnostic Groupings with Subsumed Clinical Diagnoses .....	35
3. Descriptive Statistics for Indicator Variables and Total Scores .....	43
4. Correlations Between Indicator Variables and Total Scores .....	44
5. BIC(LL) for Cluster Models Based on RADS Indicator Variables .....	45
6. Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS Only) .....	48
7. Cluster Characteristics Across Indicator Variables .....	49
8. Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS Only; Combined Across All Diagnoses) .....	51
9. Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS Only; Primary Diagnosis Only) .....	52
10. Descriptive Statistics for Psychosocial Variables .....	53
11. Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS Only) .....	55
12. Gender Differences Within Clusters (RADS Only) .....	56
13. BIC(LL) for Cluster Models Based on RADS and RCMAS Indicator Variables .....	57
14. Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS and RCMAS) .....	61
15. Cluster Characteristics Across Indicator Variables .....	62
16. Descriptive Statistics for Clusters and Total Sample within Diagnostic Categories (RADS and RCMAS; Combined Across All Diagnoses) .....	63

Table	Page
17. Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS and RCMAS; Primary Diagnosis Only) .....	65
18. Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS and RCMAS) .....	68
19. Gender Differences Within Clusters (RADS and RCMAS).....	69
20. Typical Individual Per Cluster .....	72
21. Cluster Severity Labels .....	78

## LIST OF FIGURES

Figure	Page
1. Path values for four RADS indicator variables.....	46
2. Z scores of four indicator variables across clusters .....	47
3. Path values for six cluster model with RADS and RCMAS indicator variables .....	58
4. Z score of seven indicator variables across clusters.....	59

## CHAPTER I

### INTRODUCTION

Major depressive disorder (MDD) is a complex, prevalent, etiologically multifaceted, and clinically heterogeneous disorder. From a broad perspective, the depressive disorders or mood disorders are among the most common mental health problems with substantial financial and quality of life costs. It has been estimated that the financial costs related to mood disorders are currently well above \$44 billion a year (Lynch & Clarke, 2009). The World Health Organization (WHO, 2009) asserted that the spectrum of depressive disorders are responsible for more total impairment than arthritis, asthma, and diabetes combined; by the year 2020, it is predicted that only cardiovascular disease will have more negative overall impact (e.g., Mossavi et al., 2007; Murray & Lopez, 1996). Epidemiological studies indicate that one out of every six U.S. adults will meet the diagnostic criteria for a mood disorder some time in their life (Kessler et al., 2005). Comparatively, studies involving children and adolescents reveal that they endorse a disproportionate number of depressive symptoms (Kessler, Foster, Webster, & House, 2001). Evidence indicates that first episodes of depression are occurring at increasingly younger ages with escalation of reoccurrence across childhood and adolescence (Kessler et al., 2005). Additionally, comorbidity with anxiety disorders makes definitive diagnoses difficult due to clinical presentation and conceptual overlap (Robins, Locke, & Regier, 1991).

Nevertheless, depression is assumed to comprise a robust and naturally distinguishing presentation of symptoms that demarcates itself from other disorders.

However, depression as a taxonomic construct has generated considerable debate surrounding the structure underlying symptom observations and self-report. Therefore, the taxonomy of mental disorders and specifically MDD continues to be ardently debated (Pickles & Angold, 2003). Some have gone as far as to assert that the American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; 2000) criterion thresholds are somewhat arbitrary and the "rarity" of symptoms between margins of the mental disorders is not entirely supported (Kendler, Gardner, & Prescott, 1998; Widiger & Samuel, 2005). Rather, current diagnostic systems force inclusion or exclusion into separate diagnostic categories based on the presence of specific symptoms; evidenced by diagnostic thresholds that have been created, eliminated, or simply changed as the DSM has evolved. Further, the conceptual organization of psychological disorders reflects a medical-model of pathology with strict category thresholds and margins that are complicated by diagnostic comorbidity. Meehle (1954) was among the first to call for taxonomy based upon "naturally occurring joints" or "rarity of symptoms" between disorders without forcing a category merely for the sake of convention or convenience.

As mentioned, the incidence of depression significantly increases from adolescence into early adulthood. Prospective epidemiological studies affirm that adolescents with MDD are at a two to four times greater risk for depression in early adulthood (Pine, Cohen, Cohen, & Brook, 1999). Depressive symptoms such as hopelessness, psychomotor retardation, guilt, disruption of mood, low energy, and reduced motivation combine to form a valid, well recognized, and distinct disorder and

yet many individuals may not meet the criterion threshold for diagnosis despite significant symptoms.

The term subclinical refers to the presence of some symptoms of a mental health disorder that are not sufficient or adequate in meeting diagnostic criteria for that mental disorder. However, subclinical symptoms of depression are not equivalent to being asymptomatic and are predictive of later depressive events (Fergusson, Horwood, Ridder & Beautrais, 2005). The DSM-IV-TR (APA, 2000) criterion for the depressive disorders, while useful and generally assumed to be accurate, forces important subclinical information to be excluded (Andrews et al., 2007). Subclinical symptomology may be especially useful in identifying adolescents who experience depressive symptoms and comorbid problems that may lead to later depression. Yet, this information is not currently captured by the DSM diagnostic system.

Adolescent depression commonly co-occurs with anxiety (Ferdinand, De Nijs, van Lier, & Verhulst, 2005) and the research literature confirms that adolescent depression and anxiety have a high rate of comorbidity (e.g., Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Karlsson, Wallerström, Götherström, & Holmlund, 2000), with similar patterns of comorbidity among adults (e.g., Angold & Costello, 1993; Biederman, Faraone, Mick, & Lelon, 1995; Keller, Kocsis, & Thase, 1998; Lewinsohn, Gotlib, & Seeley, 1995; Rohde, Lewinsohn, & Seeley, 1991). Often, adolescents who meet diagnostic criteria for major depressive disorder experience distressing, but subclinical, levels of anxiety (and vice versa). Comorbidity complicates diagnosis and is generally given a secondary position by the DSM-IV categorical classification system. Use of strict

diagnostic categories can result in valuable information unheeded because those who score just below clinical threshold are regarded as “noncases.” However, emerging statistical approaches such as latent-class-analysis (LCA) have utility for understanding the co-occurrence of anxiety and depression, as well as the latent structure of depression. LCA identifies mutually exclusive classes of data or clusters. Each cluster has unique characteristics, and ideally each would be homogenous within the cluster on the variables assessed (e.g., symptoms of depression) with large differences exist between classes (Ferdinand et al., 2005).

A number of important questions can be addressed applying LCA to depressive symptoms, and depressive and anxious symptoms in combination. These analyses may add to our conceptual understanding of adolescents with depression and co-occurring anxiety symptoms, and inform our interventions for these adolescents. The current proposed study will add to the body of literature on the possible latent structure of depression and co-occurring anxiety in a juvenile in-patient sample.



## CHAPTER II

### REVIEW OF LITERATURE

#### **Introduction to Adolescent Depression**

This review of literature will provide a framework for the current research. This literature review will begin with a brief discussion of depression in general (e.g., history, prevalence, and epidemiology). Next, the etiology of depression will be outlined, including the biological risk factors, cognitive disruptions, genetic risk factors, and psychological/social risk factors. Then, the research foundations in child and adolescent depression will be highlighted, including the research on comorbidity, subclinical symptoms, and their implications. Next, the significance of taxonomy of depression will be discussed from a categorical vs. dimensional perspective. Latent Class Analysis will next be reviewed, and its applications to this study. Finally, summary and conclusions of the current literature will be considered.

#### **History**

The mood disorders and specifically MDD have been labeled the “common cold” among mental health problems. Much of the experience of depression is expected as a normal reaction to common life circumstances such as loss, failure, and other distressing events. It is assumed that a “normal” cycle of depressive affect is time-limited and even functionally adaptive by redirection of goal behaviors and resource allocation (Nesse, 2006). However, marked and unrelenting depression clearly can result in a host of complications if left untreated.

Terms such as dysthymia (bad mood) and mania (insanity) were first used to describe and categorize what currently are considered the mood disorders. Hippocrates (4<sup>th</sup> century B.C), considered the father of medical science, described depressive symptoms as an “aversion to food, despondency, sleeplessness, irritability, and restlessness.” The ancient Greeks and Romans recognized the interplay between personality, temperament, and environmental circumstances long before current diathesis-stress models implicated biological, personality, and environmental factors. From the earliest records through modern taxonomies there have been attempts to conceptualize depression beyond simple problem lists. However, it is the diffuse nature of depression that makes universal acceptance of a conclusive taxonomy so challenging and debate continues on this conceptually elusive disorder.

As recently as the 1970s, it was maintained that children and adolescents were unable to experience depression similar to adult depression. The bulk of researchers and clinicians no longer hold this view and depression in youth is seen as comparable to depression in adulthood. During adolescence, rates of MDD rise in an approximately linear fashion with a notable distinction; the rate of adolescent males’ depression declines slightly while that of adolescent females increases noticeably (Anderson, Williams, McGee, & Silva, 1987). By their early 20s, females are twice as likely to be diagnosed with MDD compared to their male counterparts.

### **Frequency and Prevalence**

MDD is pervasive. Nearly one in six individuals in the U.S. experience at least one lifetime depressive episode of clinical significance and many have multiple episodes

(Sutton, 2007). At any given time, significant symptoms of depression affects from 5 to 20 million U.S. adults (Kessler, Chiu, Demler, & Walters, 2005). Lifetime prevalence estimates for MDD range as high as 17% of the U.S. population and 12-month prevalence rates conservatively ranging from 3.5-7%, with more liberal estimates proposed (Ebmeier, Donaghey, & Steele, 2006; Kessler et al., 1994; Waddell, Hua, Godderis, & McEwan, 2004).

The WHO (2009) maintained that MDD is the leading cause for psychological disability in the U.S. between ages 15 and 44. Experts predict that by the year 2020, depression will be the second leading cause of all disabilities (physical and psychological) worldwide—including many chronic health concerns such as diabetes and hyper-tension (Mossavi et al., 2007; National Institute of Mental Health, 2003). Following MDD, anxiety disorders are the second most frequent mental health concern (American Psychiatric Association, 2009).

### **Epidemiology**

Generally, the average age of onset for the first episode of clinical depression occurs between the mid-20s and mid-30s. However, there is considerable variance in severity, duration, and heterogeneity (Jyhla, 2008). The Baltimore Epidemiological Catchment Area study reports the average duration of MDD is from 8 to 12 weeks (Eaton et al., 1997), while a more recent study reports that the average duration of MDD lasts much longer, up to 28 weeks (Kennedy, Abbott, & Paykel, 2003). The average duration of a MDD episode fluctuates upon criterion and methodology of data collection but a general consensus of 12 weeks is typical (Ustun & Kessler, 2002). Factors such as prior

episodes and their severity, as well as comorbid conditions foretell longer recovery times and relapse.

Of note, roughly 80% of adults who have experienced a single episode of MDD will have at least one additional lifetime episode (Mueller et al., 1996). In a 5-year follow-up study after initial diagnosis, a large majority of adults experienced one further episode while 29.3% had no reoccurrences, contrasted by 27.9% who had three or more subsequent episodes (Holma, Melartin, Holma, & Isometsä, 2008).

### **Etiology**

The etiology of MDD is affected by several factors in line with a diathesis-stress model with individual and environmental factors assumed responsible in origin and maintenance. These factors include but are not limited to: genetic predispositions (Levinson, 2006), low birthweight (Costello, Erkanli, & Angold, 2006), hormonal and neurobiological effects (Nestler et al., 2002), predisposing personality traits (Hirschfeld, Klerman, Clayton, & Keller, 1989), poor parenting and parental depression (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002), parental loss (Kessler, Zhao, Blazer, & Swartz, 1997), parental conflict and divorce (Gilman, Kawachi, Fitmaurice, & Buka, 2003), childhood physical and or sexual abuse (Kendler & Prescott, 2006), early anxiety disorder (Kessler et al., 1996), nominal social support (Kendler & Prescott, 2006), substance abuse (Kessler et al., 1996), prior MDD (Lewinsohn, Hoberman, & Rosenbaum, 1988), and stressful life events (Kendler & Prescott, 2006). By the age of 18, a sizable 15% to 20% of adolescents have experienced a major depressive episode; this

does not include subclinical depressive features that do not meet diagnostic criterion. For reasons not fully understood, the depressive disorders are occurring earlier in successive cohorts (Birmaher et al., 1996). As previously stated, there is a persistent gender effect with females consistently at two to three times greater risk for depression compared to males across all ages. Possible socializing effects, biological predisposition, and cultural expectation/demands may partially explain the effects of gender on rates of depression. There is also a persistent family effect, with first-degree relatives at two to three times greater risk compared to controls (Klerman & Weissman, 1989a, 1989b).

### **Biological Risks**

At one time, depression was seen as being solely the result of environmental factors such as developmental history, trauma, and/or stress. Research in the last few decades confirms that depression, like many other disorders, has a strong biological foundation. A large body of evidence supports that depressed individuals often have disturbances of endocrine, immune, and neurotransmitter system functioning.

Current imaging technology reveals that the hippocampus area of the brain is smaller in many depressed individuals. On average, the hippocampus of the brain is statistically 9% to 13% smaller in depressed individuals compared with those who are not depressed. In general, the more frequent the episodes of depression, the smaller the hippocampus. Stress, which plays a role in depression, may be an important factor in hippocampal loss, as long-term stress suppresses the production of neurons in the hippocampus. Animal models of stress suggest that the increased release of glucocorticoid over a prolonged period result in excitotoxic damage and reduced

neurotrophins, explaining hippocampal volume loss (Campbell & Macqueen, 2004).

Antidepressants appear to counter the loss of hippocampus volume and result in improved mood and functioning. While antidepressants almost immediately boost the concentration of neurotransmitters in the brain, typically their positive effects are not experienced for several weeks to months after initiation of medication treatment. Researchers have questioned why there was a pronounced delay in improved mood if depression was primarily the result of low levels of neurotransmitters, which were immediately elevated by antidepressant medication. One explanation posits that neurons first need to grow and form new synaptic connections that occurs over many weeks. Therefore, synaptic growth may be the foundation for improved mood rather than an immediate increase in neurotransmitters per se. Animal model studies reveal that antidepressants stimulate neurogenesis and dendritic branching of nerve cells in the hippocampus (Eisenberger, Lieberman, & Kipling, 2003).

### **Cognitive and Neurochemical Disruption**

Research clearly supports neurochemical alterations in depression with impairment of cognitive functioning. Episodic memory is especially affected in those with MDD, as well as executive functioning and psychomotor slowing (Ebmeier et al., 2006). Compared to nondepressed controls, disruption of working memory, verbal fluency, set-shifting, and inhibition processes have been observed in adults and juveniles diagnosed with MDD. From a clinical perspective, cognitive disruptions may further impede clinical therapeutic progress.

The importance of the monoamines, especially noradrenalin and serotonin, in the

treatment of clinical depression is well accepted. Almost all antidepressants, including tricyclics and monoamine oxidase inhibitors, increase synaptic concentrations of a particular monoamine; dopamine, serotonin, and noradrenalin (Malhi, Parker, & Greenwood, 2004). However, a simple monoamine deficiency hypothesis is not fully satisfactory in explaining the genesis and pathophysiology of depression.

Pharmacological studies strongly implicate serotonin, dopamine, and noradrenalin as neurochemical sites of action. However, to target a cause of depression as one or more neurotransmitters does not take into account, in many cases, the moderate failure of antidepressants to ameliorate depressive symptoms (Malhi et al., 2004).

### **Genetic Risks**

MDD is believed to have a strong genetic component with early age of onset and relapse variance likely inherited (Bierut et al., 1999; Kendler & Magee, 1993; Sullivan, Prescott, & Kendler, 2002).

The Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPUD; Kendler & Prescott, 2006) systematically explored the role of genetic and environmental risk factors and their interaction in the etiology of common disorders. Internalizing and externalizing disorders such as generalized anxiety disorder, major depression, phobias, childhood conduct disorder, adult antisocial personality disorder, and substance use were broken down into four developmental time-frames. Similar to other genetic studies of depression, the omnibus model for this study (Kendler & Prescott, 2006) accounted for an average of 50% of the probability for an episode of MDD. Interestingly, Kendler, Kuhn, and Prescott (2004a) indicated that the genetic risk factors

for internalizing disorders (e.g., depression and anxiety disorders) were different than the genetic risk factors for externalizing disorders (e.g., conduct disorder and antisocial personality disorder). Further, the internalizing disorders reveal a strong common genetic link for nearly all of the risk for depressive and anxiety disorders, suggesting a common neurobiological mechanism for internalizing disorders. In contrast, poor parenting, parental loss, childhood sexual abuse, and the ill-defined term “low-self-esteem” were only modestly related for later risk for mental health problems underlying depression (Kendler et al., 2004a).

### **Psychological/Social Risks**

Risk factors influencing depression include problematic patterns of thinking, deficits in coping skills, impaired emotional regulation, and under-developed emotional intelligence. Additional factors such as traumatic experiences, early separation, and lack of social support are also some of the psychological correlates (e.g., Goodman & Gotlib, 1999). Research in this area indicates that significant and long-term stress is capable of serving as a trigger for the expression of genes resulting in changes in brain functioning that may lead to subsequent depressive symptoms (Hankin & Abela, 2005). The probability of developing these problems is influenced by a wide range of interrelated risk factors including genetic liability, neurophysiologic dysfunctions, predisposing temperament/personality traits, adverse childhood circumstances, limited interpersonal resources, and chronic and traumatic events (e.g., Ormel & Neeleman, 2000; Rothman & Greenland, 1998). Additionally, since twice as many women suffer with depression, female gender could be considered a risk factor as well.



### **Child and Adolescent Depression Research**

Research on depression has focused primarily on adults, with considerably less attention paid to the understanding of depression in childhood and adolescence. However, compelling longitudinal studies have established the impact of depression across all ages, including young children who were once thought unable to experience depression due to developmental naïveté (Jyhla, 2008; Kessler et al., 2005; Waddel et al., 2004).

For a diagnosis of MDD, an individual must experience persistent depressive or irritable symptoms, or suffer significant loss of interest/pleasure in most activities for at least two weeks. Marked changes in mood, thoughts, and behaviors must also be accompanied by at least four additional criterion symptoms: insomnia or hypersomnia, psychomotor agitation or retardation, significant weight loss or gain, fatigue or loss of energy, feelings of worthlessness or excessive or inappropriate guilt, inability to think or concentrate, and recurrent thoughts of death, suicide ideation, suicide attempts, or a credible and specific plan for carrying out suicide (APA, 2000). Further, symptoms of depression must substantially impact an individual's capacity in domains of home, school, work, and interpersonal functioning.

There is no definitive test for depression and thus any diagnosis is based upon multiple sources including client report, detailed history including review of medical records/past mental health reports, objective measures, projective assessments, and even confidant reports to round out expert observations (APA, 2008; Waddel et al., 2004).

As aforementioned, depression was once considered the sole domain of adulthood. Most would now agree that "...today the question is not whether children can

suffer from depression but rather how many adult mood disorders are truly ‘adult onset,’ and how many are recurrent episodes of a disorder that had its onset in childhood or adolescence....” (NIMH, 2003, p. 56). The complex interplay between biological, psychological, and social mechanisms in the onset, maintenance, and resolution of depressive symptoms is especially important when considering emotional, cognitive, social, and physical changes occurring in childhood and adolescence (Lewinsohn, Pettit, Joiner, & Seely, 2003a; NIMH, 2008).

Depression that begins in youth has implications for later adult depression. Lewinsohn and colleagues (2003a) reported that the differences between relative rates of depression and symptoms between adolescents and young adults are small and lack clear qualitative boundaries. While others have found that the overall manifestation of MDD in youth was not markedly different than in adults. An epidemiological study of psychological disorders concluded differences between adolescent and adult symptoms of depression were small—so small as to conclude that depression in adolescence and adulthood are essentially equivalent (Lewinsohn et al., 2003a; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003b). The Oregon Adolescent Depression Project’s (OADP) data supports previous results suggesting that MDD in adolescents and young adults is fundamentally indistinguishable (i.e., Carlson & Kashani, 1988). These findings reinforce that DSM criterion for adults are valid and useful with adolescents.

Results from the OAPD indicate that the most common symptoms among adolescents diagnosed with MDD were depressed mood (97.7%), sleep disturbances (88.6%), poor concentration (81.8%), appetite disturbances (79.5%), and anhedonia

(77.3%). Additionally, over half of the adolescents with a MDD diagnosis had frequent thoughts of suicide or death (54.5%). No significant gender differences in the expression of adolescent depression were observed other than anticipated elevated rates of MDD among females (Lewinsohn et al., 2003).

### **Adolescent Depression Leading to Early Adult Onset**

In the OADP study (Lewinsohn et al., 2003), MDD in adolescence was associated with pervasive difficulties in young adulthood. Of those adolescents (prior to age 19) diagnosed with MDD, follow-up 5 years later (age 24) found 62% of this cohort experienced significantly more difficulties including more stressful life events, more physical complaints, lower likelihood to have graduated from college, and greater unemployment. Compared to adolescents diagnosed with other psychological disorders, only those diagnosed with MDD were significantly more likely to have difficulties in young adulthood such as low academic performance, early childbearing and marriage, greater use of mental health services, and experiencing a major adversity. The impact of childhood depression on cognitive abilities, long-lasting personality changes, and susceptibility to substance abuse foreshadows a chronic course (Waddell et al., 2004). Additionally, significant negative childhood events such as sexual abuse, parental loss, and parental death are associated with a greater incidence of depression (Kendler & Prescott, 2006).

There is empirical support for “pathway” or “vulnerability” models for adult onset depression following childhood adversity (Costello et al., 1996; Korkeila et al., 2005;

Rice, van den Bree, & Thaper, 2004). Early childhood trauma, loss of parent, divorce, and sexual abuse are some of the potential predisposing factors for later depression. In addition, idiosyncratic personality styles partly explained by genetic expression influence the manner in which individual's structure and interact with their environment. It has also been asserted that individuals may engage with their environment in a manner that perpetuates a depressive cycle (Jyhla, 2008; Kendler, Kuhn, & Prescott, 2004b). For example, an individual with depressive features may interpret benign interactions as negative, reinforce opportunities to express their unhappiness, and elicit negative appraisals and therefore foster an environment that reduces support, decreases positive interactions, and limits opportunities to improve mood.

### **Comorbidity**

Like depression in adulthood, juvenile depression seldom exists in isolation. Compared to adult depression, the literature indicates that children and adolescents with depression exhibit greater variability in clinical characteristics (e.g., age of onset, course, and severity), patterns of neurobiological correlates, and social profiles of risk. In addition, treatment response varies considerably among depressed youth (NIMH, 2008). Juvenile depression commonly coexists with at least one other major mental health disorder; increasing the likelihood that individuals will also have an anxiety disorder (eight times more likely), conduct and oppositional disorders (six times more likely), and attention-deficit-hyperactivity disorder (five times more likely) when compared to juveniles who are not depressed (NIMH, 2003; Robins et al., 1991).

Moreover, depression and anxiety were more likely to co-occur than depression and attention-deficit hyperactivity disorder, the spectrum of disruptive behavior disorders or substance use disorders (Costello et al., 2003). On self-reported measures of anxiety, hopelessness, and self-esteem, Stark, Humphrey, Laurent, Livingston, and Christopher (1993) reported that children (ages 9-12) who had been diagnosed with depression, anxiety, or joint depression and anxiety symptoms did not statistically differ in clinical presentation among diagnosed groups. They concluded that among children and likely adolescents, depression and anxiety form an overriding feature that they referred to as “negative affectivity.” The tripartite model of depression and anxiety (Clark & Watson, 1991) shares a similar viewpoint. The tripartite model advances that anxiety and depression share a common feature of high negative affect. However, depression and anxiety are thought to differ on anhedonia or low positive affect (unique to depression), and physiological hyper-arousal (unique to anxiety).

### **Subclinical Symptoms**

Subclinical is a term used to describe symptoms of a disorder not numerous or severe enough to meet formal diagnostic criteria. Over the course of five revisions since 1952, the current DMS-IV-TR has incorporated clinically relevant maladies filling in intervals between more familiar and prevalent disorders. Minor depressive disorder, brief recurrent depression, and dysthymia are examples of current DSM-IV-TR attempts to add diagnostic categories that were not considered adequately severe to warrant separate diagnoses. Some researchers have even called for a new category of depression termed,

“subsyndromal symptomatic depression” (SSD) to capture significant features of depression not meeting the current standard for diagnosis but detrimental enough to warrant clinical attention (Sadek & Bona, 2000).

By DSM-IV criterion, individuals who do not endorse anhedonia and or depressed mood for at least a two week period fall short of the standard for clinical depression. Compared to the not-otherwise-specified (NOS) designation; SSD, is defined as a depressive condition having two or more symptoms of depression of the same quality as in major depression, excluding the defining markers of depressed mood and or anhedonia (Sadek & Bona, 2000). Nevertheless, SSD and similar attempts speak to the need to improve underlying diagnostic clarity.

Mounting empirical evidence indicates individuals with subclinical depression are not equivalent to being asymptomatic (Fergusson et al., 2005). Subclinical levels of depressive symptoms are implicated in a wide variety of medical and psychological problems (Pincus, Davis, & McQueen, 1999) and include increased mental health complaints (Skodol, Schwartz, & Dohrenwend, 1994), more reported substance abuse (Lewinsohn, Rohde, Seely, Kelin, & Gotlib; 2000), higher rates of attempted suicide (Fergusson et al., 2005), overall decreased functional ability (Judd, Akiskal, & Paulus, 1997), reduced health (Judd et al., 1997), increased sick days (Wells, Burnam, Rogers, Hays, & Camp, 1992), increased number of days with pain (Wells et al., 1992), and poorer outcomes on chronic conditions such as diabetes and coronary diseases (Katon, 2003). It has been estimated that in its totality, subclinical depression consumes more service resources than the total allocation assigned to the formal diagnoses of MDD and

dythstymia combined (Johnson, Weissman, & Klerman, 1992).

Research indicates that in medical settings, mental health problems may be implicated in as many as half of all patients reporting a physical complaint (Olfson, Sing, & Schlesinger, 1999). Wells and colleagues (1992) reported that participants with subclinical symptoms of depression were 25% more likely to suffer from MDD within two years. Gotlib, Lewinsohn, and Seeley (1995) further reported that among adolescents with no prior depressive diagnoses, subclinical depressive features were a risk factor predicting later MDD. Current diagnostic systems rely a great deal on the number of clinically elevated depressive symptoms when making a diagnostic decision. However, this leaves those without the necessary number of symptoms as noncases who therefore do not receive a diagnosis leading to a lack of focused care. A meta-analysis of 25 studies revealed that individuals with subclinical levels of depression had a higher morbidity compared to those free of depressive symptoms. The authors concluded that the risks of subclinical depressive features were not appreciably smaller than in clinical depression (Bostwick & Pankratz, 2000).

### **Implications of Subclinical Depressive Symptoms**

While clinical thresholds have been the standard from which to understand adolescent depression, many have also focused on subclinical symptomology in the etiology of mood disorders. Many child and adolescent cases of anxiety, disruptive behavior, moodiness, social alienation, and substance abuse are often interrelated with subclinical depressive symptoms (Lewinsohn et al., 2003; NIMH, 2008; Pine et al., 1999;

Waddell et al., 2004). From a dimensional model, severity of symptoms from minimal through severe warrant attention since depression in youth is often comorbid with developmentally related conditions such as peer problems, poor parental care, childhood sexual abuse, and personality dysfunction (Ferguson et al., 2005).

### **Comorbid Anxiety**

Childhood anxiety, in particular, is noted as a risk factor for depression and frequently precedes symptoms of depression (Pine, Cohen, Gurley, Brook, & Ma, 1998). This has led to assertions that an anxiety disorder in childhood may be predictive of later adolescent depression (Piccinelli, Rucci, Ustun, & Simon, 2007). Epidemiological studies suggest anxiety and depression even share a common genetic etiology (e.g., Rice et al., 2004).

Efforts to study subtypes of depressive and anxiety disorders have found mixed clusters that have included symptoms of both disorders. In fact, researchers have found that pure clusters/cases of adolescent depression or anxiety rarely exist without comorbid meaningful symptoms of the other (e.g., Eaton, Dryman, Sorenson, & McCutcheon, 1989). Similarly, Kendler, Neale, Kessler, Heath, and Eaves (1992) identified significant comorbidity of depression and anxiety in studies of generalized anxiety (GAD) in female twins. In their findings, a substantial 30% of the adult twins met *DSM-III-R* diagnostic criteria for GAD as well as major depressive disorder. Using the same sample of twins, Kendler and colleagues (1996) discovered three clusters of depressive subtypes: a mild depressive group, an atypical/eating-disordered depressive group, and a severe depressive group that also met criteria for GAD and specific phobias. Regular overlap between



depression and anxiety problem items has been found in quantitative analyses in clinical samples across the lifespan (Achenbach, 1991a, 1991b; Achenbach & McConaughy, 1997). These and similar studies add to a growing body of evidence suggesting that there are subtypes of the depressive disorders with comorbid anxiety and vice versa (e.g., Parker, 1999).

Pine and associates examined phobias/anxiety at age 13 and the researchers found that anxiety at age 13 predicted MDD at age 16 (Pine, Cohen, & Brook, 2001). Similarly, levels of “anxious and withdrawn behaviors” at age 8 were found to later predict risk for MDD in adolescence (Goodwin, Lewinsohn, & Seely, 2004). Further, Moffitt and colleagues (2007) demonstrated that depression and anxiety had a reciprocal relationship; where one preceded the onset of the other from childhood through middle adulthood (ages 11-32) (Moffitt et al., 2007). In a longitudinal community study (Costello et al., 1996) of juveniles (ages 9-13), the odds of a depressive and an anxiety disorder co-occurring was nearly thirty times more likely than either a pure case occurring separately.

Kovacs and Devlin (1998) suggested that contrasted to more psychologically mature adolescents, children may be more biologically sensitive to experience anxiety rather than depression due to developmental capacity. It remains unclear how the relationship between childhood anxiety and adolescent depression is affected by developmental maturity (Rice et al., 2004). Evidence suggests anxiety and depression could be regarded as a continuum of symptoms mediated by biological and psychological advances in development rather than mutually exclusive experiences (Van den Oord, Pickles, & Waldman, 2003).

### **Significance of Taxonomy**

Conceptual understanding of the latent structure of depression and other disorders may lack focus (Clark, Watson, & Reynolds, 1995). Meehl and Rosen (1955) stated that taxonomy is the science of organizing information according to naturally existing groupings and relationships. Taxonomic organization evaluates seemingly unrelated data, facts, ideas, methods, and assumptions making them more useful. The challenge of taxonomy is adhering to the ‘naturally occurring’ points of rarity between data indicative of existing groupings rather than merely imposing convenient organization. Cronbach, Meehle, and Watson asserted that the goal of science, especially in psychology, was to delineate the taxonomy among disorders and establish the boundaries of phenomena in order to understand what was being observed and how to classify it (Cronbach & Meehl, 1955; Clark et al., 1995). Meehl (1992) reasoned that distinguishing the potential latent structure of a construct such as a psychological disorder is a critical scientific goal forming basic research and refinement of theory. Therefore, clarity subtypes of depression and its relationship to anxiety is fundamental for conceptual understanding.

### **Categorical Versus Dimensional Diagnoses**

Traditional categorical systems such as the DSM-IV-TR, originating in the United States, and the ICD-10, employed by the majority of the rest of the world, reflect a categorical diagnostic disease model. Among these models of disease, clinical criterion symptoms are either present or absent (DSM-IV-TR; APA, 2000). However, mounting empirical evidence suggests that depression, rather than different in *type*, is more likely

different in *degree* when compared to the notion of “normal” (Coyne, 1994; Flett, Vredenburg, & Krames, 1997; Ruscio & Ruscio, 2000, 2002a, 2002b, 2004a, 004b, 2004c).

The DSM-IV-TR (APA, 2000, p. xxxi), states that “there is no assumption that each category of mental disorders is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder”. However, in a categorical model like the DSM-IV-TR the threshold of diagnosis for depression is met when the requisite number of criterion items allow for an all or nothing diagnosis, notwithstanding some allowances for severity once a diagnosis is established. While a vast improvement over previous versions, the current DSM-IV-TR still maintains some diagnostic boundary overlap problems due to somewhat arbitrary distinctions between classes of disorders (Widiger & Samuel, 2005).

Early researchers in the field of taxonomy have pointed out that inaccurate theory and problems in the operationalization of constructs underlie many misleading assumptions of “natural joints” that separate between and within disorders (Cronbach & Meehl, 1955). The struggle with conceptualizing what is and what is not depression is reflected in the variety of diagnostic labels and types. The depressive disorders and subtypes have spawned a variety of labels over the previous century that have included: unipolar, bipolar, mixed, dysphoric, anhedonic, neurotic misery, nuclear, incomplete, attenuated, mild, residual, recurrent, sociotropic, anaclitic, atypical, secondary, masked, postnatal, double, minor, brief, melancholic, agitated, seasonal affective, reactive, endogenous, and NOS to name some.

Like many disorders, MDD can be viewed as a dimensional continuum (Brown & Barlow, 2005) with individuals having varying levels of depressive symptoms, and these symptoms are considered as simply higher or lower in number and intensity on a range of normal through disordered. From this perspective, somewhat artificial diagnostic thresholds fail to recognize the impact of impairments at the subclinical level of symptomology (Ruscio & Ruscio, 2000). In the case of the mood disorders each subsequent version of the DSM widens the margins of inclusion suggesting that the foundations to this class of disorders are conceptually malleable due to developing understanding (Widiger & Samuel, 2005).

The taxonomic debates on the most meaningful way to organize the upcoming DSM-V have wrestled with calls for additional continuous criterion considerations. A recent APA and WHO congress on the taxonomy of disorders concluded:

...[are there] ways by which addition of continuous, “dimensional” measures into the various diagnostic domains might help resolve some of the critical taxonomic issues currently facing the field of mental health.... It was overtly recognized that categorical and dimensional approaches to diagnosis are important for clinical work and research, and the ideal taxonomy would offer both. However, to avoid diagnostic chaos, the dimensional scale must reflect the categorical definition and the two must have a clear and obvious relationship to each other. (Helzer et al., 2008. p. 116)

Therefore, the need to incorporate dimensional aspects to provide accuracy of symptoms by including subclinical features is made clear. During a recent National Institute on Mental Health (NIMH, 2008) roundtable on adolescent depression, there was general agreement that the application of the “spectrum concept of depression” would provide a more valid perspective in conceptualizing depression in youth through inclusion of subclinical symptoms. Existing DSM-IV organization does not adequately

account for clinically important characteristics and symptoms that fail to meet diagnostic criteria. Further, the high prevalence of “not otherwise specified” (NOS) diagnoses indicate that a categorical approach often fails, in practice, to discern symptoms at the subclinical level (Widiger & Samuel, 2005). Brown and Barlow (2005) commented:

... The DSM does not provide a sufficient mechanism to record the severity of disorders (e.g. the severity of depression rather than the presence-absence of comorbid mood disorder per se may be more relevant to the prediction of the treatment outcome or natural course of a principle anxiety disorder). Salient information is also lost by adherence to the DSM’s elaborate set of hierarchical exclusions and differential diagnostic decision rules. Adherence to diagnostic rules of this nature leads to considerable information loss and misleading findings about the overlap of various disorders. (p. 552).

The conceptual foundations of depression are complex. The etiology and presentation of depression offers a rich array of features. Yet the broad nature of depression can be problematic due to overlapping conditions clouding definitive diagnosis. Termed the “waste paper basket” of diagnosis, the NOS designation reflects comorbid diagnostic confusion that a dimensional model may alleviate (Widiger & Samuel, 2005).

Any taxonomy reflects, in part, the zeitgeist of its time and therefore the definitions of depression have ultimately shifted (Ruscio & Ruscio, 2000). MDD and many other mental health disorders may more likely be both “categorical *and* dimensional” rather than “categorical *or* dimensional” (Brown & Barlow, 2005; Ruscio & Ruscio, 2000). Kendler and Gardner (1998) asserted that DSM definitions of depression may be a forced diagnostic convention imposed on a natural continuum of depressive symptoms of varying severity and duration.

Conventional taxonomic approaches found in DSM-IV-TR and ICD-10 have

delineated various types and subgroupings within the depressive spectrum. The construct of depression as a continuum of symptoms rather than a dichotomous diagnosis may allow inclusion of less severe yet important subclinical characteristics (Brown & Barlow, 2005; Fergusson et al., 2005). The idea that a criterion threshold is merely an artificial convention superimposed upon a continuum of depressive symptoms has been presented in the past, and therefore is not without precedence (Brown & Barlow, 2005; Ebmeier et al., 2006). However, a clear nosology has not yet been convincingly developed.

### **Latent Class Analysis**

Over ten years ago researchers predicted that "...many studies of the continuity issue require a level of statistical sophistication that is quite advanced and further tests of the continuity issue may require the use of complex statistical techniques" (Flett et al., 1997, p. 410). As more capable computer algorithms/programs make exhaustive computations practical, the mathematical ability to investigate latent class membership of complex data sets has grown (Dunn, Sham, & Hand, 1993). LCA is a promising tool for the elaboration of the construct of depression (Morgan, Sargent, Chukwuma, & Huges, 2008).

Fundamentally, latent class/cluster analysis and related models of statistical testing classify similar objects/populations/qualities into groups when the total number of groups and the characteristics of those groups are unknown. A standard LCA method, similar to traditional cluster analysis, is used to fit data to a one-cluster model followed by a two-cluster model, then three-class model, and so on; providing a parsimonious fit to

the data.

The essential theory underlying latent class analysis involves the concept of local, or conditional, independence that asserts that persons/cases in the same latent class share a mutual probability distribution for observed variables. Within each latent cluster or subset, each variable is statistically independent of every other variable. Since persons/cases in the same latent cluster cannot be differentiated from each other based on evident responses, they are therefore homogeneous or alike with respect to the observed variables. In other words, latent clusters are distinct in that if one removes the effect of latent class membership on the data, what remains is “randomness” or more specifically “independence.” The LCA approach defines one cluster per latent class, using model-based probabilities to classify cases and permits investigation of supposed subsets of group membership (i.e., Muthen & Muthen, 2004).

LCA is also similar to cluster/factor analysis, in that both approaches are used to uncover groups of cases based on observed data. Approaches like factor and cluster analyses are “aggregative” procedures that form groups/cases based upon parameter features of a disorder. While useful, factor and cluster statistical approaches may computationally “force” categories where no natural categories exist. This may artificially force data to fit a construct rather than the other way around, resulting in incorrect assumptions of latent constructs (Haslam, 2003). While approaches such as cluster/factor analysis focus on the structure of variables/correlations; LCA is used to understand the structures of cases/latent factors. Both LCA and cluster/factor analysis are effective in data reduction but LCA also allows inference based on both observed and

unobserved data (Ferdinand et al., 2005).

There are numerous advantages in employing LCA; mixed measurement data sets comprised of nominal, ordinal, continuous, and discrete data can be employed with no confounding assumptions of linearity or equal spacing within a measurement scale. LCA takes into account both observed and assumed unobserved or latent variables that are believed to exist in most psychological constructs and relaxes the strict provisions of assumptions of local independence of linearity, normal distribution, and homogeneity. Unlike traditional statistical models that assume continuous variability within a population, LCA assumes that individuals tend to cluster around distinct subgroups. Therefore, LCA can help identify classes of data with their own relative unique set of symptom profiles and statistical probabilities. Unlike traditional clustering procedures, where ad hoc agreements within a discipline/theory are used to determine the number of clusters, LCA clusters are based on a statistical model that mathematically determines the most parsimonious number of clusters. Although this is not intended to be an exhaustive list of the advantages of LCA, they are compelling reasons to employ this statistical approach. Generally and in shortened form, when evaluating LCA results, each set of LCA probabilities are optimal when each class is homogenous and large differences exist between classes.

### **Summary and Conclusions**

Depression is astonishingly ubiquitous with nearly 1 in 6 Americans experiencing clinical episodes in their lifetime (Kessler et al., 2005). However,



depression is among the most heterogeneous disorders; it is believed there are distinctive subtypes of depression with unique developmental characteristics (Kendler et al., 1996). In addition, there is increasing recognition that subclinical depression is not equivalent to being asymptomatic but rather is associated with later potential for disability. (Lewinsohn, Soloman, Seely, & Zeiss, 2000). Depressive symptomatology that lacks the severity to meet diagnostic threshold may be common in adolescent populations and precede clinical depression in early adulthood (Fergusson et al., 2005).

While some other classes of mental disorders are more concrete in our understanding, the taxonomy of mood disorders is not as easily conceptualized. Disorders can fall along a continuum, and as many researchers in the field of taxonomy now purport, most disorders have both categorical and dimensional aspects. Distinguishing or integrating between the two perspectives has importance for both researchers and clinicians. The latent taxonomic structure of adolescent depression also exposes the foundations of how we perceive the structure of mental disorders in general.

Broadly, the current DMS-IV-TR is a categorical disease model with minimal allowances for severity such as “specifiers.” Calls have been made for the latest iteration of DSM-V to include a continuity or quantitative view, maintaining there is a linear relationship in the spectrum from mild through severe depressive symptoms. There is a long-standing taxonomic debate over whether depression is, in fact, better explained as a collection of syndromes or as a single phenomenon that differs mainly in terms of severity (Flett et al., 1997).

The latent class statistical approach may be helpful in illuminating unique subsets

of depression (Stoolmiller, Kim, & Capaldi, 2005). Use of categorical diagnostic construct, like the DSM-IV and ICD-10, "...can result in loss of valuable information about comorbidity, because those who score just below the diagnostic threshold are regarded as non-cases. A dimensional approach does not solve this problem, because it cannot be used to divide individuals in homogeneous subgroups. Latent class analysis (LCA) can be used to solve the shortcomings of both approaches" (Ferdinand et al., 2005, p. 300).

Having a useful taxonomy is essential for empirical and clinical goals. Therefore, the need for a taxonomic system that can ascertain clusters/groups of individuals with like symptoms of depression and anxiety, sharing a common etiology and accordingly may require similar treatments (Wadsworth et al., 2001). Identifying factors that differentiate subgroups and clinical trajectories are vital in providing focused treatment. As the study of depression has evolved, our underlying conceptual taxonomic foundations driving treatment assumptions must be accurate.

The present project sought to investigate depression in an in-patient juvenile population, taking into consideration comorbid anxiety and subclinical levels of symptoms. This current study sought to investigate the latent classes of depression and possible associated clinical features that could be overlooked by a categorical approach. These analyses would add to our conceptual understanding of adolescents with depression and co-occurring anxiety symptoms. The current proposed study will add to the body of literature on the possible latent structure of depression in a juvenile in-patient sample.

The utility of incorporating possible latent features/constructs of depression and anxiety in a clinical setting may expand our understanding and subsequently treatment of the experience of juvenile problems. Rather than approaching depression and anxiety as separate disorders, there may be unique facets to childhood and adolescent psychological problems that warrants approaches that incorporates treatments that targets a wider range of factors.

This investigation addressed the following research questions for adolescents' self-reported symptoms of depression, and self-reported symptoms of depression and anxiety in combination.

1. Are there latent subtypes or classes that can be identified from an in-patient sample?

2. How do the latent subtypes of depression and anxiety relate to clinical diagnoses?

3. How do participants in each latent class differ on: age, gender, and symptom severity?

## CHAPTER III

### METHODS

#### **Participants**

Participants for the current study were drawn from an extant data set of over 850 children and adolescents ages 5 through 18. This population of youth was admitted for inpatient treatment at a large academic medical center in the Midwest spanning the years 1990 through 2003. The academic medical center treats patients from a sizeable catchment area made up of rural, suburban, and urban communities. Consent for participation was obtained from the guardians of youth at the time of hospital admission as part of the intake process.

For the present study, participants were included if they were between the ages of 12 and 18 at admission and were able to complete self-report measures (RADS, RCMAS). Participants were excluded if they had a diagnosis (DSM-III-R or DSM-IV) of mental retardation or if they had more than 5% of the items missing on the RADS or RCMAS. The original data set contained 1106 cases. From this data set, 140 cases were excluded because they were not in the specified age range. An additional 102 cases and 119 cases were excluded because they did not complete either the RADS or RCMAS, respectively. An additional six cases were excluded due to a diagnosis of mental retardation and the final 17 cases were excluded due to missing data on either the RADS or RCMAS. The final data set for analysis, after all exclusionary criteria were met, contained 722 cases. Subjects for this study ranged from ages 12 through 18 years (mean

age = 14.99 years,  $SD = 1.35$ ). The sample was 59.8% female and the predominantly self-identified race/ethnicity was Caucasian (80.1%). The majority of the participants were referred for hospital admission by their legal guardians/parents (52.5%). A sizable number had previous psychiatric admissions (29.1%). Demographic variables for the participants are in Table 1.

To look meaningfully at diagnoses, individual diagnoses were collapsed into broad diagnostic labels based on current DSM-IV categories. The created diagnostic groupings, as well as the specific diagnoses that they contain, are presented in Table 2.

Out of the sample of adolescents, 684 participants were given a primary diagnosis, while 452 participants were given an additional second diagnosis, and lastly 115 of the participants were given a third diagnosis. Thirty-two participants did not have a recorded diagnosis.

## Measures

### Reynolds Adolescent Depression Scale

The Reynolds Adolescent Depression Scale (RADS; Reynolds, 1986) is a well-established self-report measure designed to assess symptoms of depression in adolescents aged 12 through 18. Comprised of 30 items rated from 1 to 4, summed scores can range from 30 to 120 with scores 77 and greater suggestive of clinical levels of depression. Four subscale scores are captured: dysphoric mood (8 items), anhedonia/negative affect (7 items), negative self-evaluation (8 items), and somatic complaints (7 items). The

Table 1

*Demographic Variables for Participant Sample (N = 722)*

Demographic variable	<i>n</i>	Valid % of sample
Age		
12	5	.7
13	116	16.1
14	152	21.1
15	174	24.1
16	159	22.0
17	112	15.5
18	4	.6
Gender		
Male	290	40.2
Female	432	59.8
Race		
Caucasian	578	80.1
African-American	76	10.5
Hispanic	34	4.7
Native American	3	.4
Other	8	1.1
Bi-racial	20	2.8
Family situation prior to admission		
Both natural parents	181	25.1
Both adoptive parents	33	4.6
Single parent	236	32.7
Single parent and step parent	119	16.5
Living with relative(s)	47	6.5
Foster parent(s)	42	5.8
Other (group home, etc.)	36	5.0
Past psychiatric Hospitalizations		
Yes	210	29.1
No	487	67.5
Special education placement		
None	536	74.2
Severely behaviorally handicapped	55	7.6
Learning disabled	50	6.9
Developmentally handicapped	35	4.8
Other	35	4.8

*Note.* Not all demographic variables were available for all subjects.

Table 2

*Diagnostic Groupings with Subsumed Clinical Diagnoses*

Diagnostic category	Included diagnoses
Mood ( $N = 604$ )	Depressive disorders Bipolar disorders Adjustment disorders with depressed mood, mixed
Anxiety ( $N = 115$ )	Anxiety disorders
Psychosis ( $N = 4$ )	Psychotic disorders
Somatoform ( $N = 1$ )	Body dysmorphic disorder
Substance-related disorders ( $N = 82$ )	
Eating disorders ( $N = 24$ )	
Disorders diagnosed in childhood ( $N = 120$ )	ADHD Tourette's syndrome Enuresis
Externalizing ( $N = 175$ )	ODD Conduct disorder Anti-social personality disorder Intermittent explosive disorder Disruptive behavior disorder NOS

*Note.* The  $N$  sizes provided represents all diagnoses given to each participant (multiple participants had up to three diagnoses).

internal consistency reliability of the four RADS subscales is moderately high, ranging from .80 to .87 (Reynolds, 1987).

The RADS has moderate to high convergent validity with similar measures of clinical depression; a review of ten studies demonstrated that the Pearson correlations between the RADS and the Beck Depression Inventory-Adolescent (BDI-A) range from .70 to .76 (Reynolds, 1987). The RADS has high internal consistency, with a coefficient alpha ranging from .909 to .939 for inter-item consistency (Reynolds, 1987). The RADS also has good test retest reliability ranging from .80 at 6 weeks to .79 at 3 months (Reynolds, 1987). Subscale scores and total score data will be used in the present study.

### **Revised Children's Manifest Anxiety Scale**

The Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1985) is a self-report measure designed to assess the level and nature of trait anxiety in youth ages 6 through 18. Comprised of 37 items, this measure assesses anxiety (28 items) and social desirability (9 items). The RCMAS items are responded to in a yes/no format and scored 0 (no) or 1 (yes). Summed scores for the anxiety items range from 0 through 28. The RCMAS has age and gender based norms, and a RCMAS total raw score above 19 is considered in the clinical range. The measure has three subscale scores: Physiological Anxiety (10 items), Worry/Oversensitivity (11 items), and Social Concerns/Concentration (7 items). The RCMAS has internal consistency reliability of subscales with scores ranging from .64 to .76 (Reynolds & Richmond, 1985). The RCMAS has good convergent validity ( $r = .85$ ) with another well-known measure of anxiety, the State-Trait Anxiety Scale for Children (Reynolds & Richmond, 1985). The test-retest reliability coefficients are also high, ranging from .98 at three weeks to .68 at 9 months (Reynolds & Richmond, 1985). Subscale scores and total score data will be used in the present study.

### **Millon Adolescent Personality Inventory**

The Millon Adolescent Personality Inventory (MAPI; Millon, Green, & Meagher, 1982) is a well-established self-report measure designed to identify, predict, and understand a wide range of psychological attributes characteristic of adolescents aged 13-18 years. Comprised of 150 true/false items, the measure takes 20-30 minutes to complete. Subscales include eight personality styles (introversive, inhibited, cooperative,



sociable, confident, forceful, respectful, sensitive), eight expressed concerns (self-concept, personal esteem, body comfort, sexual acceptance, peer security, social tolerance, family rapport, academic confidence), and four behavioral correlates (impulse control, social conformity, scholastic achievement, attendance consistency). The internal consistency reliability of all 20 MAPI subscales is moderately high, ranging from .67 to .84 (Millon et al., 1982).

The MAPI has moderate convergent validity with similar measures of adolescent personality (California Psychological Inventory, 16 PF, and Edwards Personal Preference Scale) ranging from .38 to .70. Two test-retest studies produced stability coefficients generally within the acceptable range. Only five subscale scores will be used as psychosocial data in the present study (sociable, impulse control, self-concept, peer security, family rapport; Millon et al., 1982).

### **Procedures**

The Institutional Review Board (IRB) at the medical center approved the procedures for collecting data from participants beginning in 1993 and was reviewed annually for compliance to IRB standards. Use of the extant data for research purposes was approved in 2005 by the IRB at the medical center and for the current study in 2008 by the Utah State University IRB. Data used in the present study were collected from each participant within several days of hospital admission as part of their routine intake psychological evaluation. All of the data were obtained as part of the course of regular treatment protocols. The children, adolescents, or their parents/guardians received no

reimbursement for participation.

Collected information included psychosocial history, medical history, clinical interview, and a range of self-report and parent-report measures. Those with poor reading skills were administered the self-report measures orally by either a psychology intern or a member of the nursing staff. Within 1 to 3 days of admission to the hospital, semi-structured clinical interviews with the child or adolescent and their parent(s) or guardian(s) by the psychiatry staff occurred. All self-report measures were completed within four days of hospital admission.

All data were entered into a data base by the original investigator for the study (Dr. Michael Carey) or by one of several psychology interns completing predoctoral psychology internships at the site. All data were collected between 1990 and 2003. Additionally, the extant data were then verified by a review of the patient's charts several years after initial data collection ended to ensure accuracy and completeness. To ensure confidentiality, no identifying information of any individual was included in the data set.

### **Analyses**

Analyses in the present study were guided by three questions that sought to investigate latent groupings of adolescent depression using LCA. As a statistical method, latent class models encompass a group of similar methods for finding subtypes of related cases of latent classes from complex multivariate data. Latent class analysis also offers a way to confirm hypothesized subtypes such as diagnostic subcategories from larger multivariate data. More specifically, an LCA model refers to any statistical model in

which unobserved subgroups differ on some identified parameters. The difference in model parameters distinguishes cases in different latent classes from one another (Vermut & Magidson, 2004).

Following initial LCA analyses, a conditional bootstrap (Bootstrap -2LL Diff) may be used to help determine the number of classes to include in a model. The conditional bootstrap analysis assesses whether a more restrictive form of a model (e.g., one containing fewer classes) has the best fit. In the present research, multiple models were estimated with different numbers of latent classes. Then, various statistical criteria, including the conditional bootstrap, were used to identify the most statistically robust model.

### **Overview: Estimating LC Cluster Models with Continuous Variables**

The following is a brief conceptual overview of the process of running a Latent Cluster Analysis within the Latent Gold 4.0 system (Vermut & Magidson, 2004). First, indicator variables are identified (in the present study, the indicator variables were the subscales of the RADS and RCMAS). Next, the numbers of desired clusters are designated (in the present study, one-cluster through eight-cluster models were evaluated).

The Latent Gold Program can then estimate a model summary for each of the designated models and summary statistics and indicators of model fit are examined; the specific indicator of model fit that was used in the current study was the BIC(LL). Decreasing values indicates that one is approaching the best model fit. The model with

the lowest BIC(LL) value is likely the best model fit; however, this needs to be confirmed through further analysis.

To confirm that the model with the lowest BIC(LL) value is truly the model with the best fit, a bootstrap -2LL difference test is conducted. The identified model with the lowest BIC(LL) value is compared to the models with one more and one less cluster to see if there is a statistically significant difference between the models. Each time the bootstrap  $p$  value is estimated, 500 samples from the data set are randomly selected. Therefore, each time the bootstrap  $p$  value is estimated the results will be somewhat different due to random sampling of the data. If the bootstrap analysis yields a significant  $p$  value ( $p < .05$ ), then the lowest BIC(LL) value is indeed statistically significantly lower than the compared others and the model is identified as having the best fit. If the difference test is not statistically significant, then the fit of the two models is equivalent. In such cases, alternative criteria can be used to identify the best fitting model including the extant literature, relevant theory, and parsimony.

After identifying the model with the best fit, the coefficients for each loading or path for each indicator variable can be estimated. The variance accounted for by each indicator variable can be calculated through the square of the path value ( $R^2$ ). The path values and  $R^2$  indicate the relative strength and predictive value of each indicator variable in determining cluster assignment.

In the current study, the research questions were addressed individually using LCA. The focus of the project was adolescent depression and potential subtypes within adolescent depression, and the sample was analyzed based on the four indicator variables

of the RADS initially. Afterwards, due to frequent comorbidity of adolescent anxiety and depression, the sample was analyzed using the RADS and the additional three subscale indicator variables of the RCMAS. The first analyses utilized only the four RADS indicator variables, and needs to be differentiated from the second analyses, which utilized the four RADS indicator variables combined with the three RCMAS indicator variables. For ease of understanding, when the three research questions are evaluated using the first set of indicator variables (RADS only) , they are referred to as Research Question #1(a), # 2(a), and #3(a). When the three research questions are evaluated using the second set of indicator variables (RADS and RCMAS), they are referred to as Research Question #1(b), # 2(b), and #3(b). In addition to utilizing Latent Gold to conduct the LCA, SPSS was used for additional data analyses including descriptive statistics, analysis of variance, and chi-square statistics to investigate the make-up of each cluster, their differences, and the factors which determine cluster assignment of participants.

## CHAPTER IV

### RESULTS

This section begins with descriptive statistics for the entire sample related to the seven indicator variables (four subscales of the RADS [dysphoric mood, anhedonia, negative self-evaluation, somatic complaints] and three subscales of the RCMAS [physiological anxiety, worry/oversensitivity, social concerns]). The research questions are then addressed individually using LCA. As previous stated, the research questions will be addressed using the four indicator variables from the RADS. Afterwards, due to frequent comorbidity of adolescent anxiety and depression, the research questions were addressed using the seven indicator variables from the RADS and the RCMAS. Therefore, two LCA models will be developed and analyzed.

#### **Preliminary Analyses**

For each of the indicator variables and total scores from the RADS and RCMAS descriptive statistics are provided in Table 3 including mean, standard deviation, range, skewness, kurtosis, and internal consistency reliability. Internal consistency reliability is generally acceptable, with the exception of the social concerns and physiological anxiety scales, which were somewhat low (.64 and .67, respectively). As expected, participants reported significant depressive symptoms and the mean on the RADS approached the recommended clinical cutoff of 77. The shape of the distribution for the subscales and total scores was generally normal.

Correlations between indicator variables and RADS and RCMAS total scores are

Table 3

*Descriptive Statistics for Indicator Variables and Total Scores (N = 722)*

Variable	Mean	SD	Range	Skewness	Kurtosis	Internal consistency
Dysphoric mood	21.05	5.912	8-32	-.378	-.634	.86
Anhedonia	16.61	5.849	7-28	.233	-.953	.86
Negative self-evaluation	19.34	6.432	8-32	-.113	-.999	.87
Somatic complaints	18.95	4.925	7-28	-.439	-.328	.80
RADS total	75.95	16.886	30-115	-.408	-.478	.93
Physiological anxiety	4.42	2.695	0-10	.159	-.944	.67
Worry/oversensitivity	5.82	3.552	0-11	-.130	-1.261	.76
Social concerns	3.65	2.206	0-7	-.047	-1.144	.64
RCMAS total	13.88	7.459	0-28	-.027	-1.063	.82

*Note.* Clinical cutoff scores (raw) for RADS total = 77, clinical cutoff scores (raw) for RCMAS = 19.

presented in Table 4. Correlations ranged from .009 through .918. In general, correlations between and across measures were statistically significant. An exception to the high correlations was the Anhedonia scale, which had appreciably lower correlations than the other indicator variables, and ranged from .004 to .375.

### **Initial Latent Cluster Analyses**

In the following pages, Research Questions 1 through 3 will be addressed utilizing LCA with the four indicator variables from the RADS. This first LCA analysis will address each of the three research questions utilizing depressive symptoms only. To differentiate the first analysis (four RADS indicator variables) from the second analysis (which utilized four RADS indicator variables *and* three RCMAS indicator variables), the initial analyses will refer to Research Question # 1(a), #2(a), and #3(a), while the second analyses will be identified as Research Question #1(b), #2(b), and #3(b).

Table 4

*Correlations Between Indicator Variables and Total Scores*

Variable	Dysphoric mood	Anhedonia	Negative self-evaluation	Somatic complaints	RADS total score	Physiological anxiety	Worry/oversensitivity	Social concerns
Anhedonia	-.009							
Negative self-evaluation	.744*	.061						
Somatic complaints	.764*	.028	.689*					
RADS total score	.853*	.375*	.864*	.831*				
Physiological anxiety	.612*	.064	.587*	.706*	.666*			
Worry/oversensitivity	.761*	.004	.609*	.600*	.675*	.652*		
Social concerns	.695*	.103*	.702*	.586*	.717*	.627*	.698*	
RCMAS total score	.789*	.056	.710*	.714*	.774*	.857*	.918*	.855*

\* Correlation is significant at the .0001 level (2-tailed).

**Research Question #1(a)**

The first research question asked whether there are latent subtypes of adolescent depression that can be identified from an in-patient sample. LCA was conducted to determine the underlying structure and potential latent class models of depression using the raw scores of the four subscales of the RADS as indicator variables. The four indicator variables were: (RADS) dysphoric mood, anhedonia/negative affect, negative self-evaluation, and somatic complaints. All variables were identified as continuous in the analysis. One- through eight-cluster models were investigated and the BIC(LL) was used as the primary indicator of model fit. The BIC(LL) values for one- through eight-cluster solutions are presented numerically in Table 5.

A review of the BIC values indicates that there is a decrease at the six-cluster solution model; with the BIC(LL) value beginning to increase at the seven-cluster model. Therefore, initial review of the BIC(LL) values indicates that the six-cluster model was



Table 5

*BIC(LL) for Cluster Models Based on RADS Indicator Variables*

Model solution	BIC(LL)
One-cluster model	18350.769
Two-cluster model	17299.095
Three-cluster model	17063.499
Four-cluster model	16965.683
Five-cluster model	16916.559
Six-cluster model	16890.088 <sup>a</sup>
Seven-cluster model	16891.487
Eight-cluster model	16917.303

<sup>a</sup>The six-solution model was the most statistically meaningful fit for the data.

the best solution. However, it was not clear if the six-cluster solution was statistically significantly lower than the other cluster solutions. The six-cluster model was deemed to be superior to the seven-cluster model, as it had a lower BIC value and was more parsimonious. The fit of the six-cluster model was empirically compared to that of the five-cluster model. To evaluate which cluster solution provided the best fit to the data, a bootstrap -2LL difference test was conducted comparing the six-cluster model to the five-cluster model. The six-cluster model provided a statistically significantly better fit than the five-cluster model (-2LL Diff = 85.71,  $p < .0001$ ). Therefore, the six-cluster model was identified as the best solution and was the basis for answering the remaining research questions.

The path values for the four indicator variables are shown in Figure 1. The path values ranged from .64 to .88. All path values were statistically significant and suggest that each variable significantly impacts cluster assignment. The variance accounted for by the predictors ranged from 41% to 77%.

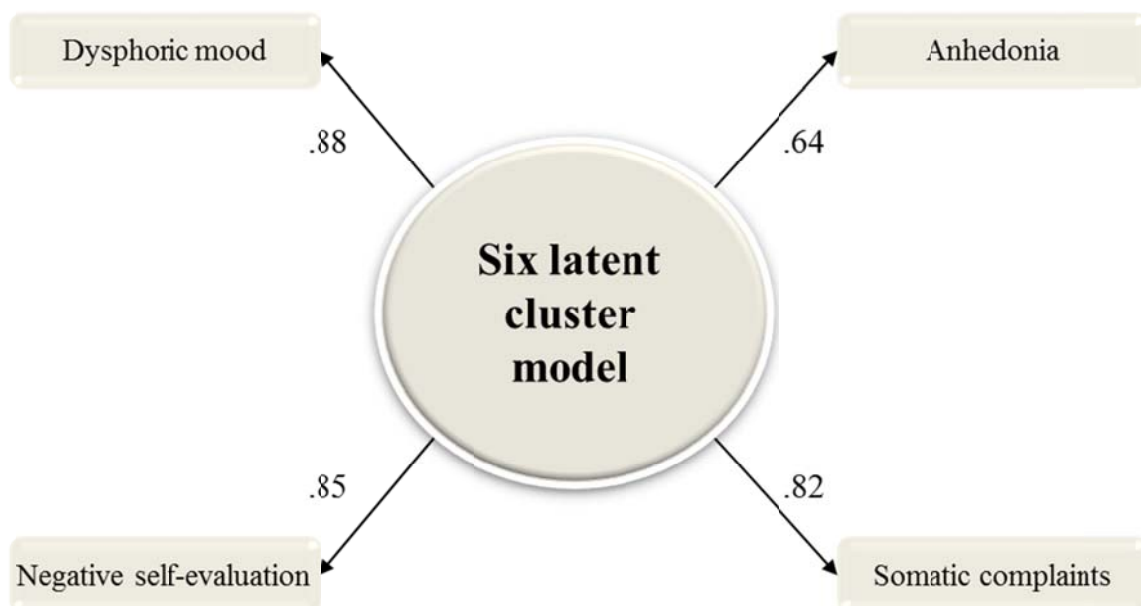


Figure 1. Path values for four RADS indicator variables.

Z scores for the four indicator variables were calculated using SPSS-16 program. Z scores were calculated so that nonstandardized raw scores across measures could be discussed and compared using a standardized measure. Z scores are graphed in Figure 2 to show the pattern of scores for each cluster.

In general, four clusters differed primarily on the severity of symptom endorsement, with each having relatively low scores on anhedonia (clusters 1, 2, 3, and 5). Two clusters showed high scores on anhedonia, with relatively lower scores on the remaining scales (clusters 4 and 6). Thus, anhedonia appears to covary differently from the other symptoms of depression. These clusters will be discussed in greater detail in the discussion section.

Descriptive data for the indicator variables and RADS total score for each cluster was calculated and is presented in Table 6. As expected, the six clusters differed on the

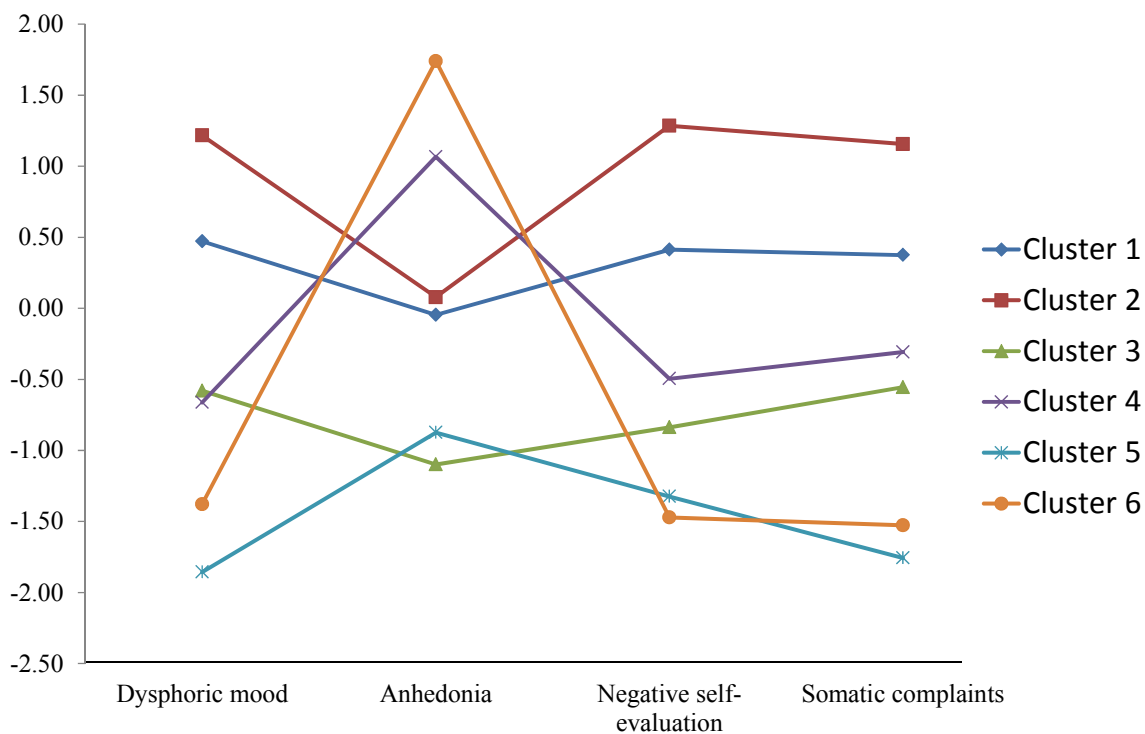


Figure 2. Z scores of four indicator variables across clusters.

indicator variables, as well as the RADS total score. The results of statistical comparisons including the  $F$  test,  $p$  values, effect size, and post hoc analyses are also displayed in the Table 6.

Table 7 provides a summary of the cluster characteristics. As previously indicated, symptom severity appears to be a critical determinant in cluster assignment. For convenience, each cluster was given a descriptive name. These descriptive names can also be seen in Table 7.

### Research Question #2(a)

The second research question asked how the latent subtypes of depression

Table 6

*Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS Only)*

Variable	Cluster 1 Moderately distressed/anxious		Cluster 2 Highly distressed/ anxious, moderately anhedonic (predominately female)		Cluster 3 Minimally distressed/anxious		Cluster 4 Minimally distressed/anxious, extremely anhedonic		Cluster 5 Extremely distressed/anxious, moderately anhedonic (almost exclusively female)		Cluster 6 Minimally distressed/anxious, moderately anhedonic (predominantly males)		<i>F</i> test	<i>p</i> value	Eta <sup>2</sup>
	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>			
Dysphoric mood	23.84	2.69	28.25	2.08	17.62 <sup>a</sup>	2.89	17.13 <sup>a</sup>	3.12	10.08	1.84	12.90	2.61	579.367	<.0001*	.802
Anhedonia	16.34 <sup>a</sup>	4.52	17.07 <sup>a</sup>	3.54	10.18 <sup>b</sup>	2.26	22.85	3.42	11.49 <sup>b</sup>	4.65	26.79	0.95	197.535	<.0001*	.580
Negative self- evaluation	22.00	3.45	27.61	2.43	13.96	3.86	16.15	3.88	10.82 <sup>a</sup>	2.57	9.88 <sup>a</sup>	1.90	400.776	<.0001*	.737
Somatic complaints	20.79	2.71	24.64	2.05	16.22	2.81	17.43	3.38	10.29 <sup>a</sup>	2.41	11.43 <sup>a</sup>	3.09	323.658	<.0001*	.693
RADS total	82.96	7.42	97.57	5.19	57.97 <sup>a</sup>	6.24	73.57	6.68	42.69	6.71	61.00 <sup>a</sup>	5.95	778.623	<.0001*	.845

<sup>a, b</sup> = There are NO significant statistical differences between clusters with the same superscripts ( $p > .05$ ).

\*  $p$  value is statistically significant at  $p < .05$ .

Table 7

*Cluster Characteristics Across Indicator Variables*

Cluster	Dysphoric mood	Anhedonis	Negative self-evaluation	Somatic complaints
Cluster 1 (highly distressed)	Moderate	Moderate	Moderate	Moderate
Cluster 2 (extremely distressed)	High	Moderate	High	High
Cluster 3 (moderately distressed)	Moderate	Minimal	Moderate	Moderate
Cluster 4 (moderately anhedonic)	Moderate	High	Moderate	Moderate
Cluster 5 (minimally distressed)	Minimal	Minimal	Minimal	Minimal
Cluster 6 (extremely anhedonic)	Minimal	High	Minimal	Minimal

identified by the LCA analyses relate to DSM clinical diagnoses. As outlined in the Methods section, individual diagnoses were collapsed into broad diagnostic categories. The created diagnostic groupings, as well as the specific diagnoses that they contain, were presented earlier in Table 2.

Out of the sample of adolescents, 684 were given a primary diagnosis, while 452 participants were given an additional second diagnosis, and lastly 115 of the participants were given a third diagnosis. In addition, 32 participants did not have a recorded diagnosis in the data set. Table 8 contains all diagnoses for all participants. For example, in a given column (cluster), each subject who had received multiple diagnoses would contribute to the percentage for each assigned diagnosis (whether primary, secondary, or tertiary). Therefore, if one participant had three diagnoses, all three diagnoses would be represented in this table. The frequency counts and percentages of each cluster within the diagnostic categories are presented in Table 8. To address if the clusters varied by diagnosis, a chi-square difference test was conducted for each diagnosis.

There was a statistically significant difference between clusters on the proportion

of the mood disorder, anxiety disorder, childhood disorder, and externalizing disorder diagnoses. This means that particular clusters had higher rates of these four diagnostic categories than other clusters. There was no difference between clusters on the proportion of psychosis, somatoform, substance, or eating disorder diagnoses.

To get a complete picture of symptom presentation among individuals, the previous analysis considered the multiple diagnoses of the participants. The use of multiple diagnoses for each participant captures the full range of symptomatology; however, it may also complicate the analyses and may be somewhat misleading since secondary and tertiary diagnoses are given equal weight as primary diagnoses. So, the analyses were repeated using only the primary diagnoses of participants. As previously stated, 684 participants had a primary diagnoses and chi-square analyses based on these diagnoses are presented in Table 9. The frequency counts and percentages of each cluster's primary diagnoses are also presented.

There was a statistically significant difference between clusters on the proportion of the mood disorder, anxiety disorder, substance disorder, childhood disorder, and externalizing disorder diagnoses as primary diagnoses. This means that particular clusters had higher rates of these four diagnostic categories than other clusters when looking at primary diagnoses only. There was no difference between clusters on the proportion of psychosis, somatoform, or eating disorder diagnoses as primary diagnoses. It should be noted that when looking only at primary diagnoses, there is a statistically significant difference in the proportion of substance disorder diagnoses between clusters. This difference was not observed when all diagnoses were considered for participants.

Table 8

*Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS Only; Combined Across All Diagnoses)*

Diagnosis	Cluster 1 (N = 298)		Cluster 2 (N = 118)		Cluster 3 (N = 116)		Cluster 4 (N = 97)		Cluster 5 (N = 51)		Cluster 6 (N = 42)		Total sample (N = 722)		Chi squared	p value
	%	n	%	n	%	n	%	n	%	n	%	n	%	n		
No diagnosis recorded	6.0	18	4.2	5	6.0	7	4.1	4	2.0	1	7.1	3	5.3	38	2.414	.789
Mood	86.2	257	91.5	108	74.1	86	86.6	84	72.5	37	76.2	32	83.7	604	21.416	.001*
Anxiety	13.1	39	28.0	33	16.4	19	17.5	17	5.9	3	9.5	4	15.9	115	19.898	.001*
Psychosis	00.0	0	00.8	1	1.7	2	1.0	1	0	0	0	0	00.6	4	5.646	.342
Somatoform	0	0	0	0	00.9	1	0	0	0	0	0	0	00.1	1	5.231	.388
Substance	12.4	37	11.0	13	8.6	10	11.3	11	11.8	6	11.9	5	11.4	82	1.229	.942
Eating	4.0	12	5.9	7	00.9	1	3.1	3	0.0	0	2.4	1	3.3	24	7.030	.218
Childhood disorder	15.4	46	5.9	7	14.7	17	22.7	22	29.4	15	31.0	13	16.6	120	25.169	<.0001*
Externalizing	21.8	65	13.6	16	25.0	29	35.1	34	35.3	18	31.0	13	24.2	175	18.922	.002*

*Note.* Cluster labels are as follows: Cluster 1 moderately distressed/anxious, Cluster 2 highly distressed/anxious, moderately anhedonic (predominantly female), Cluster 3 minimally distressed/anxious, Cluster 4 minimally distressed/anxious, extremely anhedonic, Cluster 5 extremely distressed/anxious, moderately anhedonic (almost exclusively female), Cluster 6 minimally distressed/anxious, moderately anhedonic (predominantly males).

In a given column (cluster), each subject who had received multiple diagnoses would contribute to the percentage for each assigned diagnosis (whether primary, secondary, or tertiary).

\*p value is statistically significant at  $p < .05$ .

Table 9

*Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS Only; Primary Diagnosis Only)*

Primary diagnosis	Cluster 1 (N = 298)		Cluster 2 (N = 118)		Cluster 3 (N = 116)		Cluster 4 (N = 97)		Cluster 5 (N = 51)		Cluster 6 (N = 42)		Total sample (N = 722)		Chi squared	p value
	%	n	%	n	%	n	%	n	%	n	%	n	%	n		
No diagnosis recorded	6.0	18	4.2	5	6.0	7	4.1	4	2.0	1	7.1	3	5.3	38	2.41	.789
Mood	83.6	249	88.1	104	66.4	77	80.4	78	56.9	29	73.8	31	78.7	568	36.21	<.0001*
Anxiety	2.3	7	6.8	8	11.2	13	3.1	3	5.9	3	7.1	3	5.1	37	15.45	.009*
Psychosis	0	0	0	0	1.7	2	1.0	1	0	0	0	0	.4	3	7.81	.167
Somatoform	0	0	0	0	.9	1	0	0	0	0	0	0	.1	1	5.23	.388
Substance	0	0	0	0	0	0	0	0	0	0	2.4	1	.1	1	16.21	.006*
Eating	.3	1	0	0	0	0	1.0	1	0	0	0	0	.3	2	2.94	.709
Childhood disorder	3.7	11	0	0	5.2	6	2.1	2	15.7	8	4.8	2	4.0	29	24.46	<.0001*
Externalizing	4.0	12	.8	1	8.6	10	8.2	8	19.6	10	4.8	2	6.0	43	29.94	<.0001*

*Note.* Cluster labels are as follows: Cluster 1 moderately distressed/anxious, Cluster 2 highly distressed/anxious, moderately anhedonic (predominantly female), Cluster 3 minimally distressed/anxious, Cluster 4 minimally distressed/anxious, extremely anhedonic, Cluster 5 extremely distressed/anxious, moderately anhedonic (almost exclusively female), Cluster 6 minimally distressed/anxious, moderately anhedonic (predominantly males).

\* p value is statistically significant at  $p < .05$ .



### Research Question #3(a)

The third research question asked how the participants in each latent class differ on age, gender, symptom severity, and other psychosocial variables. It was hypothesized that subtypes of adolescent depression would differ on a variety of psychosocial variables, specifically: sociability, impulse control, self-concept, peer security, and family rapport. These five psychosocial variables were derived from the participants' raw scores on these five subscales of the MAPI (Millon Adolescent Personality Inventory). Descriptive statistics for the sample on the five psychosocial variables are found in Table 10. It is noted that MAPI data was only available for 351 out of the 722 participants.

To address this research question one-way ANOVAs were conducted for each variable (age, sociability, impulse control, self-concept, peer-security, family rapport, and RADS total) by cluster. Follow-up post hoc tests were conducted if the ANOVA  $F$  test reached statistical significance. There were no statistically significant differences in age between the clusters. However, the six clusters did differ on symptom severity, as indicated by the RADS total score. Clusters 3 and 6 were statistically significantly higher in symptom severity than the other clusters, which did not differ from each other. There

Table 10

#### *Descriptive Statistics for Psychosocial Variables (N = 351)*

Variable	Mean	SD
Sociability	15.75	5.51
Impulse control	15.94	6.19
Self-concept	14.82	7.23
Peer security	8.77	5.22
Family rapport	9.12	5.16

were statistically significant differences across clusters for all five psychosocial variables. However, due to the unequal distribution of the individuals within each cluster who completed the MAPI (cluster 1 = 236, cluster 2 = 104, cluster 3 = 11, clusters 4, 5, and 6 = 0) post hoc analyses were unable to be completed due to statistical limitations. There were statistically significant gender differences between clusters. Cluster 1 and Cluster 2 were statistically and predominantly made up of females (66.1% and 88.1%, respectively), whereas Cluster 5 and Cluster 6 were statistically and predominantly male (66.7% and 73.8%). The remaining Clusters (3 and 4) were not statistically different in terms of gender. The means for age, symptom severity (RADS total scores), and psychosocial variables are displayed in Table 11, as well as the *F* test, *p* values, effect size, and results of post hoc analyses. The gender differences within clusters are displayed in Table 12.

### **Second Latent Cluster Analyses**

In the following pages, Research Questions 1 through 3 will be addressed utilizing LCA with the seven indicator variables from the RADS and RCMAS. This second LCA analysis will fully address each of the three research questions utilizing symptoms of depression and anxiety. To differentiate the first analysis (four RADS indicator variables) from the second analysis (which utilized four RADS indicator variables *and* three RCMAS indicator variables), the initial analyses will refer to Research Question # 1(a), #2(a), and #3(a), while the second set of analyses will be identified as Research Question #1(b), #2(b), and #3(b).

Table 11

*Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS Only)*

Variable	Cluster 1 Moderately distressed/anxious		Cluster 2 Highly distressed/ anxious, moderately anhedonic (predominately female)		Cluster 3 Minimally distressed/anxious		Cluster 4 Minimally distressed/anxious, extremely anhedonic		Cluster 5 Extremely distressed/anxious, moderately anhedonic (almost exclusively female)		Cluster 6 Minimally distressed/anxious, moderately anhedonic (predominantly males)		F test	p value	Eta <sup>2</sup>
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Age	14.99 <sup>a</sup>		15.14 <sup>a</sup>		15.00 <sup>a</sup>		14.99 <sup>a</sup>		14.75 <sup>a</sup>		14.95 <sup>a</sup>		0.62	.685	.004
Sociability	16.85	5.02	13.88	5.79	9.82	4.79							18.807	<.0001*	.098
Impulse control	14.87	6.27	18.06	5.54	18.91	4.37							11.507	<.0001*	.062
Self-concept	12.28	6.14	19.78	6.59	22.27	5.22							59.947	<.0001*	.256
Peer security	7.39	4.43	11.36	5.51	14.09	5.82							31.473	<.0001*	.153
Family rapport	8.16	5.20	11.01	4.52	11.82	4.17							13.514	<.0001*	.072
RADS total	82.96	7.42	97.57	5.19	57.97 <sup>a</sup>	6.24	73.57	6.68	42.69	6.71	61.00 <sup>a</sup>	5.95	778.623	<.0001*	.845

Note. Missing data in the table indicates that no participants within the cluster completed the MAPI.

<sup>a</sup> There are NO significant statistical differences between clusters with the same superscripts ( $p > .05$ ).

\*  $p$  value is statistically significant at  $p < .05$ .

Table 12

*Gender Differences Within Clusters (RADS Only)*

Cluster	Male		Female		Chi square	<i>p</i> value
	%	<i>n</i>	%	<i>n</i>		
Cluster 1 (highly distressed)	33.9	101	66.1	197	30.926	<.0001*
Cluster 2 (extremely distressed)	11.9	14	88.1	104	68.644	<.0001*
Cluster 3 (moderately distressed)	56.0	65	44.0	51	1.690	.194
Cluster 4 (moderately anhedonic)	46.4	45	53.6	52	.505	.477
Cluster 5 (minimally distressed)	66.7	34	33.3	17	5.667	.017*
Cluster 6 (extremely anhedonic)	73.8	31	26.2	11	9.524	.002*

**Research Question #1(b)**

The first research question asked whether there are latent subtypes of adolescent depression that can be identified from an in-patient sample. LCA was conducted to determine the underlying structure and potential latent class models of depression using the raw scores of the four subscales of the RADS and the three subscales of the RCMAS as indicator variables. The seven indicator variables were: (RADS) dysphoric mood, anhedonia/negative affect, negative self-evaluation, somatic complaints, and (RCMAS) physiological anxiety, worry/oversensitivity, social concerns/concentration. All variables were identified as continuous in the analysis. Two through eight cluster models were investigated and the BIC(LL) was used as the primary indicator of model fit. The BIC(LL) values for one through eight cluster solutions are presented numerically in Table 13 .

Table 13

*BIC(LL) for Cluster Models Based on RADS and RCMAS Indicator Variables*

Model solution	BIC(LL)
One-cluster model	28938.715
Two-cluster model	26603.677
Three-cluster model	26000.354
Four-cluster model	25794.757
Five-cluster model	25685.886
Six-cluster model	25622.284 <sup>a</sup>
Seven-cluster model	25645.714
Eight-cluster model	25513.971

<sup>a</sup>The six-solution model was the most statistically meaningful fit for the data.

A review of the BIC(LL) values indicates that there is a decrease at the six cluster solution model, with the BIC(LL) value beginning to increase at the seven cluster model. Therefore, initial review of the BIC(LL) values revealed that the six cluster model was the best solution. However, it was not clear if the six cluster solution was statistically significantly lower than the other cluster solutions. The six cluster model was deemed to be superior to the seven cluster model, as it had a lower BIC (LL) value and was more parsimonious. The fit of the six cluster model was empirically compared to models with fewer clusters. To evaluate which cluster solution provided the best fit to the data, a bootstrap -2LL difference test was conducted comparing the six cluster model to the five cluster model and the four cluster model. The six cluster model provided a statistically significantly better fit than both the 5 and 4 cluster models (-2LL Diff = 162.33,  $p < .0001$ ; -2LL Diff = 369.93,  $p < .0001$ , respectively). Therefore, the six cluster model was identified as the best solution and was the basis for answering the remaining research

questions.

The path values for the seven indicator variables are shown in Figure 3. The path values ranged from .64 to .87. All path values were statistically significant, and suggest that each variable significantly impacts cluster assignment. The variance accounted for by the predictors ranged from 41% to 69%.

Z scores for the seven indicator variables were calculated using SPSS-16 program. Z scores were calculated so that non-standardized raw scores across measures could be discussed and compared using a standardized measure. Z scores are graphed in Figure 4 to show the pattern of scores for each cluster.

In general, four clusters differed primarily on the severity of symptom endorsement, with each having comparatively low scores on anhedonia (Clusters 1, 2, 3, 5). Two clusters showed high scores on anhedonia, with relatively lower scores on the

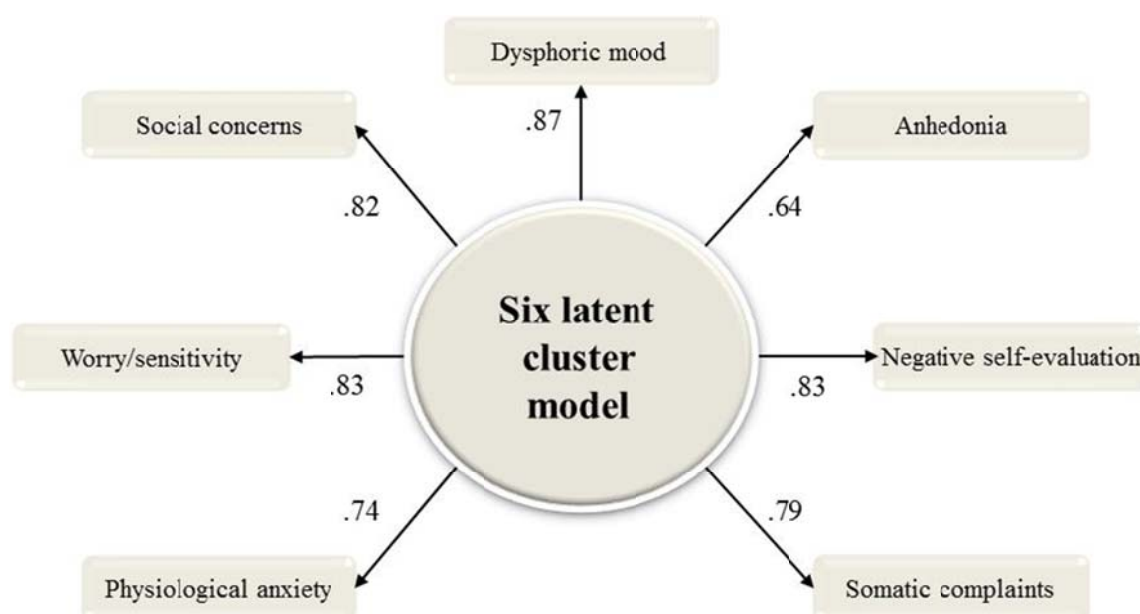


Figure 3. Path values for six cluster model with RADS and RCMAS indicator variables.

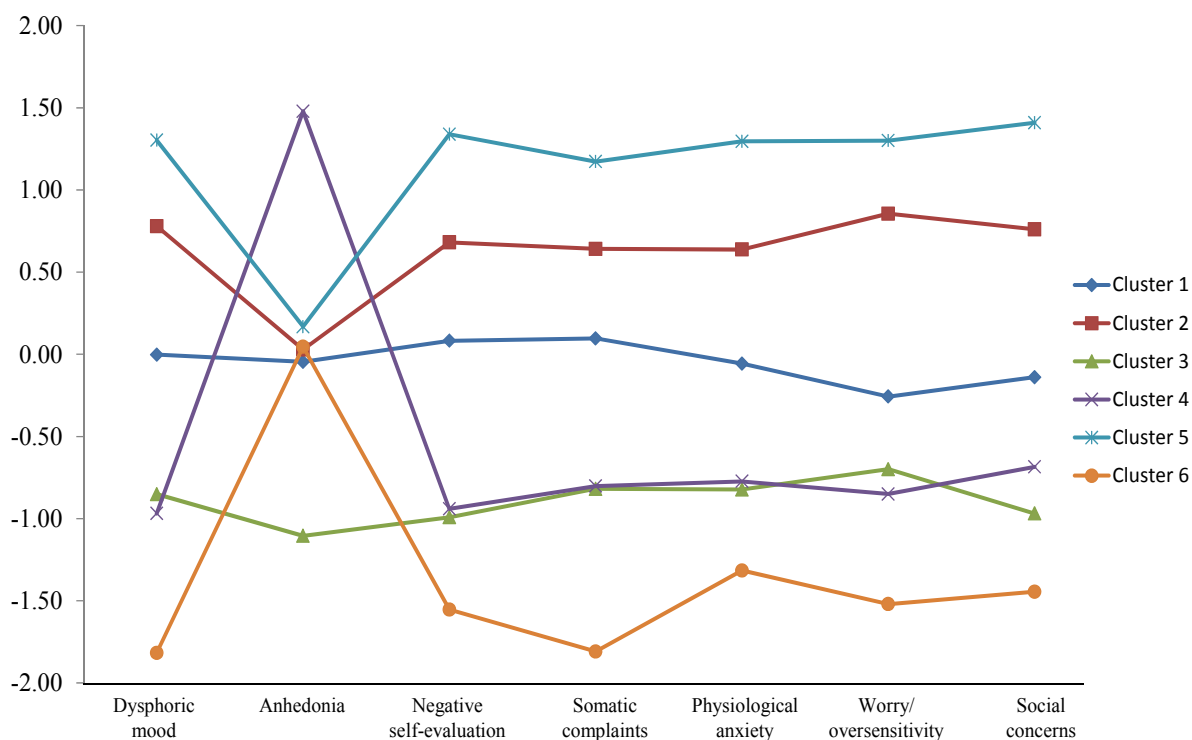


Figure 4. Z score of seven indicator variables across clusters.

remaining scales (Clusters 4 and 6). Thus, anhedonia appears to covary differently from the other symptoms of depression and anxiety. These clusters will be discussed in greater detail in the discussion section.

For this six cluster model with seven indicator variables, cluster assignment was unambiguous with 81.89% of the sample differing in probability of cluster assignment by at least 25% between the assigned cluster and the next most likely cluster; and over 96% of the sample differing in probability of cluster assignment by at least 50% between the assigned class and the next most likely cluster. Therefore, the likelihood of an individual being assigned membership to a specific cluster was distinct.

Descriptive data for the seven indicator variables, RADS total, and RCMAS total

scores for each cluster was calculated and is presented in Table 14. As expected, the six clusters differed on the indicator variables, as well as the RADS and RCMAS total scores. The results of statistical comparisons including the *F* test, *p* values, effect size, and post Hoc analyses are also displayed in Table 14.

Table 15 provides a summary of the cluster characteristics. As previously indicated, symptom severity appears to be a critical determinant in cluster assignment. For convenience, each cluster was given a descriptive name. These descriptive names can also be seen in Table 15.

### **Research Question #2(b)**

The second research question asked how the latent subtypes of depression identified by the LCA analyses relate to DSM clinical diagnoses. As outlined in the Methods section, individual diagnoses were collapsed into broad diagnostic categories. The created diagnostic groupings used, as well as the specific diagnoses that they contain, were presented previously in Table 2.

Table 16 contains all diagnoses for all participants. For example, if one participant had three diagnoses, all three diagnoses would be represented in this table. The frequency counts and percentages of each cluster within the diagnostic categories are presented in Table 16. To address if the clusters varied by diagnosis, a chi-square difference test was conducted for each cluster.

There was a statistically significant difference between clusters on the proportion of the mood disorder, childhood disorder, and externalizing disorder diagnoses. This means that particular clusters had higher rates of these three diagnostic categories than



Table 14

*Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS and RCMAS)*

Variable	Cluster 1 Moderately distressed/anxious		Cluster 2 Highly distressed/ anxious, moderately anhedonic (predominately female)		Cluster 3 Minimally distressed/anxious		Cluster 4 Minimally distressed/anxious, extremely anhedonic		Cluster 5 Extremely distressed/anxious, moderately anhedonic (almost exclusively female)		Cluster 6 Minimally distressed/anxious, moderately anhedonic (predominantly males)		F test	p value	Eta <sup>2</sup>
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Dysphoric mood	21.04	3.23	25.66	2.30	16.02 <sup>a</sup>	3.76	15.32 <sup>a</sup>	3.18	28.75	2.03	10.31	1.76	457.484	< .0001*	.762
Anhedonia	16.34 <sup>a</sup>	5.14	16.77 <sup>a</sup>	3.95	10.15	2.35	25.26	2.71	17.59 <sup>a</sup>	4.00	16.88 <sup>a</sup>	8.54	99.873	< .0001*	.411
Negative self- evaluation	19.88	3.95	23.72	3.68	12.96 <sup>a</sup>	3.19	13.30 <sup>a</sup>	3.70	27.96	2.48	9.35	1.71	343.075	< .0001*	.706
Somatic complaints	19.43	2.94	22.11	2.89	14.92 <sup>a</sup>	3.16	15.00 <sup>a</sup>	3.84	24.72	1.95	10.04	2.81	250.802	< .0001*	.637
RADS total	76.68	8.25	88.27	6.77	54.05	6.92	68.88	7.88	99.03	5.80	46.58	9.70	585.237	< .0001*	.803
Physiological anxiety	4.27	1.91	6.14	1.91	2.20 <sup>a</sup>	1.73	2.34 <sup>a</sup>	1.80	7.91	1.69	.88	.87	179.532	< .0001*	.556
Worry/oversensitivity	4.90	2.44	8.86	1.58	3.33 <sup>a</sup>	2.50	2.80 <sup>a</sup>	2.00	10.42	.65	.42	.61	323.155	< .0001*	.693
Social concerns/ concentration	3.34	1.47	5.32	1.17	1.51	1.24	2.14	1.44	6.75	.43	.46	.54	317.825	< .0001*	.689
RCMAS total	12.51	3.54	20.32	2.78	7.05 <sup>a</sup>	3.77	7.27 <sup>a</sup>	3.61	25.10	1.92	1.75	1.36	666.302	< .0001*	.823

<sup>a</sup> There are NO significant statistical differences between clusters with the same superscripts ( $p < .05$ ).

\*  $p$  value is statistically significant at  $p < .05$ .

Table 15

*Cluster Characteristics Across Indicator Variables*

Cluster	Dysphoric mood	Anhedonia	Negative self-evaluation	Somatic complaints	Physiological anxiety	Worry/oversensitivity	Social concerns
Cluster 1 (moderately distressed/anxious)	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate
Cluster 2 (highly distressed/anxious, moderately anhedonic—predominantly females)	High	Moderate	High	High	High	High	High
Cluster 3 (minimally distressed/ anxious)	Minimal	Minimal	Minimal	Minimal	Minimal	Minimal	Minimal
Cluster 4 (minimally distressed/ anxious, extremely anhedonic)	Minimal	Extreme	Minimal	Minimal	Minimal	Minimal	Minimal
Cluster 5 (extremely distressed/anxious, moderately anhedonic—almost exclusively females)	Extreme	Moderate	Extreme	Extreme	Extreme	Extreme	Extreme
Cluster 6 (minimally distressed/anxious, moderately anhedonic—predominantly males)	Minimal	Moderate	Minimal	Minimal	Minimal	Minimal	Minimal

Table 16

*Descriptive Statistics for Clusters and Total Sample within Diagnostic Categories (RADS and RCMAS; Combined Across All Diagnoses)*

Primary diagnosis	Cluster 1 (N = 216)		Cluster 2 (N = 207)		Cluster 3 (N = 108)		Cluster 4 (N = 74)		Cluster 5 (N = 69)		Cluster 6 (N = 48)		Total sample (N = 722)		Chi squared	p value
	%	n	%	n	%	n	%	n	%	n	%	n	%	n		
No diagnosis recorded	6.0	13	6.3	13	3.7	4	5.4	4	4.3	3	2.1	1	5.3	38	2.296	.807
Mood	88.4	191	84.5	175	73.1	79	77.0	57	92.8	64	79.2	38	83.7	604	19.698	.001*
Anxiety	12.5	27	18.8	39	13.9	15	16.2	12	26.1	18	8.3	4	15.9	115	10.932	.053
Psychosis	0.9	2	0.5	1	.9	1	0	0	0	0	0	0	0.6	4	1.896	.863
Somatoform	0	0	0	0	0.9	1	0	0	0	0	0	0	0.1	1	5.693	.337
Substance	13.4	29	11.6	24	6.5	7	10.8	8	10.1	7	14.6	7	11.4	82	4.099	.535
Eating	1.9	4	4.8	10	0.9	1	5.4	4	7.2	5	0	0	3.3	24	10.803	.055
Childhood disorder	16.2	35	14.5	30	19.4	21	27.0	20	2.9	2	25.0	12	16.6	120	18.915	.002*
Externalizing	26.9	58	17.4	36	29.6	32	40.5	30	10.1	7	25.0	12	24.2	175	25.986	<.0001*

*Note.* Cluster labels are as follows: Cluster 1 moderately distressed/anxious, Cluster 2 highly distressed/anxious, moderately anhedonic (predominantly female), Cluster 3 minimally distressed/anxious, Cluster 4 minimally distressed/anxious, extremely anhedonic, Cluster 5 extremely distressed/anxious, moderately anhedonic (almost exclusively female), Cluster 6 minimally distressed/anxious, moderately anhedonic (predominantly males).

In this chart, *N* denotes the number of diagnoses given. *N* will add up to greater than 722 due to multiple diagnoses per individual.

\**p* value is statistically significant at  $p < .05$ .

other clusters. There was no difference between clusters on the proportion of anxiety, psychosis, somatoform, substance, or eating disorder diagnoses.

To get a complete picture of symptom presentation among individuals, the previous analysis considered the multiple diagnoses of the participants. The use of multiple diagnoses for each participant captures the full range of symptomatology; however, it may also complicate the analyses. Therefore, the analyses were repeated using only the primary diagnoses of participants. As previously stated, 684 participants had a primary diagnoses and chi-square analyses based on these diagnoses are presented in Table 17. The frequency counts and percentages of each cluster's primary diagnoses are also presented.

There was a statistically significant difference between clusters on the proportion of the mood disorder, substance disorder, childhood disorder, and externalizing disorder diagnoses as primary diagnoses. This means that particular clusters had higher rates of these four diagnostic categories than other clusters when looking at primary diagnoses only. There was no difference between clusters on the proportion of anxiety, psychosis, somatoform, or eating disorder diagnoses as primary diagnoses. It should be noted that when looking only at primary diagnoses, there is a statistically significant difference in the proportion of substance disorder diagnoses between clusters. This difference between clusters disappears when all diagnoses are considered for participants.

### **Research Question #3(b)**

The third research question asked how the participants in each latent class differ on age, gender, symptom severity, and other psychosocial variables. It was hypothesized

Table 17

*Descriptive Statistics for Clusters and Total Sample Within Diagnostic Categories (RADS and RCMAS; Primary Diagnosis Only)*

Primary diagnosis	Cluster 1 (N = 216)		Cluster 2 (N = 207)		Cluster 3 (N = 108)		Cluster 4 (N = 74)		Cluster 5 (N = 69)		Cluster 6 (N = 48)		Total sample (N = 722)		Chi squared	p value
	%	n	%	n	%	n	%	n	%	n	%	n	%	n		
No diagnosis recorded	6.0	13	6.3	13	3.7	4	5.4	4	4.3	3	2.1	1	5.26	38	2.30	.807
Mood	82.4	178	82.6	171	64.8	70	73.0	54	88.4	61	70.8	34	78.67	568	23.15	<.0001*
Anxiety	3.2	7	3.4	7	9.3	10	6.8	5	7.2	5	6.3	3	5.12	37	7.84	.165
Psychosis	.9	2	0	0	.9	1	0	0	0	0	0	0	0.42	3	3.70	.593
Somatoform	0	0	0	0	.9	1	0	0	0	0	0	0	0.14	1	5.69	.337
Substance	0	0	0	0	0	0	0	0	0	0	2.1	1	0.14	1	14.06	.015*
Eating	0	0	.5	1	0	0	1.4	1	0	0	0	0	0.28	2	4.64	.462
Childhood disorder	2.3	5	3.4	7	7.4	8	5.4	4	0	0	10.4	5	4.02	29	13.42	.020*
Externalizing	5.1	11	3.9	8	13.0	14	8.1	6	0	0	8.3	4	5.96	43	16.84	.005*

*Note.* Cluster labels are as follows: Cluster 1 moderately distressed/anxious, Cluster 2 highly distressed/anxious, moderately anhedonic (predominantly female), Cluster 3 minimally distressed/anxious, Cluster 4 minimally distressed/anxious, extremely anhedonic, Cluster 5 extremely distressed/anxious, moderately anhedonic (almost exclusively female), Cluster 6 minimally distressed/anxious, moderately anhedonic (predominantly males).

\*p value is statistically significant at  $p < .05$ .

that subtypes of adolescent depression would differ on a variety of psychosocial variables, specifically: sociability, impulse control, self-concept, peer security, and family rapport. Data for these psychosocial variables were taken from the MAPI. It is noted that MAPI data was only available for 351 out of the 722 participants.

To address this research question one-way ANOVAs were conducted for each variable (age, sociability, impulse control, self-concept, peer-security, family rapport, RADS total, and RCMAS total). Follow-up post hoc tests were conducted if the ANOVA reached statistical significance. There were no statistically significant differences in age between the clusters. However, the six clusters did differ on symptom severity, as indicated by the RADS and RCMAS total scores. Based on the RADS, each cluster was significantly different from one another in symptom severity. The ranking of the clusters, from least severe to most severe is as follows: Cluster 6, Cluster 3, Cluster 4, Cluster 1, Cluster 2, and Cluster 5. Using the RCMAS, each cluster was significantly different from one another in symptom severity, with the exception of Clusters 3 and 4 (which were not statistically different from one another). The ranking of the clusters, from least severe to most severe is as follows: Cluster 6, then Clusters 3 and 4, then Cluster 1, then Cluster 2, and finally Cluster 5.

There were statistically significant differences across clusters for all five psychosocial variables. However, due to the unequal distribution of the individuals within each cluster who completed the MAPI (cluster 1= 108, cluster 2= 167, cluster 3= 11, cluster 5= 65, cluster 4 and 6 =0) post hoc analyses were unable to be completed due to statistical limitations.

There were statistically significant gender differences between clusters. Cluster 2 and Cluster 5 were statistically and predominantly made up of females (74.4% and 92.8% respectively), whereas Cluster 6 was statistically and predominantly male (66.7%). The remaining Clusters 1, 3, and 4 were not statistically different in terms of gender. The means for age, symptom severity, and psychosocial variables are displayed in Table 18 below, as well as the *F* test, *p* values, effect size, and results of post hoc analyses. The gender differences within clusters are displayed in Table 19.

Table 18

*Means, Standard Deviations, and ANOVA Statistics Across Clusters (RADS and RCMAS)*

Variable	Cluster 1 Moderately distressed/anxious		Cluster 2 Highly distressed/ anxious, moderately anhedonic (predominately female)		Cluster 3 Minimally distressed/anxious		Cluster 4 Minimally distressed/anxious, extremely anhedonic		Cluster 5 Extremely distressed/anxious, moderately anhedonic (almost exclusively female)		Cluster 6 Minimally distressed/anxious, moderately anhedonic (predominantly males)		F test	p value	Eta <sup>2</sup>
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Age	14.95 <sup>a</sup>	1.34	15.06 <sup>a</sup>	1.34	14.87 <sup>a</sup>	1.27	14.93 <sup>a</sup>	1.35	15.19 <sup>a</sup>	1.41	15.02 <sup>a</sup>	1.47	.646	.665	.004
Sociability	17.22	4.80	16.01	5.39	9.91	4.97			13.62	5.84			10.913	< .0001*	.086
Impulse control	14.65	5.97	15.90	6.44	19.00	4.41			17.69	45.64			4.321	.005*	.036
Self-concept	11.97	6.24	14.53	6.84	21.00	5.88			19.23	7.32			18.949	< .0001*	.141
Peer security	7.14	4.33	8.37	4.78	13.82	6.24			11.68	5.85			15.791	< .0001*	.120
Family rapport	7.86	5.15	9.41	5.35	11.18	4.49			10.09	4.35			3.771	< .011*	.032
RADS total	76.68	8.25	88.27	6.77	54.05	6.92	68.88	7.88	99.03	5.80	46.58	9.70	585.237	< .0001*	.803
RCMAS total	12.51	3.54	20.32	2.78	7.05 <sup>a</sup>	3.77	7.27 <sup>a</sup>	3.61	25.10	1.92	1.75	1.36	666.302	< .0001*	.823

Note. Missing data in the table indicates that no participants within the cluster completed the MAPI.

<sup>a</sup> There are NO significant statistical differences between clusters with the same superscripts ( $p < .05$ ).

\*  $p$  value is statistically significant at  $p < .05$ .



Table 19

*Gender Differences Within Clusters (RADS and RCMAS)*

Cluster	Male		Female		Chi square	<i>p</i> value
	%	<i>n</i>	%	<i>n</i>		
Cluster 1 (moderately distressed/anxious)	44.4	96	55.6	120	2.667	.102
Cluster 2 (highly distressed/anxious, moderately anhedonic—predominantly female)	25.6	53	74.4	154	49.280	< .0001*
Cluster 3 (minimally distressed/anxious)	59.3	64	40.7	44	3.704	.054
Cluster 4 (minimally distressed/anxious, extremely anhedonic)	54.1	40	45.9	34	.486	.485
Cluster 5 (extremely distressed/anxious, moderately anhedonic—almost exclusively female)	7.2	5	92.8	64	50.449	< .0001*
Cluster 6 (minimally distressed/anxious, moderately anhedonic—predominantly males)	66.7	32	33.3	16	5.333	.021*

\* *p* value is statistically significant at  $p < .05$ .

## CHAPTER V

## DISCUSSION

The incidence of depression significantly increases from adolescence into early adulthood. Prospective epidemiological studies support that adolescents with MDD are at a two to four times greater risk for depression later in early adulthood (Pine et al., 1999). Depressive symptoms such as sadness, psychomotor retardation, guilt, anhedonia, low mood, suicidality, low energy, and reduced motivation combine to form a valid, well recognized, and distinct disorder and yet may not meet criterion threshold for diagnosis. A large proportion of adolescents with subclinical depressive features have the potential for later clinical episodes of major depression (Horwath, Johnson, Klerman, & Weissman, 2007). In addition, adolescents who meet diagnostic criteria for major depressive disorder experience distressing, but subclinical, levels of anxiety and vice versa (Ferdinand et al., 2005).

Past studies have reported clusters of individuals with significant distress and impairment (e.g., distress, negative affect) but who do not meet diagnostic criteria for either depression or anxiety. The notion of a mixed anxiety-depressive disorder (MADD) as a provisional diagnosis exists in ICD-10 and DSM-IV (APA, 1994; WHO, 2009). This provisional diagnosis suggests that the presence of both sub-threshold depressive and anxiety symptoms encompass its own unique construct associated with significant psychological distress. Clearly, the research shows that subthreshold symptoms of anxiety and depression have important implications for functioning. However, the use of a categorical classification system found in the DSM-IV may disregard the importance of

these subclinical features but important in their impact upon functioning. In addition, use of strict diagnostic categories can result in valuable information being lost because those who score just below clinical thresholds are regarded as non-cases even in light of significant problems.

The current research utilized LCA twice; once, including four indicator variables for depression from the RADS, and next including the same four indicator variables for depression with an additional three indicator variables for anxiety from the RCMAAS. Interestingly, results from both analyses were highly congruent. Both LCA analyses yielded six distinct subtypes of the sampled population. Also, both LCA analyses groups differed primarily on the overall severity of the majority of indicator variables. The exception was Anhedonia which did not covary with the other symptoms.

In this discussion, we first discuss the current research project and the “typical” individual in each cluster, further clarifying and discussing how the identified clusters differ from each other, and how this may be meaningful. Second, the current research will be discussed in the context of the existing research literature, discussing existing models of depression as well as the current conceptual framework of adolescent depression. Next, the discussion will address the broad consideration of dimensional vs. categorical model of diagnosis, and the meaningfulness of the current research to that topic. Finally, limitations and future directions for research will be addressed.

### **Description of Clusters**

Latent subtypes of adolescent depression, and depression and anxiety were

identified in an adolescent in-patient sample. The “typical” individual in each cluster will be presented followed by a discussion outlining how the clusters differed on diagnosis, age, gender, and symptom severity.

### Typical Individual in Each Cluster

Based on the mean values of the indicator variables and demographic composition, a description of a “typical” individual is presented in Table 20.

Table 20

#### *Typical Individual Per Cluster*

Cluster	Description
Cluster 1	Male or female, 15 years old, with <b>moderate</b> symptoms of distress (depression and anxiety) as well as <b>moderate Anhedonia</b> , diagnosed with a Mood Disorder  *Above clinical cutoff for RADS total score
Cluster 2	Female, 15 years old, with <b>high</b> symptoms of distress (depression and anxiety) and <b>moderate Anhedonia</b> , diagnosed with a Mood Disorder  *Above clinical cutoff for RADS total and RCMAS total scores
Cluster 3	Male or Female, 15 years old, with <b>minimal</b> symptoms of distress (depression and anxiety) and <b>minimal Anhedonia</b> , diagnosed with a Mood Disorder
Cluster 4	Male or Female, 15 years old, with <b>minimal</b> symptoms of distress (depression and anxiety) and <b>high Anhedonia</b> , with a Mood Disorder, highest likelihood of additional diagnosis of Childhood Disorder (ADHD, Tourette’s Syndrome, Enuresis) and Externalizing Disorder (ODD, Conduct Disorder, Intermittent Explosive Disorder, Disruptive Behavior Disorder NOS)
Cluster 5	Female, 15 years old, with <b>extreme</b> symptoms of distress (depression and anxiety) but <b>moderate Anhedonia</b> , diagnosed with a Mood Disorder, lowest likelihood of additional diagnosis of Childhood Disorder (ADHD, Tourette’s Syndrome, Enuresis) and Externalizing Disorder (ODD, Conduct Disorder, Intermittent Explosive Disorder, Disruptive Behavior Disorder NOS)  *Above clinical cutoff for RADS total and RCMAS total scores
Cluster 6	Male, 15 years old, with <b>minimal</b> symptoms of distress (depression and anxiety) and <b>moderate Anhedonia</b> , diagnosed with a Mood Disorder

### **Diagnostic Variability**

There is some diagnostic variability between the six clusters. There was a statistically significant difference between clusters on the proportion of the mood disorder, childhood disorder, and externalizing disorder diagnoses.

First, looking closely only at the distribution of the mood disorders within this research population, there was a statistically significant difference between clusters. However, the preponderance of individuals in all clusters who were diagnosed with mood disorders with percentages ranging from 73-92%. Thus, the finding may have limited clinical relevance in an in-patient population. However, the distribution of childhood disorders (ADHD, Tourette's, and Enuresis) between clusters shows more variability. At least one quarter of the adolescents in both cluster four and cluster six have a childhood disorder diagnosis. Additionally, almost no adolescents in cluster five (3%) had a childhood disorder diagnosis. Therefore, adolescents with particular subtypes of depression are more likely than others to present with an additional childhood disorders diagnosis.

Similarly, the distribution of externalizing disorders (ODD, conduct disorder, intermittent explosive disorder, and disruptive behavior disorder NOS) between clusters may also be clinically meaningful. Over 40% of adolescents in cluster four had an externalizing disorder whereas only 10% of adolescents in cluster five had such a disorder. Therefore, externalizing disorders may also hold clinical relevance when discussing differences between clusters. It is likely that Cluster 4 has a higher level of acting out, aggressive, and agitated behaviors; this depressive presentation may have elicited more intensive reactions from the adolescents' environments (family, school,

police, medical personnel) resulting in hospitalization. It is also possible that for adolescents with such intense externalizing behaviors, depression is a potential resulting condition that results from their comorbid externalizing disorder.

It appears from the current research that there exists a substantial amount of comorbidity of symptoms of anxiety and depression. However, the comorbidity *at the level of diagnosis* is not reflected in the sample; only 15.9% of the total sample of adolescents was diagnosed with an anxiety disorder. Theoretically, adolescents with high levels of anxious and depressive symptoms would have diagnoses of both a depressive and anxiety disorders. However, that does not seem to be the case and may be due to the relatively low discriminant validity of self-report measures. This will be further discussed in the Limitations section.

### **Age**

Participants within each latent class do not differ on age. Looking closely at age, the six clusters are remarkably similar in their mean age, despite an age range within this research sample (ages 12-18). Each of the six identified clusters maintained a mean age extremely close to the mean age of the overall sample at 14.99 years old and there were no statistically significant differences between any clusters on the variable of age. Perhaps age is not a critical determinant for the subtype of depression within adolescence.

### **Gender**

There were interesting and significant findings between the six clusters on gender. Cluster 6 was predominantly male while Cluster 2 and 5 were predominantly female

(74.4 and 92.8%, respectively). These two predominantly female clusters revealed profiles with the highest levels of distress among all subgroups; they were labeled as “extreme distress” and “high distress.” There was one notable distinction among these predominantly female clusters with elevated distress; both cluster profiles presented a marked dip in their measure of anhedonia. The cause or meaning of this relative decline in anhedonia on clusters 2 and 5 of predominantly female patients leaves room for debate. Possibly, the etiology of adolescent depression and anxiety among young females interact in such a way as for them to experience elevated levels of distress (high depression and anxiety) while maintaining some relative capacity to enjoy positive experiences. It is possible this capacity to enjoy positive experiences may parallel the higher incidence of hypomania in women compared to men. This theory is congruent with the evidence that hypomanic episodes, as well as Bipolar II Disorder, are more common in women than in men (APA, 2000).

Contrasting this dip in relative anhedonia among predominantly female clusters 2 and 5, was an equally prominent rise in anhedonia on Cluster 6. Cluster 6, made up predominately of males (66.7%), displays a sharp relative rise on measures of anhedonia compared to all other symptoms of anxiety and depression. This predominantly male cluster had the lowest level of distress among all six clusters, but revealed the greatest difference among scores of anhedonia. Again, the meaning of this spike in cluster 6 made up of predominantly male patients speaks to a group of individuals who report relative low levels of distress with contrasting high levels of anhedonia. It should be noted that this predominantly male Cluster 6 (with high levels of anhedonia) also contained

significantly higher proportion of individuals with externalizing disorder diagnoses (ODD, conduct disorder, antisocial personality disorder, intermittent explosive disorder, and disruptive behavior disorder NOS). These adolescents will likely present with problematic externalizing behaviors initially; upon closer examination, they may reveal high levels of anhedonia. Adolescents with this symptom picture are less likely to demonstrate some of the typical symptoms of depression (e.g., depressed mood, decreased energy). This is consistent with higher prevalence rates of externalizing disorders among juvenile males. Possibly, in efforts to experience some measure of physiological or psychological arousal due to extreme anhedonia, this population of young males may engage in problematic behaviors.

The remaining three clusters had equal gender distributions. Cluster 4, composed of almost equal proportions of males and females, displayed the highest level of anhedonia while maintaining the second lowest level of all other symptoms of distress. Two clusters with statistically equal gender distribution (Cluster 1 and Cluster 3) maintained levels of anhedonia that were on par with the severity of other symptoms. Therefore, gender is not a clear determinant of either the severity of distress or the congruence or incongruence of anhedonia severity with distress severity. Some patterns are relatively unique to females (much higher distress than anhedonia), and some patterns are relatively unique to males (much higher anhedonia with distress, paired with externalizing problems). However, three clusters had equal gender distribution, suggesting that other patterns of depression (equal anhedonia and distress; higher anhedonia than distress without externalizing problems) are applicable to both male and



female adolescents.

### **Symptom Severity**

Overall, the findings reveal that adolescent depression and anxiety co-occur in a remarkably consistent manner. Again, the general difference between the six identified subtypes is the overall intensity level of depressive and anxious features. Rather than a dichotomous presentation of either depressive or anxious symptomology, the results reveal a pattern of patient responses that display increasing intensity on all levels of both depression and anxiety. For example, there was no subgroup identified that had high levels of depressive features with low levels of anxious features. It appears, with the exception of anhedonia, that depressive symptomology mirrors anxious symptomology and vice versa. Within this inpatient sample, anxiety and depression occurred at almost identical levels within each subgroup and could be considered as part of the same psychological construct. For the purposes of this discussion, this pattern of symptom presentation (made up of dysphoric mood, negative self-evaluation, somatic complaints, physiological anxiety, worry/oversensitivity, social concerns) has been referred to as “Distress.” Anhedonia appears to be a unique symptom that does not consistently vary with the other six indicator variables. However, anhedonia may be considered a unique feature of certain subtypes of adolescent depression that offers additional information about that particular subtype of depression. This finding relates interestingly to the tripartite model of depression (Clark & Watson, 1991). This will be discussed further in a future section.

Symptom severity appears to be a critical determinant in cluster assignment. The

way in which six of the seven variables (dysphoric mood, negative self- evaluation, somatic complaints, physiological anxiety, worry/sensitivity, and social concerns) “hung” together with varying levels of symptom severity was remarkable. Ultimately, the clusters were labeled on the basis of severity alone, with the exception of the unique feature of anhedonia. The clusters have been labeled accordingly in Table 21. It is noted that these descriptive labels are relative terms and give a relative rank order of severity in comparison to one another. Further, only Clusters 1, 2, and 5 have clinical levels of depression according to the RADS Total score.

Looking at these descriptive labels, a conceptual formula to determine class membership has emerged. The conceptual formula is: adolescent distress = anxiety and depression (Level X) + anhedonia (Level Y). Simply knowing the level of severity of anxiety/depression symptoms and the level of anhedonia, one could determine cluster membership. Further, as six symptoms of anxiety and depression (with the exception of anhedonia) were so consistent with each other, one could presumably only evaluate any

Table 21

*Cluster Severity Labels*

Cluster	Description
Cluster 1	Moderately distressed anxious
Cluster 2	Highly distressed/anxious, moderately anhedonic (predominantly females)
Cluster 3	Minimally distressed/anxious
Cluster 4	Minimally distressed/anxious, extremely anhedonic
Cluster 5	Extremely distressed/anxious, moderately anhedonic (predominantly female)
Cluster 6	Minimally distressed/anxious, moderately anhedonic (predominantly males)

*one* symptom of either anxiety and depression *and* anhedonia to determine cluster membership.

The patients within this study population received an abundance of psychological measurements across the initial days of their in-patient hospitalization stay, with professional clinical evaluations as well. However, clinically significant anxiety symptoms suggested in the self-report data were not broadly represented in clinical diagnoses assigned. Clusters 2 and 5 had the highest levels of total anxiety on the RCMAS with scores above the recommended clinical cut off. However, the individuals in Cluster 2 and Cluster 5 were still only diagnosed with an Anxiety Disorder at approximately 19% and 26%, respectively. This may suggest that adolescent distress is a complex and multi-faceted construct not easily captured by self-report measures. Alternatively, the anxious symptoms may not have met criterion for a categorical disorder and, in fact, individuals are not typically hospitalized for anxiety disorders alone except in extreme cases. What is notable about the current research findings is the lack of variability in severity between symptoms of two disorders that are currently considered different diagnostic categories. These findings highlight the well-known limitations in the discriminant validity of self-report measures of anxiety and depression. Further, these findings highlight the potential for diagnostic confusion in the presence of clinical levels of multiple symptoms. Mounting empirical evidence suggests that depression, rather than different in *type*, is more likely different in *degree* when compared to the notion of “normal” (Coyne, 1994; Flett et al., 1997; Ruscio & Ruscio, 2000, 2002a, 2002b, 2004a, 2004b, 2004c).

Again, the present findings link back to previous studies that found that

depression and anxiety among juveniles were remarkably comorbid. The co-occurrence of depression and anxiety within this in-patient sample suggests some level of a mixed diagnostic entity. Perhaps symptom severity, as well as the presence or lack of anhedonia, yields the most valuable information about adolescent distress rather than, or perhaps along with, the clinical diagnosis.

### **In the Context of Existing Literature**

These current research findings are consistent with past research findings. Further, by investigating a sample of in-patient, adolescents with clinical diagnoses of depression and anxiety we expand the knowledge base of these classes of psychological problems. Eaton and colleagues (1989) used LCA to identify three discrete classes of depressive and anxiety symptoms in young adults; a large class of individuals with no problems, an “anxiety” class with characteristics of MDD, and an “MDD” class with characteristics of anxiety disorders. No distinct classes without comorbid anxiety and depression were identified. Often, in making diagnoses a categorical decision is made without inclusion of the range of severity of impairment, which appears to be particularly important in the distinction between the clusters found in the current research. Hudziak and colleagues (1998) demonstrated that other common childhood problems such as inattention, hyperactivity, and impulsivity also tended to collect into a range of severity clusters rather than simple categorical affected and unaffected cases. As previously stated, symptom severity is may be more critical than symptom type in distinguishing between distressed adolescents.

The finding that six subtypes of adolescent depression and anxiety emerged

compares favorably with preliminary research on latent subtypes of depression. In Das-Munshi et al. (2008), the authors reported finding five latent subtypes made up of mixed depression and anxiety of clusters; while Chen, Eaton, Gallo, Nestadt, and Crum (2000) revealed four subtypes of depression; and Wadsworth and colleagues (2001) revealed three subtypes of mixed depression and anxiety without pure types of either depression or anxiety. It is possible that the variability in number of clusters identified in these previous studies is due to sample size and statistical power. It is also possible that previous studies have found various numbers of subtypes of depression due to the population from which their samples were drawn, and the potential differences between populations (e.g., inpatient hospital sample, outpatient sample, school setting). These parallel findings suggest that there are multiple underlying subtypes of depressed and anxious children and adolescents rather than only one or two distinct diagnoses or categories.

Similar to past research, our findings suggest a consistent comorbidity of depression and anxiety specific to an adolescent population (Costello et al., 2003; Karlsson et al., 2000). This finding is notable in light of some past research that suggests that depressive disorders in children and adolescents are nearly always preceded by symptoms of anxiety (Goodwin et al., 2004; Pine et al., 2001). It is possible that depression and anxiety could be seen as part a larger construct that initially emerges with anxiety as a stepping stone to later depression. This is in contrast with the idea that depression and anxiety are separate but co-occurring constructs. With the consistent comorbidity across clusters, results from the present study supports that at least a portion of distressed adolescents experience clinical levels of both depression and anxiety that

warrant treatment intervention. While the current research cannot speak to the longitudinal course of these co-occurring symptoms, it highlights the comorbidity of symptomology and leaves the question of onset to future research.

The notion of global distress or negative affect is consistent with Clark and Watson's (1991) tripartite model of depression and anxiety that has received considerable scientific and clinical attention. The tripartite model advances that there are three main components: (a) general negative affect (NA; nonspecific factor of depression and anxiety), (b) anhedonia or low positive affect (specific to depression), and (c) physiological hyper-arousal (PH; specific to anxiety). The tripartite model is in line with findings from the current study; distress exists, and is similar in intensity for both anxiety and depression; however, anhedonia is unique and does not covary as other depressive and anxious symptoms do with each other. The current research adds to our understanding of the tripartite model in that anhedonia does not vary in intensity with distress as would be expected. While high levels of distress indicate high levels of depression and anxiety, high anhedonia does not necessarily occur with high distress and vice versa.

In light of these findings, the taxonomic structure of separate or pure cases of juvenile mood and anxiety disorders may be debatable. Clearly, the case for a construct we might label as negative affect or distress made up of both depressive and anxious features seems to have gained traction in the research literature (Watson, 2003). Viewing depression and anxiety as a single problem with different co-occurring features offers alternative treatment considerations. It is possible that combining treatment components

for both depression and anxiety may be effective for many distressed adolescents. Additionally, if we assume, as the literature suggests, that childhood anxiety heralds later adolescent depression, are there treatments considerations that might reduce this effect? What is not clear, from the current research, is the amount of depression found in adolescents with an anxiety disorder diagnosis alone. While the findings suggest that anxiety is likely to co-occur with clinical levels of depression, it is unclear whether the reverse is true.

### **Categorical Versus Dimensional Diagnostic Models**

The comorbidity among disorders challenges both how we assess and treat many classes of mental health disorders. Comorbidity is widely recognized to be a pervasive problem throughout the Diagnostic and Statistical Manual (Clark et al., 1995; Widiger & Clark, 2000). Given that comorbidity appears to be the rule rather than the exception, treatments that focus on single diagnostic constructs are called into question (e.g., Biederman et al., 1995; Keller et al., 1998; Rohde et al., 1991). There clearly exists an ongoing debate that depression and anxiety among children and adolescents should be considered a single taxonomic/diagnostic entity (Flannery-Shroeder, 2006).

Our findings are consistent with those of many other researchers focusing on the child and adolescent depression and anxiety who have focused on the “the comorbidity problem” within DSM-IV. While there are numerous RCT findings that support targeted treatments for depression and anxiety separately, there are few if any treatments that target both depression and anxiety as parts of a larger construct that include both aspects.

A focus on the combined presentation of depressive and anxious features may impart greater treatment success compared to a unitary model of childhood and adolescent problems. Our findings may contribute to the taxonomic constructs regarding the nature of anxiety and affective disorders in children and adolescents. Quantitative approaches such as LCA can offer added perspectives beyond categorical constructs (Gould, Bird, & Jaramillo et al., 1993).

In addition, current findings did not indicate grouping into simple affected and unaffected classes—a finding that would have supported a categorical approach to a mixed anxiety/depressive disorder. Rather, our research sample of inpatient adolescents formed six latent classes, supportive of a continuous distribution of problems spanning a combined anxiety and depression construct. This continuous distribution resembled the distribution found in another LCA study of attention problems (Hudziak et al., 1998). Our findings suggest the presence of a continuum of symptoms (severity rating—e.g., high, medium, low distress) made up of both affective and anxiety problems. While pure cases of either depression or anxiety exist, with individuals falling at the extreme opposite end of the continuum, most individuals appear to fall in the middle of the continuum of problems made up of both depression and anxiety problems.

A dimensional system rather than a simple categorical approach (presence versus absence of symptoms) would allow for greater communication regarding the severity of dysfunction. As stated earlier, severity is a significant predictor of a wide-range of clinical presentations, including both comorbidity and the course and chronicity of disorder (Clark et al., 1995). Researchers have found that continuous (dimensional)



scores are more stable over time, with higher levels of reliability than dichotomous (presence versus absence) measures; dimensional scores are generally unaffected by minor shifts in psychopathology (Widiger & Clark, 2000). In contrast, even a small change in total symptom count can move an individual above or below a dichotomous threshold. This is evident in a case where an individual with only four symptoms of depression would not meet the threshold for diagnosis while a single additional symptom would meet criteria. The difference between a clinical diagnosis and noncase by a single symptom would not be equivalent to lack of psychopathology. In a dimensional system this lower-level psychopathology would be captured and valuable information would be retained.

### **Clinical Implications**

Our findings support that rather than “pure” cases of depression and or anxiety there appears to be a unique clinical presentation we have termed “distress” made up of both depression and anxiety. While on the surface this may appear a matter of simple semantics our findings suggest otherwise. We have described juvenile distress as a more complex interplay and expression of negative mood and anxiety. While many aspects of juvenile psychological problems are similar to adult experiences there may be developmental distinctions and limitations that make childhood and adolescent problems different and worthy of clinical attention.

From a treatment perspective, knowing the various clinical presentations or cluster types one might encounter are important. Given that our findings revealed six ascending levels of distress the implications from a clinical perspective are worthy of

note when conceptualizing juvenile clients and working towards treatment goals as well as their capacity to move towards targeted goals.

For example, a clinician might approach treatment of male and female adolescents in a different manner given the uneven gender distribution among some of the clusters within our findings. Individuals with the highest distress were female while those with the least distress were males. From our findings, looking at the extremes of low and high distress levels in an in-patient population, young females may present as overtly distressed while males may contrastingly present as relatively free from distress. However, males may present with more externalizing problems and with relative high rates of anhedonia. Thus, when assessing for depression in males, specifically addressing anhedonia may be particularly important as these individuals may not endorse many of the other typical depressive symptoms. Alternatively, depressed adolescent females are likely to retain the ability to enjoy pleasurable activities despite significant distress. This may mean that behavioral activation will be a particularly important intervention for adolescent males.

The bulk of this research population was given a diagnosis of depression while very few were recognized as being anxious (although many endorsed clinical significant symptoms of anxiety). Realistically, a hospitalized patient's depressive features may predominate the clinical presentation, and potentially be the motivation for the hospital admission. Given the wide array of problems that may be responsible for an individual who are hospitalized, aspects of anxiety may be given less clinical importance in an in-patient setting. However, anxious symptoms may be present and particularly important

once adolescents are discharged from the hospital and the more immediate symptoms have been addressed. In addition, our findings suggest that depressive and anxious factors occur at almost equal rates and further highlight the need to evaluate and treat both aspects of patient distress.

### **Limitations**

Like all research, the current study contains limitations. Since the bulk of data were collected during acute in-patient hospitalization stays, that limits the generalizability to other in-patient samples and may not be replicated in an out-patient population. As this was not a longitudinal study, there are no data regarding changing patterns of adolescent distress over time. Longitudinal data would add much needed clarity to symptom presentation, cluster membership, and diagnosis over time compared to a single data point. Additionally, the data were collected over an approximate ten year span from the early to late 1990s and the psychological assessments utilized in data collection have since been updated. While true for the majority of studies with long data collection periods, data collection may have varied and standardization compromised.

In the last ten to twenty years, the constructs, assumptions, and clinical practices have undoubtedly evolved with additional changes to the DSM. Of note is the increasing role of technologies (internet, Facebook, cellphones, tweeting, texting, and similar positive and negative access to mass communication); the growing pace and availability of information has certainly influenced juvenile problems. Consequently, rapid and ever-changing vagaries of the culture which children and adolescents experience and influence

may be currently very different compared to when the original data was collected and warrants at least recognition that many things have significantly changed.

Perhaps the most significant limitation of the current study is the reliance on self-report measures exclusively. As already discussed, the self-report data did not consistently mirror the diagnoses given to the adolescents. Also as previously mentioned, self-report have minimal discriminant validity when used to distinguish anxiety and depressive symptoms.

However, the limitations of the current study are balanced by significant strengths of the design. This was a study that utilized a large sample size ( $N = 722$ ) and several commonly used measures were employed. Further, the sample was relatively homogenous regarding age, which may be critical when speaking about juveniles. Additionally, this study was balanced for gender and utilized a clinical population (adolescent psychiatric inpatients). In sum, the limitations of the current research are balanced by significant strengths that results in a meaningful contribution to existing literature.

### **Future Directions for Research**

Clearly, pure cases of depressive and anxiety disorders such as bipolar, obsessive compulsive disorder and specific phobias exist; however, the consistent research findings sustain that discrete or pure cases are the exception and not the rule. Rather, the research involving children and adolescents supports the notion that mixed/overlap/comorbidity of cases are more than typical (Eaton et al., 1989; Ryan et al., 1987; Sullivan, Neale, &

Kendler, 2000). However, others maintain that there is still insufficient data to support the notion that depression and anxiety are similar overlapping constructs (Kovacs & Devlin, 1998; Murphy, Marelich, & Hoffman, 2000; Muirs, Schmidt, Merckelbach, & Schouten, 2001). Future research focused on individuals diagnosed with both depression and anxiety, as well as the treatment of these individuals may lead to the development of treatments that are effective with the comorbid symptom picture commonly seen. Results from this current research have key implications for the classification of affective and anxiety disorders. Assessment of both comorbidity and severity of symptoms appears to be essential for an adequate clinical evaluation from a theoretical framework and from a practical clinical treatment perspective. Furthermore, these findings reinforce earlier work on child and adolescent populations demonstrating the validity of subthreshold consideration of depression and anxiety. Finally, we are at least raising the possibility that anxiety and depression may be conceptually useful if we were to consider them as a dimensional construct rather than from a strict categorical model. At the least, a semi-dimensional approach to the taxonomy of depression and anxiety reflects more accurately the clinical severity and course of these disorders. When taken in light with similar research, our current findings stress the importance of augmenting the diagnostic thresholds for anxiety and depression; while concurrently assessing severity as an essential component of the taxonomy of both disorders. To help resolve this discussion, continued research focus on the developmental ontogeny of anxiety and depression will require even more refined analysis of the existing data.

## REFERENCES

- Achenbach, T. M. (1991a). *Manual for the Child Behavior Checklist Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M. (1991b). *Manual for the Youth Adult Self-Report and Young Adult Behavior Checklist*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M., & McConaughy, S. H. (1997). *Empirically based assessment of child and adolescent psychopathology: Practical applications* (2<sup>nd</sup> ed.). Thousand Oaks, CA: Sage.
- American Psychological Association. (1994). *Publication manual of the American Psychological Association* (4<sup>th</sup> ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> ed.). Washington, DC: Author.
- American Psychological Association. (2008). *Electronic resources*. Retrieved from <http://www.apastyle.org/elecref.html>
- American Psychiatric Association. (2009). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> ed.). Washington, DC: Author.
- Anderson, J. C., Williams, S., McGee, R., & Silva, P. A. (1987). DSM-III disorders in preadolescents children: Prevalence in a large sample from the general population. *Archives of General Psychiatry*, *44*, 69-77.
- Andrews, G., Brugha, T., Thase, M. E., Duffy, F. F., Rucci, P., & Slade, T. (2007). Dimensionality and the category of major depressive episode. *International Journal of Methods in Psychiatric Research*, *16*, S41-S51.
- Angold, A., & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: Empirical, theoretical, and methodological issues. *American Journal of Psychiatry*, *150*, 1779-1791.
- Biederman, J., Faraone, S., Mick, E., & Lelon, E. (1995). Psychiatric comorbidity among referred juveniles with major depression: Fact or artifact? *Journal of the American Academy Child Adolescent Psychiatry*, *34*, 579-590.
- Bierut, L. J., Heath, A. C., Bucholz, K. K., Dinwiddie, S. H., Madden, P. A., Statham, D. J., ... Martin, N. G. (1999). Major depressive disorder in a community-based twin sample: Are there different genetic and environmental contributions for men and women? *Archives of General Psychiatry*, *56*, 557-563.

- Birmaher, B., Ryan, N.D., Williamson, D.E., Brent, D.A., Kaufman, J., Dahl, R., ... Nelson, B. (1996). Childhood and adolescent depression: A review of the past 10 years Part I. *Journal of the American Academy of Child Adolescent Psychiatry*, 35, 1427-1439.
- Bostwick, J. M., & Pankratz, S. (2000). Affective disorders and suicide risk: A reexamination. *American Journal of Psychiatry*, 157, 1925-1932.
- Brown, T. A., & Barlow, D. H., (2005). Dimensional versus categorical classification of mental disorders in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders and beyond: Comment of the special section. *Journal of Abnormal Psychology*, 114, 551-556.
- Campbell, S., & Macqueen, G. (2004). The role of the hippocampus in the pathophysiology of major depression. *Journal of Psychiatry Neuroscience*, 29, 417-425.
- Carlson G. A., & Kashani, J. H. (1988). Phenomenology of major depression from Childhood through adulthood: Analysis of three studies. *American Journal of Psychiatry*, 145, 1222-1225.
- Chen, L-S., Eaton, W. W., Gallo, J. J., Nestadt, G., & Crum, R. M. (2000). Empirical examination of current depression categories in a population-based study: Symptoms, course, and risk factors. *American Journal Psychiatry*, 157, 573-580.
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, 100, 316-336.
- Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current systems and future directions. *Annual Review of Psychology*, 46, 121-153.
- Costello, E. J., Angold, A., Burns, B. J., Erkanli, A., Stangl, D. K., & Tweed, D. L. (1996). The Great Smoky Mountains Study of Youth: Functional impairment and severe emotional disturbance. *Archives of General Psychiatry*, 53, 1137-1143.
- Costello, E. J., Erkanli, A., & Angold, A. (2006). Is there an epidemic of child or adolescent depression? *Journal of Child Psychology and Psychiatry*, 47, 1263-1271.
- Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, 60, 837-44.

- Coyne, J. C. (1994). Self-reported distress: Analog or ersatz depression? *Psychological Bulletin*, *116*, 29-45.
- Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281-302.
- Das-Munshi, J., Goldberg, D., Bebbington, P. E., Brugha, D. K., Brugha, T. S., & Dewey, M. E. (2008). Public health significance of mixed anxiety and depression: beyond current classification. *British Journal of Psychiatry*, *192*, 171-177.
- Dunn, G., Sham, P. C., & Hand, D. J. (1993). Statistics and the nature of depression. *Journal of the Royal Statistical Society*, *156*, 63-87.
- Eaton, W. W., Anthony, J. C., Gallo, J., Cai, G., Tien, A., Romanoski, A., ... Chen, L. S. (1997). National history of Diagnostic Interview Schedule/DSM-IV major depression: The Baltimore epidemiologic catchment area follow-up. *Archives of General Psychiatry*, *54*, 993-999.
- Eaton, W. W., Dryman, A., Sorenson, A., & McCutcheon, A. (1989). DSM-III major depressive disorder in the community: A latent class analysis of data from the NIMH Epidemiologic Catchment Area programme. *British Journal of Psychiatry*, *155*, 48-54.
- Ebmeier, K. P., Donaghey, C., & Steele, J. D. (2006). Recent developments and current controversies in depression. *The Lancet*, *367*, 153-167.
- Eisenberger, N. I., Lieberman, M. D., & Kipling, W. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, *302*, 290-292
- Ferdinand, R. F., De Nijs, P. F. A., van Lier, P. A. C., & Verhulst, F. C. (2005). Latent class analysis of anxiety and depressive symptoms in referred adolescents. *Journal of Affective Disorders*, *88*, 299-306.
- Fergusson, D. M., Horwood, L. J., Ridder, E. M., & Beautrais, A. L. (2005). Subthreshold depression in adolescence and mental health outcomes in adulthood. *Archives of General Psychiatry*, *62*, 66-72.
- Flannery-Schroeder, E. (2006). Treatment integrity: Implications for training. *Clinical Psychology: Science and Practice*, *12*, 388-390.
- Flett, G. L., Vredenburg, K., & Krames, L. (1997). The continuity of depression in clinical and nonclinical samples. *Psychological Bulletin*, *121*, 395-416.
- Gilman, S. E., Kawachi, I., Fitmaurice, G. M., & Buka, S. L. (2003). Family disruption in childhood and risk of adult depression. *American Journal of Psychiatry*, *160*, 939-946.



- Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, *106*, 458-490.
- Goodwin, R. D., Lewinsohn, P. M., & Seely, J. R. (2004). Respiratory symptoms and mental disorders among youth: Results from a prospective, longitudinal study. *Psychosomatic Medicine*, *66*, 943-949.
- Gotlib, I. H., Lewinsohn, P. M., & Seeley, J. R. (1995). Symptoms versus a diagnosis of depression: Differences in psychosocial functioning. *Journal of Consulting and Clinical Psychology*, *63*, 90-100.
- Gould, M. S., Bird, H., & Jaramillo, B. S. (1993). Correspondence between statistically derived behavior problem syndromes and child psychiatric diagnoses in a community sample. *Journal of Abnormal Child Psychology*, *21*, 287-313
- Hankin, B. L., & Abela, J. R. Z. (Eds.). (2005). *Development of psychopathology: A vulnerability-stress perspective*. Thousand Oaks, CA: Sage.
- Haslam, N. (2003). Categorical versus dimensional models of mental disorder: The taxometric evidence. *Australian & New Zealand Journal of Psychiatry*, *37*, 696-704.
- Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H-U., Sirovatka, P. J., & Regier, D. A. (2008). *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V*. Washington, DC: American Psychiatric Association.
- Hirschfeld, R. M., Klerman, G. L., Clayton, P. J., & Keller, M. B. (1989). Personality and depression. *Archives of General Psychiatry*, *40*, 993-998.
- Holma, K. M., Melartin, T. K., Holma, I. A., & Isometsä, E. T. (2008). Predictors for switch from unipolar major depressive disorder to bipolar disorder type I or II: A 5-year prospective study. *Journal of Clinical Psychiatry*, *69*, 1267-1275.
- Horwath, E., Johnson, J., Klerman, G. L., & Weissman, M. M. (2007). What are the public health implications of subclinical depressive symptoms? *Psychiatric Quarterly*, *65*, 323-337.
- Hudziak, J. J., Heath, A. C., Madden, P. F., Reich, W., Burcholz, K. K., Slutske, W., ... Todd, R. D. (1998). Latent class and factor analysis of DSM-IV ADHD: A twin study of female adolescents. *Journal of the American Academy Child & Adolescent Psychiatry*, *37*, 848-857.
- Johnson, J., Weissman, M., & Klerman, G. (1992). Service utilization and social morbidity associated with depressive symptoms in the community. *Journal of the American Medical Association*, *267*, 1478-1483.

- Judd, L. L., Akiskal, H. S., & Paulus, M. P. (1997). The role and clinical significance of subsyndromal depressive symptoms (SSD) in unipolar major depressive disorder. *Journal of Affective Disorders, 45*, 5-18.
- Jyhla, P. (2008). Depression, anxiety, psychiatric comorbidity and dimensions of temperament and personality. *European Journal of Human Genetics, 14*, 963-970.
- Karlsson, A. O., Wallerström, T., Götherström, A., & Holmlund, G. (2000). *American Psychiatric Association diagnostic and statistical manual of mental disorders* (4<sup>th</sup> ed). Washington, DC: American Psychiatric Association.
- Katon, W. J. (2003). Clinical and health services relationships between major depression, depressive symptoms, and general medical illness. *Biological Psychiatry, 54*, 216-226.
- Keller, M. B., Kocsis, J. H., & Thase, M. E. (1998). Maintenance phase efficacy of sertraline for chronic depression: A randomized controlled trial. *Journal of the American Medical Association, 280*, 1665-1672.
- Kennedy, N., Abbott, R., & Paykel, E. S. (2003). Remission and recurrence of depression in the maintenance era: Long-term outcome in a Cambridge cohort. *Psychological Medicine, 33*, 827-838.
- Kendler, K. S., Eaves, L. J., Walters, E. E., Neale, M. C., Heath, A. C., & Kessler, R. D. (1996). The identification and validation of distinct depressive syndromes in a population-based sample of female twins. *Archives of General Psychiatry, 53*, 391-399.
- Kendler, K. S., & Gardner, C. O. (1998). Boundaries of major depression: An evaluation of DSM-IV criteria. *American Journal of Psychiatry, 155*, 172-177.
- Kendler, K. S., Gardner, C. O., & Prescott, C. A. (1998). A population-based twin study of self-esteem and gender. *Psychological Medicine, 28*, 1403-1409.
- Kendler, K. S., Kuhn, J. W., & Prescott, C. A. (2004a). Childhood sexual abuse, stressful life events, and risk for major depression in women. *Psychological Medicine, 34*, 1475-1482.
- Kendler, K. S., Kuhn, J. W., & Prescott, C. A. (2004b). The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *American Journal of Psychiatry, 161*, 631-636.
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). A population based twin study of major depression in women: The impact of varying definitions of illness. *Archives of General Psychiatry, 49*, 257-266.

- Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders—Virginia adult twin study of psychiatric and substance use disorders (VATSPUD)*. New York, NY: Guilford.
- Kendler, R. C., & Magee, W. J. (1993). Childhood adversities and adult depression: Basic patterns of association in a US national survey. *Psychological Medicine*, *23*, 679-690.
- Kessler, K. S., Zhao, S., Blazer, D. G., & Swartz, M. (1997). Prevalence, correlates, and course of depressive subtypes in the national comorbidity survey. *Journal of Affective Disorders*, *45*, 19-30.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., Rush, A. J., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, *62*, 593-601.
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E.E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, *62*, 617-709.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hugues, M., Eshleman, S., ... Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 8-19.
- Kessler, R. C., Nelson, C. B., McGonagle, K. A., Edlund, M. J., Prank, R. G., & Leaf, P. J. (1996). The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *American Journal of Orthopsychiatry*, *66*, 17-31.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives General Psychiatry*, *62*, 617-627.
- Klerman, G. L., & Weissman, M. M. (1989a). Increasing rates of depression. *Journal of the American Medical Association*, *261*, 2229-2235.
- Klerman, G. L., & Weissman, M. M. (1989b). Special communication: Increasing depression. *Journal of the American Medical Association*, *261*, 2229-2235
- Korkeila, K., Hankin, J., Vahtera, J., Kivimaki, M., Kivela, S. L., Sillanmaki, L., & Koskenvuo, M. (2005). Childhood adversities, adult risk factors, and depressiveness. *Social Psychiatry & Epidemiology*, *40*, 700-706.

- Kovacs, M., & Devlin, B. (1998). Internalizing disorders in childhood. *Journal of Child Psychology & Psychiatry, 39*, 47- 63.
- Levinson, D. F. (2006). The genetics of depression: A review. *Biological Psychiatry, 60*, 84-92.
- Lewinsohn, P. M., Hoberman, H. M., & Rosenbaum, M. A. (1988). A prospective study of risk factors for unipolar depression. *Journal of Abnormal Psychology, 97*, 251-64.
- Lewinsohn, P. M., Gotlib, I. H., & Seeley, J. R. (1995). Adolescent psychopathology: IV. Specificity of psychosocial risk factors for depression and substance abuse in older adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 34*, 1221-1229.
- Lewinsohn, P. M., Pettit, J. W., Joiner, T. E., & Seely, J. R. (2003a). The symptomatic expression of major depressive disorder in adolescent and young adults. *Journal of Abnormal Psychology, 112*, 244-252.
- Lewinsohn, P. M., Rohde, P., Seeley, J. R., Klein, D. N., & Gotlib, I. H. (2003b). Psychosocial functioning of young adults who have experienced and recovered from major depressive disorder during adolescence: The Oregon Adolescent Depression Project 'OAPD'. *Journal of Abnormal Psychology, 112*, 353-363.
- Lewinsohn, P. M., Rohde, P., Seely, J. R., Kelin, D. N., & Gotlib, I. H. (2000). Natural course of adolescent major depressive disorder in a community sample: Predictors of recurrence in young adults. *American Journal of Psychiatry, 157*, 1584-1591.
- Lewinsohn, P. M., Solomon, A., Seeley, J. R., & Zeiss, A. (2000). Clinical implications of subthreshold depressive symptoms. *Journal of Abnormal Psychology, 109*, 345-351.
- Lieb, R., Isensee, B., Hofler, M., Pfister, H., & Wittchen, H. U. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of General Psychiatry, 59*, 365-74.
- Lynch, F., & Clarke, G. (2009). Estimating the economic burden of depression in children and adolescents. *American Journal of Preventive Medicine, 31*, 143-151.
- Malhi, G. S., Parker, G. B., & Greenwood, J. (2004). Structural and functional model of depression: From sub-types to substrates. *Acta Psychiatrica Scandinavica, 111*, 94-105.
- Meehl, P. E. (1954). Comment on "Analyzing the clinical process." *Journal of Counseling Psychology, 1*, 207-208.

- Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality*, *60*, 117-174.
- Meehl, P. E., & Rosen, A. (1955). Antecedent probability and the efficiency of psychometric signs, patterns or cutting scores. *Psychological Bulletin*, *52*, 194-216.
- Millon, T., Green, C. J., & Meagher, R. B. (1982). *Millon Behavioral Health Inventory manual* (3<sup>rd</sup> ed.). Minneapolis, MN: National Computer Systems.
- Moffitt, T. E., Harrington, H-L., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., & Poulton, R. (2007). Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Achieves of General Psychiatry*, *64*, 651-660.
- Mossavi, S., Chatterji, S., Verdes, E., Tandon, A., Patel, V., & Ustun, B. (2007). Depression, chronic diseases, and decrements in health: Results from the World Health Surveys. *Lancet*, *370*, 851-858.
- Morgan, D., Sargent, M., Chukwuma, J., & Huges, G. (2008). Audit of metabolic syndrome in adults prescribed clozapine in community and long-stay in-patient populations. *Psychiatric Bulletin*, *32*, 174-177.
- Murphy, D. A., Marelich, W., & Hoffman, D. (2000). Assessment of anxiety and depression in young children: Support for two separate constructs. *Journal of Clinical Child Psychology*, *29*, 383-391.
- Muthen, L. K., & Muthen, B. O. (2004). *Mplus: The comprehensive modeling program for allied research: User's guide* (3<sup>rd</sup> ed.). Los Angeles, CA: Muthen & Muthen.
- Mueller, T. I., Keller, M. B., Leon, A. C. Solomon, D. A., Shea, M., T., Coryyell, W., & Endicott, J. (1996). Recovery after 5 years of unremitting major depressive disorder. *Archives of General Psychiatry*, *53*, 794-799.
- Muir, P., Schmidt, H., Merchelback, H., & Schouten, E. (2001). Anxiety sensitivity in adolescents: Factor structure and relationships to trait anxiety and symptoms of anxiety disorders and depression. *Behaviour Research & Therapy*, *39*, 89-100
- Murray, C. L., & Lopez, A. D. (1996). Evidence-based health policy: Lessons from the Global Burden of Disease Study. *Science*, *274*, 740-743.

- National Institute of Mental Health. (2003). *Breaking ground, breaking through: The strategic plan for mood disorders research of the National Institute of Mental Health*. Washington, DC: U.S. Department of Health and Human Services, National Institutes of Health. Retrieved from <http://www.nimh.nih.gov/about/strategic-planning-reports/breaking-ground-breaking-through--the-strategic-plan-for-mood-disorders-research.pdf>
- National Institute of Mental Health. (2008). *Research roundtable: Heterogeneity in child and adolescent depression*. Retrieved from <http://www.nimh.nih.gov/research-funding/scientific-meetings/2008/research-roundtable-heterogeneity-in-child-and-adolescent-depression.shtml>
- Nesse, R. M. (2006). Evolutionary explanations for mood disorders. In D. J. Stein, D. J. Kupfer, & A. F. Schatberg (Eds.), *Textbooks of mood disorders* (pp. 159-175). Washington, DC: The American Psychiatric Publishing.
- Nestler, E. J., Barrot, M., DiLeone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of depression. *Neuron*, *34*, 13-25.
- Olfson, M., Sing, M., & Schlesinger, H. J. (1999). Mental health/medical care cost offsets: Opportunities for managed care. *Health Affairs*, *18*(2), 79-90.
- Ormel, J., & Neeleman, J. (2000). Toward a dynamic stress-vulnerability model of depression: The role of neuroticism, life events and gender. In T. Harris (Ed.), *Where inner and outer worlds meet: Psychosocial research in the tradition of George W. Brown* (pp. 151-170). London, England: Routledge.
- Parker, G. (1999). Bipolar depression: Does its clinical expression inform us about the clinical features of melancholia? *Bipolar Disorders*, *1*(1), 93.
- Sullivan, P. F., Prescott, C. A., & Kendler, K. S. (2002). The subtypes of major depression in a twin registry. *Journal of Affective Disorders*, *68*, 273-284.
- Pickles, A., & Angold, A. (2003). Natural categories or fundamental dimensions: On carving nature at the joints and the rearticulation of psychopathology. *Development & Psychopathology*, *15*, 529-551.
- Piccinelli, M., Rucci, P., Ustun, B., & Simon, G. (1999). Typologies of anxiety, depression and somatization symptoms among primary care attenders with no formal mental disorder. *Psychological Medicine*, *29*, 677-688.
- Pine, D. S., Cohen, P., & Brook, J. (2001). Adolescent fears as predictors of depression. *Biological Psychiatry*, *50*, 721-724.

- Pine, D. S., Cohen, E., Cohen, P., & Brook, J. (1999). Adolescent depressive symptoms as predictors of adult depression: Moodiness or mood disorder? *American Journal of Psychiatry*, *156*, 133-135.
- Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). Anxiety and depression adolescence as predictors of anxiety and depression in adulthood. *Archives of General Psychiatry*, *55*, 56-66.
- Pincus, H. A., Davis, W. W., & McQueen, L. E. (1999). 'Subthreshold' mental disorders: A review and synthesis of studies on minor depression and other 'brand name'. *British Journal of Psychiatry*, *174*, 288-296.
- Reynolds, C. R., & Richmond, B. O. (1985). *Revised Children's Manifest Anxiety Scale. RCMAS Manual*. Los Angeles, CA: Western Psychological Services.
- Reynolds, W. M. (1986). A model for screening and identification of depressed children and adolescents in school settings. *Professional School Psychology*, *1*, 117-129.
- Reynolds, W. M. (1987). *Suicidal ideation questionnaire*. Odessa, FL: Psychological Assessment Resources.
- Rice, F., van den Bree, M. B., & Thapar, A. A. (2004). A population-based study of anxiety as a precursor for depression in childhood and adolescence. *BMC Psychiatry*, *4*, 43-43.
- Robins, L. N., Locke, B. Z., & Regier, D. A. (1991). An overview of psychiatric disorders in America. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorder in America: The epidemiological catchment area study* (pp. 328-366). New York, NY: Free Press.
- Rohde, P., Lewinsohn, P., & Seeley, J. (1991). Comorbidity of unipolar depression: Comorbidity with other mental disorders in adolescents and adults. *Journal of Abnormal Psychology*, *100*, 214-222.
- Rothman, K. J., & Greenland, S. (1998). *Modern epidemiology*. Philadelphia, PA: Lippincott, Williams, & Wilkins.
- Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology*, *109*, 473-487.
- Ruscio, A. M., & Ruscio, J. (2002a). The latent structure of analogue depression: Should the BDI be used to classify groups? *Psychological Assessment*, *14*, 135-145.
- Ruscio, J., & Ruscio, A. M. (2002b). A structure-based approach to psychological assessment: Matching measurement models to latent structure. *Assessment*, *9*, 4-16.

- Ruscio, J., & Ruscio, A. M. (2004a). Clarifying boundary issues in psychopathology: The role of taxometrics in a comprehensive program of structural research. *Journal of Abnormal Psychology, 113*, 24-38.
- Ruscio, J., & Ruscio, A. M. (2004b). A conceptual and methodological checklist for conducting a taxometric investigation. *Behavior Therapy, 35*, 403-447.
- Ruscio, J., & Ruscio, A. M. (2004c). A nontechnical introduction to the taxometric method. *Understanding Statistics, 3*, 151-193.
- Ryan, N. D., Puig-Antich, J., Ambrosini, P., Rabinovich, H., Robinson, D., Nelson, B., ... Twomey, J. (1987). The clinical picture of major depression in children and adolescents. *Archives of General Psychiatry, 44*, 854-861.
- Sadek, N., & Bona, J. (2000). Subsyndromal symptomatic depression: A new concept. *Depression and Anxiety, 12*(1), 30-9.
- Skodol, A. E., Schwartz, S., & Dohrenwend, B. P. (1994). Minor depression in a cohort of young adults in Israel. *Archives of General Psychiatry, 51*, 542-551.
- Stark, K., Humphrey, L., Laurent, J., Livingston, R., & Christopher, J. (1993). Cognitive, behavioral, and family factors in the differentiation of depressive and anxiety disorders during childhood. *Journal of Consulting and Clinical Psychology, 61*(5), 878-886.
- Stoolmiller, M., Kim, H. K., & Capaldi, D. M. (2005). The course of depressive symptoms in men from early adolescence to young adulthood: identifying latent trajectories and early predictors. *Journal of Abnormal Psychology, 114*, 331-345.
- Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and meta-analysis. *American Journal of Psychiatry, 157*, 1552-1562.
- Sutton, J. M. (2007). Prevention of depression in youth: A qualitative review and future suggestions. *Clinical Psychology Review, 27*, 552-571.
- Ustun, T. B., & Kessler, R. C. (2002). Global burden of depressive disorders: The issue of duration. *The British Journal of Psychiatry, 181*, 181-183.
- Van den Oord, E. J. C. G., Pickles, A. P., & Waldman, I. D. (2003). Normal variation and abnormality: An empirical study of the liability distributions underlying depression and delinquency. *Journal of Child Psychology and Psychiatry, 43*, 180-192.
- Vermut, J. K., & Magidson, J. (2004). *Technical appendix for Latent GOLD 3.0*. Retrieved from [http://www.statisticalinnovations.com/products/lg\\_app3.pdf](http://www.statisticalinnovations.com/products/lg_app3.pdf)



- Wadsworth, M. E., James, B. A., Hudziak, J., Andrew, M. D., Heath, C., Phil, D., & Achenbach, T. M. (2001). Latent class analysis of Child Behavior Checklist Anxiety/Depression in Children and Adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 106-114.
- Waddel, C., Hua, J., Godderis, R., & McEwan, K. (2004). *Preventing and treating depression in children and youth: A research report prepared for the British Ministry of Children and Family Development*. Vancouver, British Columbia: Children's Mental Health Policy Research Program, University of British Columbia.
- Watson, D. (2003). Subtypes, specifiers, epicycles, and eccentrics: Toward a more parsimonious taxonomy of psychopathology. *Clinical Psychology: Science and Practice, 10*, 233-238.
- Wells, K. B., Burnam, M. A., Rogers, W., Hays, R., & Camp, P. (1992). The course of depression in adult outpatients. Results from the Medical Outcomes Study. *Archives of General Psychiatry, 49*, 788-794.
- Widiger, T. A., & Clark, L. A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin, 126*, 946-963.
- Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition. *Journal of Abnormal Psychology, 114*, 494-504.
- World Health Organization. (2009). *Mental health*. Retrieved from [http://www.who.int/mental\\_health/management/depression/definition/en/index.html](http://www.who.int/mental_health/management/depression/definition/en/index.html)



- on-call duties (ER consultation, night on-call for mental health clinic, and domestic violence on-call services).  
Supervisor: Anne C. Dobmeyer, Ph.D.
- August 2009 – Present  
 Bear River Community Mental Health Clinical Assistantship  
Position: Graduate Assistant  
Responsibilities: Intakes, assessments, group therapy, psycho-education, individual therapy, treatment planning, and treatment coordination in a community mental health setting. Weekly evaluations and assessments in a local jail.  
Supervisor: Scott Blickenstaff, PhD  
Hours: Total intervention/assessment hours 97
- August 2009 – Present  
 Cache Valley Cancer Treatment Center Clinical Practicum  
Position: Practicum Student  
Responsibilities: Assessment, formulation and implementation of behavioral interventions with adult oncology patients undergoing medical treatment (e.g., chemotherapy, radiation, surgery). Interventions include individual therapy, end of life planning, and consultation with nursing and medical staff.  
Supervisor: Scott DeBerard, PhD  
Hours: Total intervention/assessment hours 33
- August 2009 – Present  
 Disability Resource Center, Utah State University Veterans Liaison Officer  
Position: Graduate Volunteer  
Responsibilities: Liaison team member assisting veterans returning to an academic setting offering psycho-education and treatment coordination in a university disability resource center.  
Supervisor: Mary Doty, PhD  
Hours: Total intervention/assessment hours 25
- August 2008 – May 2009  
 Utah State University Student Counseling Center Clinical Assistantship  
Position: Graduate Assistant  
Responsibilities: Individual and group therapy. Assessment, diagnosis, and case formulation; interventions with college students who presented with diverse concerns (e.g., depression, anxiety, relationship problems, sexual orientation, conflicts of religion). Therapy using a variety of theoretical orientations (e.g., CBT, DBT, ACT).  
Supervisor: Mark Nafziger, PhD and David Bush, PhD  
Hours: Total intervention/assessment hours 260
- January 2008 – Present  
 Utah State University Student Health and Wellness Center and Clinical Practicum
- August 2006 – April 2007  
Position: Practicum Student  
Responsibilities: Assessment, formulation and implementation of behavioral interventions. Consultation with nursing and medical staff.  
Supervisor: Scott DeBerard, PhD  
Hours: Total intervention/assessment hours 385

- January 2008 – May 2008 Utah State University Survivors of Sexual Abuse Treatment Group and Clinical Practicum
- Sept. 2006 – April 2007 Position: Practicum Student  
Responsibilities: Student co-therapist in weekly process and support group for female survivors of physical, emotional, and/or sexual abuse.  
Supervisor: Carolyn Barcus, PhD  
Hours: Total intervention/assessment hours 60
- January 2008 – May 2008 Avalon Hills Eating Disorder Treatment Facility and Clinical Assistantship
- January 2007 – May 2007 Position: Graduate Assistant  
Responsibilities: Individual therapy and group therapy (DBT, relapse prevention, interpersonal process, and didactic groups), and intakes in a multi-disciplinary team.  
Supervisor: Nathaniel Wood, PhD  
Hours: Total intervention/assessment hours 205
- January 2005 – April 2006 Utah State University Psychology Community Clinic Clinical Practicum  
Position: Practicum Student  
Responsibilities: Intakes, evaluations, assessments, report writing, psycho-education, behavioral parent training, and individual adult and individual child therapy.  
Supervisor: Gretchen Gimple Peacock, PhD, and Scott DeBerard, PhD  
Hours: Total intervention/assessment hours 163

### **Other Professional Positions**

- February 2008 – Present Journal of Terrorism Research  
Position: Student Manuscript Reviewer
- June 2003 – August 2004 McNair Scholars Program, California State University, San Bernardino, CA  
Position: Writing Consultant  
Responsibilities: Evaluation of and consultation with under-represented and economically disadvantaged college students' research designs, written works, and presentations for professional conferences.  
Supervisor: Roy Ramon, PhD
- August 2001 – August 2002 University Center for Developmental Disabilities, California State University, CA  
Position: Graduate Research Assistant  
Responsibilities: Data collection, interviews, and behavioral observations of parents and siblings of children with autism and other developmental delays for long-term treatment plans.  
Supervisor: Charles Hoffman, PhD

### Publication

Armstrong, V., Riechel, C., **Doti, J.**, Crawford, C., & McDougall, S. (2004). *Repeated amphetamine treatment causes a persistent elevation of glial fibrillary acidic protein in the caudate-putamen*, European Journal of Pharmacology, spring.

### Professional Presentations

DeBerard, M. S., Gundy, M. J., **Doti, F. J.**, Grewe, R. J., LaCaille, A. R. *The Use of Retrospective Cohort Studies in Behavioral Medicine Research*. Poster presented at the annual conference of the Society of Behavioral Medicine, San Diego, CA, spring 2008.

**Doti, J.**, Cullum, J. L., & Schroder, K.E.E. *Development and Validation of a Dieting Abstinence Violation Effect (DAVE) Scale*. Poster presented at the annual conference of the Society of Behavioral Medicine, San Francisco, CA, spring 2006.

**Doti, J.**, Hoffman, C.D., & Sweeney, D. *Perceptions of Resources and Psychological Adjustment of Parents Raising a Child With and Without Autism*. Poster presented at the annual meeting of the American Psychological Association, Honolulu, HI, August 2004.

Benitez, C.P., Hoffman, C.D., Sweeney, D, & **Doti, J.** *Maternal parentification of siblings in families with and without a child with a developmental disability*. Poster presented at the annual meeting of the American Psychological Association, Honolulu, HI, August 2004.

**Doti, J.**, Hoffman, C.D., & Sweeney, D. *Perceptions of Resources and Psychological Adjustment of Parents Raising a Child With and Without Autism*. Round-table presentation at the second annual research and scholarship symposium of the College of Education at California State University, San Bernardino, May 2003.

### Teaching Experience

August 2005 – May 2006	Psychology of Human Adjustment (Utah State University) and Independent Instructor: 4 Semesters
August 2004 – May 2005	Supervised five teaching assistants
August 2005 – Dec. 2005	Health Psychology (Utah State University) Independent Instructor: 1 Semester.  Supervised one teaching assistant
August 2005 – May 2006	Introduction to Psychology (Utah State University) Teaching Assistant: 2 Semesters
Sept. 2002 – May 2003	Experimental Psychology (California State Univ. San Bernardino) Independent Instructor: 3 Quarters Supervised two teaching assistants

### Military Experience

August 1987 – Present	US Air Force Reserves 4th Combat Camera Squadron, March Air Force Base, CA Combat Photographer/Unit Deployment Manager
-----------------------	---

Rank: Master Sergeant (E-7)

Duties: Preparation and recovery of military stateside and overseas deployments of multimedia teams in support of normal and crisis operations. Aircrew aeronautical-rated photographer. Numerous short and long-term military overseas deployments to over fifteen countries in support of real-world contingencies.

Commander: Lieutenant Colonel Kimberly Garcia, MS

March 2007 – Nov. 2007

Operations Enduring Freedom (Iraq)

Duties: Photojournalist documenting military combat missions in Iraq and civilian activities for Pentagon operational and historical needs.

Commander: Lieutenant Colonel Kimberly Garcia, MS

May 2006 – Sept. 2006

Operations Iraqi Freedom (Iraq)

Duties: Photojournalist documenting Army combat missions in Iraq and civilian activities for Pentagon operational and historical needs.

Commander: Lieutenant Colonel Kimberly Garcia, MS

August 1982 – July 1986

US Air Force Active Duty

Duties: Aircraft maintenance mechanic at Mildenhall Air Base, England and Holloman Air Force Base, NM.

### **Professional Affiliations**

Society of Behavioral Medicine (SBM), Student Affiliate

American Psychological Association (APA), Student Affiliate

### **Volunteer & Leadership Experience**

March 2001 – August 2004

Riverside Crisis/Suicide Hotline, Riverside County, CA

Duties: Primary telephone contact for general public for immediate emotional and or physical crisis needs.

Supervisor: Gina Cuevas, MA

August 2001 – February 2002

Patton State Hospital, San Bernardino, CA

Duties: Assistant to psychiatric technicians and psychologists in reintegrating a forensic population for halfway community housing.

Supervisor: Jerry Shure, MS

### **Specialized Training**

May/2011

Deployment Psychology in the Military Presented by the Center for Deployment Psychology, Navy National Medical Center, DC

Total: 40 hours

May/2011

Emotion Processing Therapy for Post-Traumatic Stress Disorder Presented by the Center for Deployment Psychology, Bethesda Navy National Medical Center, DC

Total: 16 hours

April/2011

Couples Focused Emotional Processing Therapy Presented by the Center for Deployment Psychology, Wright Patterson AFB, OH

Total: 16 hours

November/2010	Cognitive Processing Therapy for Post-Traumatic Stress Disorder, Presented by Priscilla Schulz, LCSW, Bethesda Navy National Medical Center, DC Total: 16 hours
October/2010	Cognitive Behavioral Therapy for Insomnia, Presented by the Center for Deployment Psychology, Wright Patterson AFB, OH Total: 16 hours
February/2010	The Dynamics of Gottman Couples Therapy, Presented by John Gottman, PhD, Salt Lake City, UT Total: 8 hours
June/2009	Ethics in Psychology: American Psychological Association Roundtable Seminar Presented by Stephen Behnke, JD, PhD, Utah State University, Logan, UT Total: 16 hours
April/2009	Acceptance and Commitment Therapy Training, Presented by Steven Hayes, PhD, Utah State University, Logan, UT Total: 30 hours
June 2009	American Psychology Association roundtable seminar on Ethics presented by Stephen Behnke, JD, PhD (APA Lecturer) (Utah State University) Total: 16 hours
April 2009	Acceptance and Commitment Therapy (ACT) seminar and conference presented by its founder Steven Hayes (Utah State University) Total: 30 hours
March 2009	Bear River Community Mental Health: Traumatic Brain Injury Conference Total: 8 hours
November 2008	Bear River Mental Health Services (Logan, Utah) Treating Moderate and Severe Behavior Problems Associated with Neuro-cognitive Impairments. Total: 8 hours
October 2008	Salt Lake Veterans Administration Hospital (Park City, Utah), Traumatic Brain Injury and the Returning Soldier Total: 8 Hours
March 2008	Acceptance and Commitment Therapy (Utah State University), Advanced clinical application of ACT principles for an eating disordered population. Total: 16 hours
November 2008	Multicultural Seminar (Utah State University), Incorporating the tenets of Acceptance and Commitment Therapy to expand clinicians' attitudes, beliefs, and behaviors towards ethnic minorities. Total: 8 Hours

January 2008-2009	Acceptance and Commitment Therapy (Avalon Eating Disorder, Facility, Paradise, Utah), Weekly seminars reviewing and applying the tenets of ACT in a residential eating disordered treatment facility. Total: 43 Hours
September 2008 –2010	Student Clinicians' Case Review Group (Utah State University), Weekly review of psychotherapy case load by a three member graduate student group. Relevant clinical challenges including transference, case conceptualization, theoretical treatment focus. Total: 56 hours
October 2008	Latent Class Analysis (LCA) (Utah State University), On-line statistics course introducing the foundations and application for an emerging statistical approach useful for identifying latent populations within large data sets.
<b>Outreach Presentations</b>	
February, 2009	Substance Abuse Screening, Annual Utah State University mental health information and services outreach.
January, 2009	Managing Student Stress and Sleep Hygiene, Presentation to Utah State University Housing, residential assistants and students.
October 2008	Depression Screening, Annual Utah State University mental health information and services outreach.