Running head: SEX DIFFERENCES IN DEPRESSED YOUTH

A Cross-Sectional Examination of Sex Differences in Depressed Youth: Ruminative Response Theory, Pubertal Status, and Hormones

Jennifer L. Welsh

Lakehead University

Dissertation submitted in partial fulfillment

of the requirements for a Doctor of Philosophy (Clinical Psychology)

November 2007

Supervisor: Dwight Mazmanian, Ph.D.

Second Readers: Brian O'Connor, Ph.D., and Kirsten Oinonen, Ph.D.

Internal/External: Keith Brownlee, Ph.D.

External Examiner: Elizabeth Hampson, Ph.D.

Your file Votre référence
ISBN: 978-0-494-33576-5
Our file Notre référence
ISBN: 978-0-494-33576-5

Acknowledgements

First, it is most appropriate to acknowledge the researchers I have cited in this document. They provided me the initial inspiration to undertake this project. Over the last four years many others have offered me their support and encouragement to persevere through the tough times. Thank you to Dr. Mazmanian for helping me work through my *brief* moments of panic with his witty comments, and helping me to make this a better product. He has offered me sound advice that I will carry forward in my future research endeavors. I would like to express my gratitude to all the individuals on my dissertation committee who took their jobs seriously and critically evaluated my work from proposal to final draft. Although an external examiner's role only begins at the end of a dissertation, I must acknowledge Dr. Hampson for sharing her perspective in this area. Her insights have created more research ideas I hope to be fortunate enough to follow up on. Applied research would not exist without the cooperation of participants in the community. Dozens of individuals were involved in the coordination of data collection. Thank you to all the school board personnel (administration, principals, and teachers) who were willing to learn more about their students, and generously offered time from their very busy schedules to allow it to happen. I am forever indebted to the adolescents who willingly gave me the time to ask them lots of questions. Their positive attitudes were appreciated and, perhaps without knowing, they have taught me many lessons about working with young people. My research assistant freely shared her heart of gold in this project by assisting with data collection. She was instrumental in making the process efficient and organized. And finally, it took very special peeps and family members to create the right space for me to complete this dissertation. I fondly thank them all for doing their own part to get me to the end.

Abstract

Prevalence estimates indicate that during adolescence a shift in the sex ratio of depression occurs that results in twice as many females reporting symptoms as males. The purpose of the current study was to investigate the relationship between this sex shift and a set of risk factors (ruminative coping, pubertal status, and hormones) using the tripartite model. Data were collected from 213 adolescents (between ages 12 and 19) across all stages of pubertal development (pre-, mid-, and post-puberty) through paper and pencil questionnaires and measures of hand features. Results partially supported the hypotheses: sex differences in response style were found, the relationship between pubertal status and depression was moderated by sex, and the 2D:4D variable significantly predicted depressive symptoms whereas finger ridge count did not. A hierarchical interpretation of the findings is presented and discussed in the context of Nolen-Hoeksema and Girgus' (1994) developmental models. Implications for assessing and treating depression in youth are noted. Limitations of the current study are reviewed along with suggestions for future research.

Table of Contents

Page

List of Tables.

List of Figures.

Page

List of Appendices.

A Cross-Sectional Examination of Sex Differences in Depressed Youth: Ruminative Response Theory, Pubertal Status, and Hormones

Introduction

For many years clinicians and other professionals have hypothesized about the origin and progression of depression in youth. The answers to their research questions have depended on the accepted definition of depression and the age of the child or adolescent. Within the last few decades, general acceptance that children and adolescents can become depressed has been achieved (Garber, 2000; Garber & Horowitz, 2002). Dahl and Ryan (1996) credit much of this validation to psychobiological studies that found areas of continuity across child, adolescent, and adult forms of depression. Despite this progress, there are many important questions that remain with respect to how depression in youth[1] is similar to, or different from, the adult form of depression, and *if* there are differences, what accounts for them.

Currently there are three variations in the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, Text Revision* (*DSM-IV-TR*; American Psychiatric Association, 2000) in the criteria for depressive disorders among adolescents compared to adults and they relate to mood, duration, and expected gains. First, irritable mood is considered an acceptable manifestation of dysphoric mood for youth, rather than just depressed mood as for adults. Second, the duration of dysthymia is one year for youth, rather than two years as for adults. Third, failure to make expected weight gains is also an acceptable criterion for youth, rather than weight gain or loss as for adults. Thus, according to the *DSM-IV-TR*, there are few real differences in symptoms among

[1] Throughout the duration of this paper, the terms adolescent and youth will be used to refer to individuals who have not yet reached adulthood. In many of these instances children will be included in this category and clarification will be provided when this is not the case.

adolescents and adults with respect to the clinical profile of depression. This model then requires adolescents to meet basically the same criteria in terms of symptoms of depression to receive this diagnosis. In other words, *DSM-IV-TR* maintains a downward extension of adult diagnostic criteria in this instance.

Some authors would argue that rather than adopting a downward extension, it is necessary to incorporate a developmental perspective that considers depression in youth at different stages (Kaslow, Adamson, & Collins, 2000; Lee & Rebok, 2002). Thus, developmental psychopathologists suggest that manifestations of depression likely depend on the youth's level of cognitive, social, and physiological development (Cicchetti & Toth, 1998; Hankin & Abramson, 2001). This implies that there may be further distinctions that need to be made between adult and youth forms of depression.

Weiss and Garber (2003) suggest that there are two ways in which there may be developmental differences in depressive symptoms: youths and adults might differ in how they *report* specific symptoms (i.e., expressive differences), or the symptoms that comprise the disorder may *actually* be different for youths and adults (i.e., syndrome differences). For example, Kashani and Carlson (1987) suggest that younger children may be more likely than adults to express the "pain" of depression through physical or somatic symptoms. Accepting this view, it is possible that the surface expression of symptoms may *appear* different although the underlying core symptoms may be the same (Born, Shea, & Steiner, 2002), or there may be different core symptoms or processes (Lee & Rebok, 2002) that are relevant at varying developmental levels.

Although researchers and clinicians may have opinions on this issue of developmental differences in depressive symptoms, it has yet to be resolved in the

scientific literature (Kaufman, Martin, King, & Charney, 2001). Any firm statements made either way would therefore be premature.

This ongoing and undecided issue has important implications for how the assessment and treatment of depression is managed. Primarily, the tools currently in use for diagnosing depression in youth may not be accurate. Also, the techniques used for treating depression may not be appropriate. Unlike adults, it seems that depressed adolescents fail to respond to tricyclic antidepressants (Hazell, O'Connell, Heathcote, Robertson, & Henry, 1995; Steiner, Born, & Marton, 2000). With respect to psychosocial treatments, also consider the possibility that some clinicians who believe there are no developmental differences in depression may modify adult psychotherapies for use with adolescent samples by simplifying the complexity of content (Ingram, Nelson, Steidmann, & Bistricky, 2007). Currently however, there is insufficient evidence to validate that rationale because we do not know if the same symptoms should be targeted for intervention in younger samples. Obviously then, much is left to be answered with respect to developmental issues of depression. Given the high prevalence estimates of depression in adolescents, these conceptual differences have some very practical consequences with respect to clinical outcomes.

Prevalence

Epidemiological studies of depression have yielded different prevalence estimates (for both adolescents and adults) primarily due to different methods of classification and exclusion criteria. While this is true, some trends have been reported fairly consistently in the literature. It is clear that prevalence rates of depression increase with age (Hankin & Abramson, 2001; Nolen-Hoeksema, 1990; Seiffge-Krenke & Klessinger, 2000). More

specifically, rates of depression among preschool children have been reported to be around 1% (Kashani & Carlson, 1987), and among prepubertal children to be less than 3% (Fleming & Offord, 1990). By adolescence however, the rates of depression climb to 6.4% (Fleming & Offord, 1990) and are comparable to adult rates (Hankin & Abramson, 2001; Nolen-Hoeksema, Larson, & Grayson, 1999; Ziegert & Kistner, 2002). Knowing that depression can occur in very young children, and that some will struggle with depression for many years, should emphasize the importance of examining (from a developmentally appropriate perspective) the factors that lead to depression in youth.

It is during adolescence (ages 12 to 15) that a shift in the sex ratio of depression occurs in terms of prevalence (Broderick, 1998; Galambos, Leadbeater, & Barker, 2004; Garber & Horowitz, 2000; Hankin et al., 1998). Prior to adolescence the ratio of males to females reporting depressive symptoms is comparable (males are perhaps slightly higher), however, after adolescence, *twice* as many females report depressive symptoms than males (Garber, 2000; Nolen-Hoeksema, 2002). This is a considerable shift in trends that is maintained until and during adulthood, and cannot be accounted for by chronological age alone. It is this trend that is of primary interest in the present paper.

Other factors obviously impact this shift as well, and chronological age may therefore mask important developmental transitions that may more accurately reflect when this gender difference actually emerges (Hankin & Abramson, 2001). For example, pubertal development predicted the emergence of the sex difference in depression better than age as reported by Angold, Costello, and Worthman (1998).

The sex difference in rates of depression has consistently been reported in countries such as Lebanon, Taiwan, Germany, France, New Zealand, and 25 others

(Wolk & Weissmann, 1995). These findings were found across geographically widespread areas and across a diversity of cultures. This extensive prevalence speaks to the stability of this shift. However, some authors (Piccinelli & Gomez Homen, 1997) comment that the female preponderance of depression is limited, or even absent, in traditional societies and in socially homogeneous samples. These authors considered current prevalence rates, 6 month rates, 12 month rates, as well as lifetime prevalence rates using a variety of diagnostic criteria and instruments. In their summaries, the ratio of female-to-male prevalence of depression ranged from 0.8 to 3.5. They were also careful to acknowledge some of the many factors that likely influence gender differences in prevalence rates (e.g., measurement procedures, recall bias). Despite these findings, many other authors *have* identified the shift in sex differences of depression as an important issue for many societies.

Although years of research has been devoted to uncovering what exactly it is about adolescence and societies that increases individual risk for depression, and what accounts for the change in sex ratio, there are no clear or definite answers. We do know that there are associated risks that accompany the onset of depression in adolescents (e.g., anxiety disorders, eating disorders, conduct disorders [Galambos et al., 2004]; and school dropout and suicide [Marcotte, Fortin, Potvin, & Papillon, 2002]), and we have made some progress in outlining some of the pathways that may lead to depressive symptoms in males and females (Galambos et al., 2004).

It is common for individuals with depression to also have other mental health issues with which to contend. More specifically, the overlap between depression and anxiety is quite significant (Collins, Westra, Dozois, & Burns, 2004), both in youth and

adults, and comorbidity rates for these two disorders have been reported between 28% and 75% (Compas & Oppendisano, 2000). Models have been devoted to exploring the similarities and differences between these two disorders (Clark & Watson, 1991), and have been well received in the literature. Yet questions are still left unanswered. Some research that has examined psychobiological issues has suggested that anxiety disorders often precede depressive disorders in young subjects (Dahl & Ryan, 1996). Compas and Oppendisano (2000) suggest that the conceptualization of the developmental course of depression and anxiety in young people remains open to debate and is perhaps the most important question to answer.

Definitions

Before providing a summary of some proposed developmental pathways for depression in youth, it will be important to clarify some issues concerning the definition and assessment of this issue. Hankin and Abramson (2001) have delineated differences between terms that will be useful here. For the purposes of this project, depressed mood will refer to affective quality, such as feeling sad or unhappy, for an unspecified period of time. Depressive symptoms, rather than a categorical diagnosis of depression, will be the focus of this paper[2].

Another important point to address is the significant overlap between depression and anxiety referred to above. Clark and Watson (1991) propose the tripartite model that accounts for both the overlap and distinct features of depressive and anxious disorders. This model suggests that despite the general distress factor (i.e., negative affect; NA) that depression and anxiety share, another factor of temperament is also important in terms of

[2] A depressive disorder is categorically defined and is composed of a list of possible depressive symptoms that must be present for a specified period of time (i.e., clinical levels of depressive symptomatology). Categorical diagnosis is neither necessary nor required for the purposes of this project.

distinction (i.e., positive affect; PA). NA is defined as a general trait dimension with aspects ranging from mood to behavior that cause experiences of negative moods, emotions, and cognitions (Clark, Watson, & Mineka, 1994). PA is defined as a general trait dimension with aspects ranging from mood to behavior that cause experiences of positive emotionality, energy, and affiliation (Clark et al., 1994). The tripartite model proposes that low PA is relevant in depression but not anxiety. Overall then, this model proposes that depressed individuals tend to have high NA as well as low PA. Although many previous studies have examined depressive symptoms as part of a unidimensional construct, this project will consider both NA and PA as comprising important aspects of depressive symptoms. While this model was initially directed at adult samples, research is now accumulating that promotes the application of the tripartite model with youth (Laurent & Ettelson, 2001).

Not withstanding the distinction previously mentioned between depressive symptoms and diagnosis, it is interesting to note that gender differences are not found with respect to the prevalence of bipolar disorder for adolescents or adults (Piccinelli & Wilkinson, 2000). Gender differences do, however, exist with respect to the symptoms associated with a bipolar episode.

Etiology

Several theories have been put forth that attempt to explain the emergence of sex differences in depression during adolescence. As referred to previously, some theorists accept a downward extension of adult theories (with little modification), while other theorists search for perspectives that incorporate a developmental progression. In support of a downward extension model, Kovacs, Devlin, Pollock, Richards, and Mukerji (1997)

found evidence to support the notion that early (child and adolescent) and later (adult) onset forms of depression *may* share the same diathesis. Although we have achieved *better* conceptualizations of youth with depression in the last decade or so, it still remains a difficult task to delineate causal mechanisms.

Among those developmental theories that have been proposed, most research has focused on Nolen-Hoeksema and Girgus' (1994) three models. The first model (known in the literature as Model 1, but hereafter referred to as the Prevalence Model) proposes that the *same factors* cause depression in girls and boys, but the *causes become more prevalent in girls* than boys in early adolescence. The assumption here is that gender differences in depression follow the emergence of gender differences in the risk factors in early adolescence. Thus, prior to adolescence the risk for depression is equal in girls and boys, but during adolescence something changes that puts girls at more risk for depression.

The second model (known in the literature as Model 2, but hereafter referred to as the Different Factors Model) proposes that factors leading to depression are *different* for girls and boys, and that the risk factors leading to depression in girls become more common in early adolescence. Thus, risk factors for adolescent girls, although different from risk factors for adolescent boys, increase much more than those for adolescent boys.

The third model (known in the literature as Model 3, but hereafter referred to as the Interaction Model) proposes that girls are more likely to have *characteristics* that put them at risk for depression even before adolescence, but it is only when these characteristics *interact* with certain challenges of early adolescence that girls develop higher rates of depression. This model assumes that the factors that cause depression are

the *same* in girls and boys (like the Prevalence Model), but that these risk factors are already more common in girls prior to adolescence and only emerge as more salient when they interact with new challenges.

Nolen-Hoeksema and Girgus (1994) also outline three categories of variables that may provide evidence for their proposed models: social challenges (e.g., negative life events, parental and peer expectations), personality risk factors (e.g., ruminative coping), and biological challenges (e.g., hormonal changes, puberty, related psychological consequences). Many researchers over the last decade have conducted studies investigating each of these factors through simple designs where the factor of choice occupies a central role and few other competing factors are included. Many of these researchers have found significant and informative results through this approach. However, when all of this information is viewed as a whole, the results are unclear. The weakness with this line of research has been that most studies have only considered single factor designs and main effects (e.g., Broderick, 1998) to the neglect of possible interaction effects.

Few researchers have been able to investigate a number of these factors within one design. Seiffge-Krenke and Stemmler (2002) are an exception. They examined variables in all three categories discussed by Nolen-Hoeksema and Girgus (1994) by integrating variables across categories. Hankin and Abramson (2001) acknowledge that it makes sense to integrate and elaborate on existing general depression theories to explain the development of gender differences in depression, rather than scrap the empirically supported theories altogether. Accordingly, Seiffge-Krenke and Stemmler (2002) view the past research in this area as corroborative rather than inconsistent, and they attempt to

pull it all together in a refined and comprehensive manner. Their work is, however, limited in terms of generalizability because their samples only included German adolescents and they used mostly German measures of constructs.

The purpose of this dissertation will be to comprehensively study some of the domains outlined by Nolen-Hoeksema and Girgus (1994) by identifying the most relevant factors in those domains, to apply the information to a Canadian sample of adolescents, and to demonstrate the findings using measures developed for English speaking participants. A detailed summary of the research in each category (i.e., social challenges, personality risk factors, and biological challenges) will be reviewed prior to outlining the methodology used in this project.

Social Challenges and Stressors

With respect to social challenges, Nolen-Hoeksema and Girgus (1994) have considered negative life events (e.g., sexual abuse, death of a sibling), as well as parent and peer influences as etiological factors that describe how gender differences in depression might emerge during adolescence. All of the factors in this domain obviously involve relationships between adolescents and individuals, groups, or society as a whole.

Stressors can be defined as the lack or loss of a highly desirable and obtainable goal or the presence of a highly undesirable and inescapable event (Garber, 2000). Seiffge-Krenke (1995) elaborates on the issue of social challenges and stressors and proposes that the impact of adolescent stress on depression varies according to the *type* of stressor experienced as well as the *duration* of that stressor. Therefore, it is suggested that isolated major life events be considered as important, as well as frequent daily stressors (Compas, Davis, Forsythe, & Wagner, 1987).

Other researchers have commented on a link between stressful life events and health and psychological functioning in general (Newcomb, Huba, & Bentler, 1981). Watson, Clark, and Tellegen (1988) refer specifically to the relationship between frequency of unpleasant events and the NA that is associated with both depression and anxiety. It is unclear if this relationship would still be significant if the low PA that is also associated with depression were considered, and also how it would play out in youth samples. The issue of stressors will become more relevant in the section devoted to the HPA axis and cortisol levels in the biological challenges section.

Although researchers have been unable to confirm the existence of higher levels of stress in female adolescents (Seiffge-Krenke, 1995), it is likely that males and females are more vulnerable to *specific types of stressors at different times*. For example, Cyranowski, Frank, Young, and Shear (2000) found that females are more affected by stress associated with interpersonal relationships than males, and this may be especially so after puberty. Rudolph (2002) proposed that gender differences in the experience of stress and emotional reactions to stress, contribute to the development of gender differences in both anxiety *and* depression during adolescence. Rudolph also suggests that research in this area has indicated that girls experience higher levels of the type of stress that is most strongly associated with depression (i.e., interpersonal stress) than boys, *especially* during adolescence. This effect has been reported in adult women as well (Hammen, 2003).

Theory and research support a connection between depressive symptoms and both early family experiences and peer competence (Rudolph & Asher, 2000). Galambos et al. (2004) found that lack of social support was a significant predictor of change in

depressive symptoms. That is, as social support decreased, depressive symptoms increased. However, these authors were unable to specify if the low social support reflected problems within the peer or parent relationship (or both) and suggest that future studies should examine the issue further with tools that can distinguish effects from different sources.

Rudolph and Asher (2000) found that peers could serve as a social support network that can compensate for poor family relationships, and that peers have been found to be somewhat of a buffer for children during times of stress. Peer relationships, which become more important during the transition to adolescence (deMatos, Barrett, Dadds, & Shortt, 2003), are one social factor that may positively influence adolescent psychological functioning.

Alternatively, peers may not always be a positive influence or protective factor during times of stress. For example, early peer difficulties can be risk factors for later maladjustment as well (Parker & Asher, 1987). Fergusson, Wanner, Vitaro, Horwood, and Swain-Campbell (2003) examined the link between deviant peer affiliations and depression in adolescents and found that a causal chain process was implicated. That is, deviant peer affiliations led to increased externalizing behaviors with the negative consequences of that affiliation leading to depression. Considering the seemingly conflictual results in both domains (parents/peers, support/stress) then, it would be important to consider the role of *both* parents and peers in terms of social support to assess the impact of the new challenges faced during adolescence.

In summary then, a consistent link between stressful life events and depression has been revealed. It remains to be seen if the relationship holds if one considers both NA

and PA in models of depression. Studies have been fairly consistent in their support for the influence of stressful life events on increases in, and onset of, depressive symptoms in adolescents. This is perhaps less evident in children (Garber & Horowitz, 2002). It therefore seems possible that how one interprets and reacts to such stressful events will likely influence whether one will experience depressive symptoms. This phenomenon is referred to as coping and will be discussed next.

Personality Risk Factors

With respect to personality risk factors, Nolen-Hoeksema and Girgus (1994) restrict their discussion to gender intensification theory. That is, they comment that socialization pressures lead boys and girls to adopt personality characteristics that are consistent with their own gender. In turn, they suggest that feminine characteristics are more depressogenic than male characteristics. This is in line with their proposed Prevalence Model. An example they use to demonstrate this view, and that has received much research attention, is coping style. This factor is a primary variable of interest in this research.

Ruminative Coping Style

Nolen-Hoeksema and Girgus (1994) suggest that differences in coping with emotional distress might account for gender differences in depressive symptoms. Specifically, they consider ruminative coping. Papageorgiou and Wells (2004) suggest that rumination refers to the entire class of thought that has a tendency to recur. Nolen-Hoeksema's response styles theory of depression (1990) conceptualizes rumination as repetitive and passive thinking about symptoms of depression and the possible causes and consequences of these symptoms without taking action to relieve them. More recently,

Robinson and Alloy (2003) have further developed the concept of rumination to include *stress-reactive rumination*, which is the tendency to ruminate on negative inferences following stressful events but prior to the onset of depressed mood (referred to above), and *emotion-focused rumination*, which occurs in response to depressed mood. Their work suggests it may be the timing and content of rumination that may be helpful in determining who is at risk for developing depression.

Regardless of the exact definition accepted, Nolen-Hoeksema (1998) suggests that ruminative responses to depression exacerbate and prolong periods of depression through at least three mechanisms. First, they enhance the negative effects of depressed mood on one's life by making negative interpretations more accessible and more likely to be used. Second, they interfere with interpersonal problem solving by enhancing pessimistic thinking. Third, they inhibit people from engaging in instrumental behaviors.

Her research over the last two decades (see Nolen-Hoeksema, 1998 for a summary) supports the argument that women are more likely than men to demonstrate a ruminative style of coping. Other researchers have demonstrated this as well (Broderick, 1998; Spasojevic & Alloy, 2001). There is an overwhelming sense that the NA that is associated with depression is implicated in these studies, but it is unclear if the low PA that is also associated with depression (Clark et al., 1994) plays a role as well.

There is less confidence in the same ruminative coping assertion when applied to youth. Broderick (1998) focused her work exclusively on youths and found that preadolescent girls were more likely than boys to use rumination across all domains she measured (i.e., academic stressors, family stressors, peer stressors, discrete event stressors, daily hassle stressors, perceived seriousness, and perceived effectiveness). This

preliminary work suggests that there may be differences in how youth use rumination as a coping strategy compared to adults (i.e., more general tendencies applied to a variety of life issues rather than specific strategies or approaches for designated issues only).

In line with Nolen-Hoeksema (1990) and Broderick's (1998) work, Ziegert and Kistner (2002) proposed that girls' tendency toward a ruminative style of coping is carried forward from childhood into adolescence, where it interacts with new stressors that girls face. They say because girls ruminate more about the new challenges they experience, more depressive symptoms that last for longer duration are more likely. They found support for this proposition, and in turn, support for Nolen-Hoeksema and Girgus' (1994) Interaction Model. While this pattern may have been revealed in Ziegert and Kistner's (2002) sample, this concept has not been investigated directly with most adolescent samples (Seiffge-Krenke & Stemmler, 2002), nor has the distinction between NA and PA been examined in adolescent samples with respect to ruminative coping.

It is also important to consider that measures of response style have changed over the years. Previously Nolen-Hoeksema (1990) assumed that rumination and distraction were opposite ends of a continuum. Her more recent work considers them to be orthogonal concepts (Nolen-Hoeksema, 1998; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). This is in line with Schwartz and Koenig (1996) who provide some evidence that rumination and distraction represent two independent constructs.

Recently, the Response Styles Questionnaire (RSQ, Nolen-Hoeksema's measure of rumination that has been widely used with adults) has been criticized on aspects of its construct validity (i.e., overlap between rumination and emotion-focused coping, self-criticism, confound with depression, and negative affectivity-temperament focus) and

clinical utility (Kasch, Klein, & Lara, 2001). Schwartz and Koenig (1996) also suggest that response style has a greater influence on mood when stress is high (i.e., there is an interaction between response style variables and negative life events). This project will attempt to overcome some of the above limitations while examining ruminative coping style among youth.

Although this new understanding of rumination has been supported with adult samples, it is unknown if the structure of children's response style is the same. One study that specifically addresses this issue (Muris, Van Brakel, & Meesters, 1998) found that high depression scores for their sample of children were accompanied by more frequent employment of *all* types of coping strategies (e.g., active problem solving, distraction, avoidance, social support seeking, depressive reaction, expressing emotions, and comforting thoughts). This implies that children may use a trial and error approach when experiencing depressive symptoms and do not rely on any single approach consistently. Further support for this claim is needed.

To understand if the response style theory of depression is applicable to children, it is necessary to use measures of rumination and distraction for children that posses stronger psychometric properties. Ziegert and Kistner (2002) have commented on several flaws with previous measures of response styles in children (e.g., item overlap, poor construct definition, psychometric inconsistency), and have developed a new measure they feel better addresses pure response styles in preadolescents - the Children's Response Styles Scale (CRSS). A major criticism they, and others (Treynor et al., 2003) cite, is that most research has evaluated response styles for depression rather than response styles in general. When a measure contains overlapping items (e.g., items that

address both depressive symptoms and particular response styles in one question), there is always an alternative explanation for the observed findings (i.e., the relation is due to shared items and not the construct measured). One of the main benefits of their scale is that it assesses pure response style without the confound of depressive symptoms.

With this new scale, Ziegert and Kistner (2002) have found preliminary support for the downward extension of the response style theory to children. That is, they found partial support that girls use a ruminative response style more frequently than boys, and this may indeed put them at higher risk for depression.

In contrast, Abela, Brozina, and Haigh (2002) reported no sex differences in the utilization of ruminative responses. Although they did find support that a ruminative coping style increased depressive symptoms over a 6-week period, they could not attribute that to a sex effect. These authors also suggest that because rumination and depression were not moderated by initial levels of such symptoms in their sample, rumination likely plays a role in the *onset and maintenance* of depression for *both* sexes.

With such conflicting results and the recent conceptual advances made with rumination scales, it is obvious that further work in this area is warranted before answers regarding youth's coping styles and the relationship with depression can be determined. It is promising to see that more precise measures of the construct with stronger psychometric properties are now available to use with child populations and may offer more specific results. In the past, adult measures were perhaps inappropriately used with youths. Also, pairing these new measures up with aspects that have been neglected in the past (i.e., low PA) may prove to be fruitful.

Psychological Consequences – Body image

Some research has begun to focus on the psychological reactions that follow from the biological changes that occur in puberty (e.g., acceptance or dissatisfaction with body development and changes; Hankin & Abramson, 2001). We know that there is a difference in the way that boys and girls experience the physical challenges related to puberty (Marcotte et al., 2002). Specifically, boys tend to express greater satisfaction with such changes, while girls tend to experience more stress (Nolen-Hoeksema & Girgus, 1994). It has therefore been hypothesized that body image and the impact of this transition on self-esteem mediates the relationship between pubertal status and depressive symptoms.

Marcotte et al. (2002) have found support for such a relationship during the transition to high school – a stressful life event that occurs at the beginning of adolescence. Coleman (1989) suggests in his focal theory of adolescence that if normative developmental transitions occur simultaneously, they are more stressful because they do not allow the individual to adjust before new challenges are encountered. This specific event (i.e., puberty) seems to be less of an issue for boys because they enter puberty after the transition to high school has already occurred. Results from Marcotte et al. (2002) suggest that the impact of puberty differs based on the appreciation of the adolescent's appearance (i.e., body image), self-esteem, and on the level of stress associated with such changes.

Most of the research that focuses on body image and the sex differences associated with depression is correlational (e.g., Johnson & Wardle, 2005). Therefore it is difficult to tease out which effect occurs first. That is, does the response to pubertal

changes (i.e., poor body image) cause depressive symptoms, or do the depressive symptoms create a poor sense of self (including poor body image). As is the case in many areas of psychology, it is difficult to know which factor drives the effect without experimental manipulation of the variable of interest and random assignment of participants. Despite this, strong claims of directionality are sometimes made in this area (e.g., Fallon & Hausenblas, 2005). In view of practical constraints and the boundaries of primary interest in this study, body image will not be included as a variable of interest. It is mentioned purely to provide a comprehensive review of the literature.

Biological Factors

With respect to biological factors, Nolen-Hoeksema and Girgus (1994) base most of their discussion around puberty and the changes associated with it. During puberty, adolescents (both males and females) undergo dramatic transitions in their hormones, bodies, and social roles. Adolescence is defined as a period of transition characterized by accelerated processes of change in cognitive, social, and psychological functioning, accompanied by marked physical restructuring (Sieffge-Krenke, 1995). Contrary to the previous storm and stress models of adolescence, change during adolescence may not all translate into crises; rather, this transition into adolescence may be characterized as a "pileup" of stressful events (Coleman, 1989; Rudolph, 2002).

Seiffge-Krenke and Stemmler (2002) agree with Nolen-Hoeksema and Girgus (1994) that puberty, *per se*, is likely a very important factor to consider. Dorn, Susman, Nottelmann, Inoff-Germain, and Chrousos (1990) are another group of researchers who view pubertal changes (and their underlying hormonal substrates) as being implicated in mood changes associated with adolescence. In the literature, pubertal timing, pubertal

status, hormonal changes, and the psychological consequences that follow from such a transition have been investigated.

Pubertal Assessment

It is impossible to draw meaningful conclusions about puberty without having an appreciation of how the onset of puberty is measured (Coleman & Coleman, 2002). Various methods of assessment have been used in the past to measure the timing and status of puberty and all have advantages and disadvantages for specific purposes. Methods of assessing indicators of puberty affect the feasibility, reliability, and validity of the information gathered. Given the difficulty in measuring anthropomorphic characteristics (e.g., bone age, body fat), most studies have focused on the development of secondary sexual characteristics.

The gold standard in pubertal assessment is physician ratings of the five stages of development devised by Tanner (1962) and his colleagues at the Institute of Child Health. The stages range from pre-pubertal to post-pubertal development and delineate pubic hair growth in both boys and girls, breast development in girls, and genital development in boys.

Various methods have been used to illustrate and determine the Tanner stages for adolescents (e.g., photographs, line drawings, physician inspection, parental report, self-report, questionnaire) and have produced a wide pattern of results. Coleman and Coleman (2002) report that the most accurate ratings are gained from trained health professionals' inspection of adolescents. Brooks-Gunn, Warren, Rosso, and Gargiulo (1987) found that maternal correlation with physician ratings was fairly high at .85, however they noted that maternal reports of sons' development tended to be less accurate than reports of

daughters. Dorn et al. (1990) found that self-reports were slightly more accurate than parent ratings, but both were still less accurate than physician ratings. However, it is important to comment that they found approximately 50% of self-reports *were* accurate when compared to physician ratings, and even those that were inaccurate were not *dramatically* different (i.e., differed by only one stage rating).

A problem with many self-report studies is that the adolescent is asked to rate him or herself from memory, rather than from recent inspection. Although this method is sometimes useful to avoid ethical concerns, this process also seems to lower the accuracy of self-report. While Tanner stages may be quite informative in terms of visible pubertal development, they are not good indices of underlying hormonal changes (Brooks-Gunn et al., 1987). That is, there may be variability in hormone levels within the same Tanner stage. Thus, if information regarding hormones is also sought, an additional mechanism must be used.

Two questionnaires have repeatedly been used in the literature to provide self-report information about pubertal development. The Adolescence Scale (AS) devised by Kaiser and Gruzelier (1999) provides a retrospective account of pubertal development relative to other members of the same sex, age at menarche, age at voice break, age at first nocturnal emission, and age at regular shaving. Reliability estimates have been reported as .87 for women, and .85 for men. The brevity of the AS makes it an attractive alternative for situations where Tanner stages cannot be used and retrospective data are all that can be gathered. The Pubertal Development Scale (PDS; adapted from an interview format used by Petersen, Crockett, Richards, & Boxer, 1988) queries similar areas of development as the AS, but it is completed by the adolescent to provide an

accurate report of current development, rather than providing a retrospective report of past events. A more descriptive psychometric summary of the PDS is offered in the Measures section of this paper.

Although the AS and PDS may offer slightly less accurate methods of attaining pubertal development information than the physician rated Tanner stages, they are attractive alternatives in terms of time and money involved. Adolescent ratings of their own pubertal stage are generally noninvasive and can serve as reliable and valid indices of pubertal development (Dorn et al., 1990). They may also be appropriate for certain research questions in which an exact measure of pubertal status is not necessary (i.e., within one stage). The acceptability of these questionnaire methods is likely more attractive for school approval and perhaps even the adolescents themselves.

Pubertal Timing and Status

For some time the literature has focused on pubertal timing as an important factor in the relationship between sex and depression in youth. Steiner, Dunn, and Born (2003) define pubertal timing as the maturation of an adolescent relative to his or her peers. Stattin and Magnusson (1990) argued that early puberty has been associated with greater problem behaviors in girls, but with good adjustment in boys. They suggest that the impact of early puberty on the social lives of girls results in negative effects, but the impact on boys is positive. An NIMH study of puberty (Nottelmann, Susman, Inoff-Germain, et al., 1987) also found an association with *reduced* negative emotional tone in boys who matured early (as measured by hormone levels) versus an *increased* negative emotional tone in girls who matured early (measured in the same way). Quite simply, it

has been thought for some time that early maturation is good for boys but bad for girls in many respects, including depressive symptoms.

Recently, the focus has shifted from pubertal timing to pubertal status. Steiner et al. (2003) define pubertal status as the current level of physical development of an adolescent, relative to the overall process of pubertal change usually defined by a series of stages. The work of Angold, Costello, Erkanli, and Worthman (1999) and Angold et al. (1998) has been instrumental in this shift. This research indicates that it is the stage, more so than the timing, of puberty development that is associated with rates of depression. Angold et al. (1998) found that pubertal status better predicted the emergence of the expected sex ratio in depression than did chronological age. They reported that only after mid puberty were girls more likely to be depressed. The timing of this transition (e.g., early, on time, or late) had no effect on depression rates. Given that they found an increased risk for depression during mid puberty for girls, it may imply that hormonal influence is minimal since many hormone changes would have already occurred by this point (see the hormones and endocrinology section for an alternative explanation).

Piccinello and Wilkinson (2000) also commented on other studies that found pubertal status to be a better predictor of the emergence of depression and other psychopathologies (e.g., panic attack occurrence, eating disorder symptoms). Nottelmann, Susman, Dorn, et al. (1987) indicate that pubertal processes and events should relate more closely with each other than with chronological age, although the pubertal processes do not always occur in synchrony. This trend seen from Angold and others may indicate a more important role for pubertal status in the development of disorders in general than had previously been thought.

Hormones and Endocrinology

Gonadal hormones have previously been reported to have limited influence on the emergence of depression (accounting for less than 4% of the variance, Brooks-Gunn, Graber, & Paikoff, 1994), with other biological and environmental factors being more prominent. While it has been concluded (perhaps erroneously) that hormones do not play a direct role in explaining the variance in negative affect, research by Angold et al. (1999) supported the hypothesis that female hormonal levels mediated girls' increased levels of depression. These same authors suggest that sex differences exist possibly in responsivity to hormones and not just in the actual hormones that are present. It is suggested that the genetic predisposition to depression and stressful life events may be "switched on" at puberty in females (Silberg et al., 1999). Little consideration has been given to identifying how social factors may influence the hormonal system, especially environmental stressors. Even though direct hormonal effects have been small, the fact that they demonstrate stability and have interacted with psychological and social factors implicates hormonal changes at puberty at least as part of the cause.

Research is now accumulating that indicates female reproductive endocrinology is likely to be an important factor in the higher rates of depression in women compared to men (Wolk & Weissman, 1990). Associations have been found between females and affective states premenstrually and during postpartum (Ahokas, Kaukoranta, Wahlbeck, & Aito, 2001; Steiner et al., 2003), during and after menopause (Birkhauser, 2002; Miller, Conney, Rasgon, Fairbanks, & Small, 2002), and with the use of oral contraceptives (Oinonen & Mazmanian, 2002). Indirect support for the association of endocrine systems and depression with women comes from the fact that the higher rate of

female depression is not evident in prepubertal children (Fleming & Offord, 1990) and tends to drop off during old age (Wolk & Weissmann, 1990). It seems to be the case that once hormones become activated and until they drop off in intensity that the sex ratio shift in rates of depression is significant.

Given that this line of research (endocrinology and mood disorders) has highlighted important links with women, it may be that similar endocrine system changes are also associated with affective states with younger females. In fact, Steiner et al. (2003) suggest that the sudden appearance of higher levels of estrogen during puberty (the relevant time during a child's life that hormone shifts occur) may alter the sensitivity of neurotransmitter systems for females. In fact, many researchers consider the pre-post menarchal change during puberty as *the* significant event that explains sex differences (Born et al., 2002). Others suggest that it is changes even earlier in life that predispose females to mood changes later in life. Specifically, subtle organizational effects in the prenatal environment may prepare the fetus' endocrine system for postnatal diseases including depression (Papaioannou, Gerozissis, Prokopiou, Bolaris, & Stylianopoulou, 2002; Sloboda, Newnham, Moss, & Challis, 2006). This line of research is referred to as the "fetal origins hypothesis of adult disease" (Welberg & Secki, 2001).

Angold et al. (1999) suggest that causal explanations of depression in females need to focus on factors associated with changes in hormone levels rather than on the morphological changes of puberty. They suggest that changes in rates of depression in adolescence *are* related to the physical changes of puberty, however, the hormone changes underlying the physical changes may be *more important*. They hypothesized that the inclusion of hormonal measures in models with Tanner stages would reduce the

apparent effect of Tanner stages on depression. Their results showed the effect of pubertal morphological status on depression was eliminated by the addition of hormone variables to models of depression. There was consistent support for effects of both testosterone and estrogen in the increasing prevalence of depression. Specifically, findings showed that NA was associated with higher levels of androgens and estrogens in adolescent girls. The data indicate that the pubertal increases in these hormones surpass a threshold at which females are rendered more susceptible to depression. While the hormones may put females at increased risk, it may be that other factors that interact are required to explain the development of individual episodes of depression. This interpretation suggests that rising testosterone and estrogen levels potentiate risk for depression through mechanisms that are unrelated to psychosocial effects on body morphology.

Nottelmann, Susman, Dorn, et al. (1987) have reported that it is reasonable to compare research across and within studies based on different markers of pubertal stage and hormone concentrations (i.e., studies that wish to consider developmental processes would be served roughly equally well by markers of chronological age, physical growth, pubertal stage, or underlying hormone status). More specifically though, they found that the strongest hormone correlates of pubertal development for girls and boys were androgen levels and the strongest correlate for menarchal status in girls was estradiol level. They do recommend having separate measures for hormone levels and pubertal status (even though they are correlated) because there was considerable variability in hormone levels within pubertal stage increments and substantial overlap in hormone level distributions at contiguous pubertal stages.

Nottelmann, Susman, Inoff-Germain, et al. (1987) report that hormone levels are closely related to pubertal stage, however, they *may* influence psychosocial adjustment (in this project depressive symptoms) in ways that are independent of the physical changes associated with puberty. More specifically, there is a time lag between the rise in hormone levels and the first appearance and development of secondary sex characteristics (i.e., the physical changes). It is possible, therefore, that a change in hormone levels may have an immediate impact on psychosocial adjustment prior to the emergence of physical changes. They also suggest that the different pattern of relations for boys and girls may be the result of actual biological differences between boys and girls in the influence of hormones on behavior and that neuroendocrine mechanisms involved in modulating hormone-behavior interactions may be different for boys and girls (like the Different Factors Model, Nolen-Hoeksema & Girgus, 1994). Within this domain of research there is perhaps a tendency to attribute behavioral changes to biochemical processes underlying physical changes, in this case the hormone levels underlying the physical changes associated with puberty (Nottelmann, Susman, Inoff-Germain, et al., 1987).

Stress, HPA Axis, and Cortisol

Hormones have long been thought to play a role in women's depression because of the overlap in experiences or exacerbations of depression during periods when levels of hormones are undergoing significant changes (e.g., premenstrual phase of menstrual cycle, postpartum period, menopause; Nolen-Hoeksema, 2002). A time of significant hormonal shift for adolescents is puberty, and this is the time that the shift in sex ratios of depression is known to occur. Because of this overlap, and the support for the relationship between women's depression and hormonal changes, it makes it reasonable

to hypothesize that hormonal changes before, during, and after puberty may play a direct role in the increased emergence of adolescent depression in girls.

The hypothalamic-pituitary-adrenal (HPA) axis has been found to be important in the regulation of the human stress response in depressed individuals (Dahl & Ryan, 1996; Garber & Horowitz, 2002; Nolen-Hoeksema, 2002). Apparently the cortisol that is released from the adrenal glands interacts with hypothalamic peptides and pituitary releasing hormones to prepare the body to deal with stress. Some type of feedback mechanism is usually at work within this axis to prevent an excessive production of cortisol. The major hormonal changes that occur during puberty force the HPA system to adjust this feedback mechanism. During this adjustment process, external psychosocial stressors may be more influential (Steiner et al., 2003) perhaps because the HPA system has been suppressed (Dahl & Ryan, 1996). We know that between 60 and 80% of depressed inpatient adults have increased cortisol secretion (Thase, Jindal, & Howland, 2002), thus reflecting a dysregulation of the HPA response. Inconsistent findings have been reported with respect to cortisol levels in children and the association with depressive symptoms. There has, however, been a more consistent relationship found between blunted cortisol responses in depressed children and a dysregulation of the neurotransmitter serotonin (Garber & Horowitz, 2002; Steiner et al., 2003). Sloboda et al. (2006) along with Andrews and Matthews (2004) suggest that prenatal programming of neuroendocrine development and HPA axis function may, in fact, be regulated through the serotonergic system, although conclusive evidence of this predisposition remains to be found.

We know that concentrations of gonadal hormones are stable and low in prepubertal children. With the onset of puberty, females are exposed to surges of sex hormones (i.e., estrogens and progesterone). This hormonal fluctuation coincides with the change in sex ratio of rates of depression. Although gonadal hormones may explain some of the sex differences in depression, psychological and social factors also likely play explanatory roles. These factors have been investigated thoroughly in the literature, perhaps to the exclusion of hormonal and pubertal effects. The picture seems not so clear for boys due to inconsistent results in hormone studies (e.g., Ramirez, 2003). It is unclear if a genetic vulnerability is required to interact with the hormonal changes to produce depressive effects (Nolen-Hoeksema, 2002).

Also, we know that estrogen seems to be able to modulate serotonergic functions and may therefore play a role in the mechanisms associated with depression and its treatment (Joffe & Cohen, 1998; Stahl, 1998). While this has been purported to be true since the 1940s and some results have been mixed (Kumar et al., 2003), evidence seems to be converging on the importance of this relationship as an augmentation treatment strategy or as a primary treatment option (Epperson, Wisner, & Yamamoto, 1999). That is, estrogen may induce an antidepressant benefit by interacting with serotonin receptor sites in certain groups of women. This benefit can be quite rapid (i.e., within one week) even in treatment resistant patients (Ahokas et al., 2001; Cohen et al., 2003; Rasgon et al., 2002). This may highlight a potentially effective hormonal treatment for mood disorders in women. Garber and Horowitz (2002) hypothesize that developmental changes in the HPA axis may be responsible for this inconsistency in youth and may be able to provide some type of marker for depressive risk.

The purest method to gain information related to hormone levels is through blood or salivary samples (Cohen-Bendahan, van de Beek, & Berenbaum, 2005; Harris, Rushton, Hampson, & Jackson, 1996; Mead & Hampson, 1997) that are subject to endocrine laboratory testing (Dorn et al., 2003). The task associated with using this procedure to get the most accurate measures of hormone levels is onerous, whether using adult or adolescent samples. Access to research participants is hindered, medical professional involvement is sometimes required, greater costs are generated, laboratory testing and equipment is necessary, and greater commitment from participants is required (e.g., repeated testing sessions at specific times of day).

Biological influences on behavior occur both prenatally and postnatally. Those that occur prenatally are referred to as organizational effects and those that occur postnatally are referred to as activational effects (Beall, Eagly, & Sternberg, 2004). Activational effects occur in the adult brain rather than the developing brain, and are time-locked to the presence of active hormones in the bloodstream (Hampson & Moffat, 2004). In this case, hormones modify brain activity. Organizational effects on the other hand, influence a developing fetus in utero for a time limited period and cannot be undone (Hines, 2004). Activational effects do not negate the possibility of organizational effects or the possibility of nonbiological influences (Cohen-Bendahan et al., 2005; Papaiouannou et al. 2002). Often, genetic, social, developmental, cultural, and contextual factors interact to produce effects. Environmental factors can either accentuate or mitigate the difference induced by hormones. While both categories are important in research relating hormones to behavior, they are not equally accessible. That is, there is a limited critical period in which to get pure indicators of the prenatal biological

environment (i.e., in utero). Unless research is planned well in advance, organizational variables are hard to measure. Therefore, when a variable reflecting organizational effects is desired, it is common for researchers to select prenatal hormone indicators that are correlates of the prenatal hormonal environment and to infer results from those findings. In fact, Andrews and Matthews (2004) summarize recent epidemiological evidence that the fetal environment can profoundly influence susceptibility to disease later in life by altering feedback sensitivity during critical time periods. Thus, prenatal correlates can be particularly informative in many respects (Kajantie & Phillips, 2006). Given methodological limitations, it was not feasible to measure activational effects in this study. Therefore, organizational hormone effects are the focus of the current study.

Researchers interested in studying hormonal effects that are unable to acquire blood or salivary samples must, therefore, use indirect methods of gaining hormone information. Putative markers of hormonal levels that have been investigated recently include intrauterine position and otoacoustic emissions (Cohen-Bendahan et al., 2005), as well as fluctuating asymmetry (of foot size and finger ridges) and the ratio of 2^{nd} to 4^{th} digit (2D:4D) finger length (Sanders & Kadam, 2001). Relatively limited research exists that has investigated the first four markers. However, research is accumulating that suggests the latter may reflect prenatal hormone exposure and remain stable into adulthood (specifically testosterone and estrogen; Fink, Manning, Neave, & Grammer, 2004; Fink, Neave, & Manning, 2003; Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer, & Manning, 2004; Manning, Stewart, Bundred, & Trivers, 2004; Poulin, O'Connell, & Freeman, 2004; Williams et al., 2000). A recent meta-analytic review (Honekopp, Bartholdt, Beier, & Liebert, 2007) that evaluated 17 samples with a more than 1100

participants offers more confidence that 2D:4D is a suitable tool to study the effects of prenatal androgenization on human behavior. While the research base supporting 2D:4D as an indirect prenatal hormonal marker may be building, there are still some studies that fail to show significant correlations between this variable and several traits thought to be related to sex hormones (Putz, Gaulin, Sporter, & McBurney, 2004; van Anders & Hampson, 2005).

Three of the above characteristics relate to distal portions of the limbs and are thought to be determined in utero (i.e., finger length ratio by the 14th week and finger ridges by the 16th week; Holt, 1968; Putz et al., 2004; Sanders, Sjodin, & de Chastelaine, 2002). Animal research has found that prenatal development of gonadal hormones is genetically linked (via the *Hox* gene) to the development of the hands and feet (Herault, Fradeau, Zakany, & Duboule, 1997) and Manning, Scutt, Wilson, and Lewis-Jones (1998) further report that the *Hox* genes are required for the growth and patterning of digits and the differentiation of the genital bud in humans. It is also around the 8th week of development that the male fetus begins to produce testosterone (Migeon & Wisniewski, 1998). These reports indicate that patterns of digit growth may be related to fertility and hormone levels.

While the above somatic markers are *generally* reported to be indirect correlates of prenatal hormone exposure (because most research using these markers has been retrospective), a recent research study (Lutchmaya et al., 2004) provides stronger direct evidence of such an association. These authors investigated finger length ratio using amniocentesis data to gain direct testosterone and estrogen levels in utero and compared that to finger length ratio of the same children at two years of age. Results supported an

association between finger length ratio and prenatal hormone exposure. More specifically, the results indicated that a low 2D:4D (i.e., the 2nd digit [index finger] is shorter in length than the 4th digit [ring finger]) ratio in males was associated with high prenatal exposure to testosterone and lower prenatal exposure to estrogen, and a high 2D:4D ratio in females was associated with low prenatal exposure to testosterone and higher prenatal exposure to estrogen. The negative relation between 2D:4D and testosterone and the positive relation between 2D:4D and estrogen has been found in other research as well (Fink et al., 2003; Manning & Taylor, 2001; Robinson & Manning, 2000). Manning et al. (1998), who evaluated 800 participants from ages 2 to 25 years in a cross-sectional study, suggest that it may be the case that 2D:4D affords us a glimpse into the prenatal hormone environment and shows little change at puberty.

Relatively few studies investigating 2D:4D have used it in comparison to mental health outcomes. Martin, Manning, and Dowrick (1999) are an exception that found asymmetry in digit length was a significant predictor of Beck Depression Inventory (BDI) scores in men.

With respect to finger ridge count patterns, it seems that the majority of people have a higher finger ridge count on the right hand and a minority of people have a higher finger ridge count on the left hand (Holt, 1968). Research by Sanders and Kadam (2001) has found that prepubescent children with higher ridge counts on their right hands has been associated with male typical performance on sexually dimorphic tasks, and that prepubescent children with higher ridge counts on their left hands has been associated with female typical performance on sexually dimorphic tasks. Given that testosterone has been implicated in other scenarios when sexually dimorphic differences exist

(Krahnstover-Davison & Susman, 2001; Manning & Taylor, 2001), it may be the case that testosterone is also implicated here with respect to finger ridge count differences (which are determined prenatally). This explanation has also been suggested by Jamison, Meier, and Campbell (1993) as well as Sanders et al. (2002) who found that higher testosterone was associated with higher total ridge counts. Cohen-Bendahan et al. (2005) have also suggested that if testosterone, in fact, stimulates production of epidermal and nerve growth factors, then dermatoglyphics may serve as a window into the prenatal environment. This outcome remains uncertain.

Manning (2002) notes that 2D:4D ratio may be correlated with finger ridge count given the early determination of this variable as well as the sexual dimorphism findings. And in fact, that is just what has been found in some of his research with very low birth weight children. That is, low 2D:4D ratios have been significantly associated with high ridge counts in these children who have experienced high prenatal stress. It is unclear if the findings are consistent in other samples in which less prenatal stress has occurred. However, Jamison et al. (1993) also found results that suggest prenatal testosterone is significantly associated with asymmetries in ridge counts in adult men. It seems reasonable, at least, to investigate this variable further.

Despite the plethora of research that is currently being conducted that involves 2D:4D ratios, few researchers have considered the relation of this variable to mental health conditions. Manning (2002) and Manning, Baron-Cohen, Wheelwright, & Sanders (2001) are two exceptions. These authors have investigated the association between 2D:4D and depression and autism, respectively. Manning (2002) suggests that lower 2D:4D ratios (more common in men) are associated with higher levels of depression in

men. This interpretation seems to run counter to the common finding that women (who typically have higher 2D:4D ratios) have higher prevalence rates of depression than men. No known published literature has addressed the possible association between 2D:4D and depression in youth. Manning et al. (2001) suggest that 2D:4D may be a possible marker for autism through the implication of prenatal testosterone in its etiology. More specifically, these authors found that children with autism have lower than expected 2D:4D ratios. No known published research exists that considers the relation of finger ridge counts and depression with samples of any age. Obviously, with so few studies completed to evaluate this issue, more studies must be conducted. Any investigation of these variables would be truly exploratory at this point.

In summary, while blood or salivary samples would be the ideal method to gain information related to hormonal levels (specifically testosterone and estrogen, Harris et al., 1996; Mead & Hampson, 1997), indirect measures do exist that are attractive alternatives in many respects. If prenatal hormone indicators can be accessible, they may provide a rich opportunity to unravel early transactions between biology and the social environment (Cohen-Bendahan et al., 2005). It is important to clarify that neither 2D:4D ratio nor finger ridge count have been conclusively linked with current hormone levels, and that is not the aim of this project. In this project, both 2D:4D ratio and finger ridge count will be used as indirect markers of prenatal hormone exposure.

Summary

It is clear that depression is a mental health issue that has increased dramatically in prevalence over the last few decades and continues to increase (Fleming & Offord, 1990). Sex differences in prevalence rates tend to emerge during adolescence (Broderick,

1998; Garber & Horowitz, 2000). Despite the plethora of investigations that have examined sex differences in this domain, a clear understanding has not been achieved. Given that the course and prognosis of depression in childhood predicts likelihood of relapse and continuity into adulthood (Galambos et al., 2004), it is important to find answers to many of the questions mentioned in the beginning of this paper. Perhaps the foremost question to answer should be whether it is appropriate to adopt a downward extension of adult theories with respect to depression in youth (Kaufman et al., 2001). The answer to this question will inform many others.

This project takes a developmental approach to the investigation of depression in youth. That is, it examines particular issues (e.g., changes associated with puberty and ruminative coping) that are relevant to adolescents that may also be associated in some meaningful way to the development, maintenance, or expression of depression. Some important developmental transitions, such as puberty, may be overlooked if a downward extension of adult theories of depression is assumed to be equally applicable for youth with depression. This project seeks to demonstrate that it will be necessary to adopt a developmental examination of depressive factors relevant to youth and that adjustments to assessment and treatment alternatives currently in use for youth may require modification.

The work of Nolen-Hoeksema and Girgus (1994) seems to be the starting place for most research that favors a developmental approach. Three models that attempt to explain gender differences in depression were presented, as well as categories of variables that illustrate each model. Some inconsistencies in the literature were identified with respect to each factor and with respect to patterns found in adults versus youth.

Significant discrepancies have been found in the psychobiological literature. Many of these may be the result of different methodologies used. It is promising to see that recent findings are beginning to converge.

The factors of interest for this project include: stressful life events in parent and peer domains, ruminative coping, pubertal status, and prenatal hormone exposure. Factors that occupy primary attention in this project include: ruminative coping, pubertal status, and prenatal hormone exposure. As such, primary hypotheses will relate to those factors, while supplementary hypotheses will relate to the more peripheral factors (life events in two domains).

Limitations to previous approaches were highlighted. First, few researchers have investigated proposed factors across more than one category of variables (i.e., social challenges, personality risk factors, and biological challenges) in a comprehensive manner. Exceptions to this were reviewed. Second, most research presented findings with respect to depression without distinguishing the roles of NA and PA. Third, few studies did evaluate the substantial comorbidity that exists between depression and anxiety. Fourth, studies examining some issues were confounded by poor measures. Alternatives with stronger psychometric properties were introduced in this review. This study will expand on past studies by incorporating strategies to overcome these limitations.

It seems that most researchers who examine adolescent depression are certainly aware of the alternative theories that exist. Almost all articles that speak to the sex difference issue in depression at least acknowledge that rumination has been implicated in some fashion quite consistently and that the shift coincides with pubertal events. Despite the fact that many researchers usually summarize their findings with the

appreciation that multiple factors likely interact to produce depressive symptoms (i.e., genetic vulnerability interacts with situational events), few researchers have tended to compare the personality risk factor of rumination directly with the biological challenge of puberty or hormonal exposure.

Seiffge-Krenke and Stemmler (2002) are an exception to the above point, whom do not view the literatures of these topics as opposing, but instead view them as yet unconnected by many researchers. These authors have made attempts to expand and unify findings that have previously been interpreted as conflicting. Reise and Henson (2003) have highlighted that it is, in fact true, that many personality constructs are deeply embedded within psychobiological theories. It is therefore not a large inference to hypothesize that the psychological factors referred to in this study may be intimately linked with the biological factors described. Piccinelli and Wilkinson (2000) suggest that such an investigation into gender differences in depression can assess the relative importance of both risk factors from the domains discussed above. It will be the aim of this project to directly and comprehensively compare competing theories of depression in youth to identify the most sensitive time periods and factors that are implicated in the development of depression in adolescents.

Hypotheses

1) High scores on depressive symptoms will be associated with youth who use a ruminative coping style. It is predicted that this effect will be stronger in girls. This hypothesis will evaluate the RST in an adolescent sample.
2) Girls and boys will demonstrate different dominant coping styles. Specifically, girls will use a ruminative coping style, whereas boys will use a distraction coping style.

This hypothesis will evaluate the sex differences component of RST in an adolescent sample. Although Hypotheses 1 and 2 are not new ideas that are being tested, they are being investigated with a new coping measure that will attempt to overcome past interpretive difficulties and more clearly delineate the use of this mechanism in youth.

3) Positive affect will be negatively related to ruminative coping. That is, lower positive affect scores will be significantly associated with higher rumination scores. This hypothesis includes a component of the tripartite model of depression that is frequently neglected in the sex differences line of research and will evaluate the relationship with RST in an adolescent sample.

4) Pubertal status will be a better predictor than chronological age of the sex shift in depressive scores. Specifically, pre-puberty adolescents (both boys and girls) will have lower depressive scores, while mid-post puberty girls will have higher depressive scores compared to boys. Tanner stages 1-2 will be used to indicate pre-puberty, while Tanner stages 3-5 will be used to indicate mid-post puberty (Born et al., 2002). This hypothesis has been tested in the past and has resulted in conflicted findings (i.e., pubertal status accounts for little variance vs. significant variance). Refining this issue and directly comparing it to Hypothesis 5 will attempt to clarify the role of puberty in the etiology of depression in youth.

5) There will be a significant relationship between an indirect measure of prenatal hormone levels and depressive symptoms after controlling for age, sex, and pubertal status. Building on the research that has found estrogen to be an effective augmentation treatment strategy for depression in adult women, it is hypothesized that lower levels of estrogen are associated with higher levels of depression in this

adolescent sample. Following this logic, it is also hypothesized that finger ridge count (proposed to be positively correlated with testosterone) is positively associated with higher levels of depression in this adolescent sample. Although change in hormone levels has been tested through other significant life events (e.g., postpartum depression, menopause), it has not been investigated thoroughly in an analogous process that occurs during adolescence (only studies by Angold's group [1998, 1999] have considered this). Therefore, this hypothesis will attempt to combine information from relatively distinct domains in the literature.

Hypotheses 1 through 5 will allow a comparison across theories involving rumination, puberty, and hormones.

Supplementary Hypothesis:

6) There will be differences in depressive symptoms depending on the level of stress experienced in both the parent and peer domains. More specifically, if stress is associated with both domains, depressive symptoms are expected to be higher than if stress is associated with only one domain or is low across both domains. This hypothesis has not been sufficiently investigated in the past with youth samples. Rationale for this hypothesis then, comes from the adult literature.

Method

Participants

Participants were recruited through local middle and high schools with permission from the school board, the principals, and teachers. Nine schools (three high schools and six elementary schools) allowed the primary investigator to contact parents of students in their school by letter. Those students whose parents provided consent were eligible to

participate[3]. In addition, all students had to provide their own consent prior to taking part in the study. One student declined to participate despite parental consent having been provided. In total, 213 students participated (see Table 1 for sample statistics). The sample was comprised of 100 males (46.9%) and 113 females (53.1%), with an age range of 12 to 19 years (full sample mean = 14.16, *SD* = 1.75; male mean = 14.32, *SD* = 1.64; female mean = 14.02, *SD* = 1.84). Grades 7 through 13[4] were represented. This age range was chosen because it adequately represents the window in which sex differences associated with depression so frequently emerge (Garber & Horowitz, 2000; Lewinsohn, Rohde, & Seeley, 1998; Steiner et al., 2003). Also, given that the mean age of menarche is 12.5 years, adolescents who both have and have not passed through puberty were likely to be included due to normal variation around the mean (Born et al., 2002). Of the 113 females included in the study, 92 reported to have already started menarche and 16 had not[5]. Table 1 shows the distribution of the sample by Tanner category and sex. Mean Tanner stages were 2.80 (*SD* = .53) for males and 3.21 (*SD* = .56) for females. Tanner stage three denotes mid-puberty and individuals across puberty levels were sampled. With respect to ethnicity, some diversity was represented with individuals identifying themselves as Aboriginal/First Nations (5.7%), African (0.5%), Asian (1.4%), Caucasian (75.6%), and Other (16.7%).

Procedure

Ethics approval was granted from the Human Ethics Committee of the Department of Psychology, the Lakehead University Senate Research Ethics Board, and

---

[3] One student provided his own consent in place of parental consent because he was over 18 years of age.
[4] Grade 13 is no longer a recognized grade in the Ontario school system. It is surmised that the one individual who noted 13 as his grade was undertaking an upgrading year.
[5] Five females had missing data for the menarche item and it was therefore unclear if they were pre- or post-menarchal

Table 1

Sample Size by Age, Grade, and Tanner Category Across Sex

		Male (%)	Female (%)	Total (%)
Age				
	12	8 (3.8)	20 (9.4)	28 (13.1)
	13	27 (12.7)	40 (18.8)	67 (31.5)
	14	32 (15)	23 (10.8)	55 (25.8)
	15	12 (5.6)	6 (2.8)	18 (8.5)
	16	8 (3.8)	4 (1.9)	12 (5.6)
	17	6 (2.8)	12 (5.6)	18 (8.5)
	18	6 (2.8)	8 (3.8)	14 (6.6)
	19	1 (0.5)	0 (0)	1 (0.5)
	Total	100 (46.9)	113 (53.1)	213 (100)
Grade				
	7	19 (8.9)	37 (17.4)	56 (26.3)
	8	39 (18.3)	42 (19.7)	81 (38)
	9	15 (7)	7 (3.3)	22 (10.3)
	10	9 (4.2)	4 (1.9)	13 (6.1)
	11	5 (2.3)	8 (3.8)	13 (6.1)
	12	12 (5.6)	15 (7)	27 (12.7)
	13	1 (0.5)	0 (0)	1 (0.5)
	Total	100 (46.9)	113 (53.1)	213 (100)
Tanner				
	1	1 (0.5)	2 (1)	3 (1.5)
	2	26 (12.7)	11 (5.4)	37 (18.1)
	3	62 (30.4)	60 (29.4)	122 (59.8)
	4	10 (4.9)	32 (15.7)	42 (20.6)
	5	0 (0)	0 (0)	0 (0)
	Total	99 (48.5)	105 (51.5)	204 (100)

the Lakehead Public School Board. After ethics approval, the primary researcher attempted to contact all principals in the city and area schools. An overview of the research project was offered to all principals by telephone, and those interested in learning more met with the primary investigator in person. A full description (verbal and written) of the project was provided during the face to face meetings and outstanding questions were clarified. All questionnaires and procedures were deemed acceptable by all principals involved in the study. All principals who agreed to a meeting also agreed to participate conditional on teachers, parents, and students also demonstrating interest and providing their own consent. Cover letters and consent forms for parents (see Appendix A) were distributed by the principals to teachers who indicated interest in the study and who had students in the proposed age range. Teachers then forwarded the consent forms to parents by way of the students. It is uncertain how many consent forms were actually sent out to parents because not all unused consent forms were returned by the principals or teachers. It is appropriate to say that the response rate was not uniform across schools or classes. Multiple attempts were made at each school to recruit additional students during subsequent rounds of data collection. Only 15 parents did not permit their child to participate. Those students whose parents did not provide explicit written consent were not permitted to participate.

Once parental consent was obtained, arrangements were made for data collection to occur on site either in the classroom or other designated space in the school (e.g., library, science lab). Individual site preferences dictated the schedule of data collection (e.g., a full day with multiple classes throughout the day, separate classes on different days). Students also provided their own consent to participate in the study (see Appendix

B), and this was collected by the primary investigator on the day of data collection prior to commencing the process. The same instructions were given to all participants.

Data were collected through questionnaire (see Appendices C-H), and physical measurements and prints of the hands were taken. No identifying information was recorded on the questionnaires. All questionnaires were completed by the youth in a group format and the researcher was available to provide additional assistance as necessary (e.g., clarifying instructions, providing word definitions for unfamiliar terms). Of the questionnaires administered, the scale used to assess depressive symptoms was administered to students second to last to minimize any effect that focusing on depressive symptoms might have had on responses to the other measures included in the package. A measure of social desirability was administered last to distract the youth from focusing on any depressive symptoms that may have been reported. All students began the questionnaires at the same time, and they were individually called on (by number) to provide the hand measurements.

Physical measurements of hands (length of the second and fourth digits) and fingers (prints of the first and fifth digits) were taken by the primary researcher. Hand scans were made using a portable photocopier (HP PSC1510 model) and finger prints were taken with an ink press pad (Trodat 9062 black ink). All jewellery worn on the hands of participants was removed prior to the hand measurements. Pen marks were made on both hands of each participant at the basal crease of the second and fourth digits to ensure consistent measuring. Some creases do not show up well on photocopies, and adding pen marks can contribute to more consistent measurements (Bailey & Hurd, 2005). In cases where a band of creases existed on the hand, the most basal crease was

used (i.e., the crease that was furthest from the finger tip and closest to the palm). Every fifth student had each hand scanned as well as measured directly, by the primary researcher using a digital calliper (Mastercraft electronic calliper with digital display, allowing accuracy to the nearest .02mm), to ensure reliability across measurements. A digital calliper was also used for measurements taken from the photocopies. The calliper was reset to zero each time a measurement was made. While some research has established that reliability of measurements taken with photocopy scans of hands yields high intraclass correlation coefficients when compared to measurements taken in real time (Manning et al., 2000; Robinson & Manning, 2000), other studies have cast doubt on this equivalency (i.e., tendency of photocopy scans to reveal lower 2D:4D ratios; Manning, Fink, Neave, & Caswell, 2005). Two sources of measurement are perhaps most ideal for research purposes (Manning, 2002), as long as comparisons are not made across methods. Each real time measurement and every fifth hard copy scan measurement was taken twice to reduce measurement error.

Finger prints were taken using inkpads and pressed onto Domtar coverstock (white, 67lbs.)[6]. Antibacterial wet wipes were provided to all students upon completion of having the hand measures taken to remove the ink residue that remained after pressing their fingers onto the card stock. All students completed the study within 30 to 60 minutes.

Students were debriefed upon completion of the study both orally and through the provision of a handout (see Appendix I) that provided contact information for the researchers and other community supports if they required further assistance. No students

[6] Prior to data collection, the local police department was consulted on the procedures they use for taking finger prints. Thickness of paper and practice taking prints were the only special indications noted. All efforts were made to be consistent with those techniques.

indicated to the researchers or other adults involved in this process that they were distressed by participating in any part of the study, and no contacts were made to the primary researcher or supervisor after the study was completed. Students were compensated for their participation by having their names entered into a draw to win one of four gift certificates for the local mall valued at $25.00 each. At the end of June 2006 the prizes were drawn. All student consent forms were gathered and numbers were assigned based on sequence in the pile. Online software (Haar, n.d.) with a random number generator was used to select four numbers. Prizes were delivered in a sealed envelope with the students' name on the front to the principals, who then forwarded the certificates to the winners.

After completion of the study, the hardcopy fingerprint cards were scanned into PDF documents using a Canoscan LiDE 60 scanner for storage in case the quality of the prints reduced over time from aging. The Henry classification system as described by Holt (1968) was used to count finger ridges. More specifically, it was the number of dermal ridges that intersected the line between the core and tri-radial points (i.e., the meeting place of three ridges that form an angle of approximately 120 degrees) that was considered to be the ridge count for each print. To assist with this process, equipment in the Advanced Technology and Academic Centre (ATAC) on Lakehead University's campus was used. Aligning the finger prints under the document camera (Samsung Digital Presenter [SDP-900DX]) allowed each individual print to be displayed on a plasma screen with a 12X power zoom lens with 850,000 pixels CCD. The ridge counts were made from the plasma screen that allowed each dermal ridge to occupy the full screen and be viewed more clearly.

Measures

Demographic Information

A short demographic questionnaire was developed by the primary researcher (Appendix C). Items queried involved the following: chronological age, grade, sex, cultural background, medication use, family history of mental health problems, family members, broken or fractured fingers, and handedness (defined as a continuous variable). The Edinburgh Handedness Inventory (Oldfield, 1971) and research studies (McFarland & Anderson, 1980; McMeekan & Lishman, 1975; Salmaso & Longoni, 1985) over the last three decades served as guides in selecting the most discriminative items for the handedness variable. Participants reported which hand he/she used to complete the seven tasks. Responses were rated on a five point Likert-type scale of left (1) to right (5).

Children's Depression Inventory

Kovacs (1992) developed the Children's Depression Inventory (CDI) as a response to the need for a standardized self-report measure of depressive symptoms for younger age groups (i.e., ages 7 to 17). It is important to clarify that this scale measures depressive symptomatology (a state) rather than clinical depression per se. This scale concerns the consequences of depression in contexts that are relevant to children (e.g., school) and at reading levels that are appropriate (i.e., grade 1 reading level required). There are 27 items and five factors that comprise the scale: negative mood, interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem. Each item contains statements that increase in symptom severity and children are asked to select the one that best describes how they were thinking and feeling in the last two weeks. A short and long form are available, and the short form correlates well ($r = .89$) with the full form (Kovacs,

1992). Both forms have demonstrated acceptable levels of reliability in the past, however, the shorter form is sometimes more acceptable to use in school environments because the suicide item is removed (Abela et al., 2002)[7].

Cronbach's alphas for internal consistency of the whole measure, found by the test author and other researchers in various studies, range from .71 to .89 (see manual for summaries of those studies), and the alphas for the subscales ranged from .59 to .68 (Kovacs, 1992). Test-retest correlations are considered less important in this measure given that it aims to measure a state construct rather than an unfaltering trait construct. However, various studies (see Kovacs, 1992) have indicated test-retest correlations ranging from .38 (rather low) to .87 for intervals between one week and one year.

With respect to validity, the manual presents limited information in terms of numbers, however refers to an annotated bibliography that cites literature that has considered various types of validity with the CDI (e.g., convergent, discriminant, concurrent, and criterion). The overall conclusion has been that the CDI has received good support to suggest that it assesses important constructs in the characterization of depressive symptoms in youth (Kovacs, 1992).

Revised Children's Manifest Anxiety Scale

Given the significant overlap among the constructs of depression and anxiety, it is important to have a measure of each variable. The Revised Children's Manifest Anxiety Scale (RCMAS), developed in 1978, served this purpose for anxiety. This measure provides a total anxiety score, three factor based subscales of anxiety (physiological anxiety, worry/oversensitivity, social concerns/concentration), and a lie scale. It is

[7] For the purposes of this project, the long form of the CDI was used with the suicidality item removed. Raw scores were used in the analyses rather than normed scores. This was deemed more appropriate given that some of the sample exceeded the ages upon which the scale was normed.

suitable for children and adolescents between the ages of 6 and 19 years and language levels are appropriate. Higher scores represent greater levels of anxiety experienced by the child.

The test manual (Reynolds & Richmond, 1997) provides a summary of the standardization procedure as well as the psychometric properties of the scale. Internal consistency estimates have been reported to range between .76 and .85 for total anxiety scores, and between .62 and .79 for the subscales. Test-retest reliabilities for the total anxiety scores have been reported to range from .68 to .98. Factor analytic studies have been used to demonstrate construct validity of the RCMAS. Repeated studies have found support for the structure offered by the authors of the measure. Convergent validity and divergent validity were supported by expected correlations being revealed with similar or dissimilar measures, respectively (e.g., State-Trait Anxiety Inventory for Children). Overall then, the RCMAS has demonstrated adequate reliability and validity in past studies and is therefore considered sufficient for the purposes of this project.

Children's Response Styles Scale

The Children's Response Styles Scale (CRSS, see Appendix D) is a self-report measure of response style for use with children that was developed by Ziegert and Kistner (2002). Based loosely on Nolen-Hoeksema's RSQ for adults, the content was adapted for children and also modified to evaluate response styles in general, rather than response styles specifically reflective of depressive statements. In this way, response style and depressive symptomatology are not confounded in this measure (i.e., item content does not overlap with common depressive symptoms). No upper and lower age restrictions have been noted for the scale.

Ziegert and Kistner evaluated this measure in terms of validity by comparing it with scores on the Children's Responses to Hypothetical Events Questionnaire (CRHEQ) and the Children's Engagement Scale (CES). Correlations between factor scores on these measures provided support that the CRSS demonstrated adequate convergent (i.e., CRHEQ and CRSS) and divergent validity (i.e., CES and CRSS rumination subscale).

With respect to reliability, internal consistency estimates (Cronbach's alpha) were .81 for the rumination subscale, and .88 for the distraction subscale. Item-total correlations on the rumination subscale ranged from .32 to .61, whereas item-total correlations on the distraction subscale ranged from .46 to .70. Test-retest reliability of three weeks was found to be .69 for the rumination subscale, and .74 for the distraction subscale.

Overall, the CRSS successfully demonstrated adequate indices of reliability and validity in a preliminary investigation. A two-factor solution supported the notion that the scale was in fact measuring two independent factors (i.e., rumination and distraction).

Pubertal Development Scale

The Pubertal Development Scale (PDS; adapted from an interview format used by Petersen et al., 1988, see Appendix E) is a self-report measure that assesses various aspects of development through adolescence. Specifically, it queries a current account of growth spurt, body hair, and skin changes in all adolescents, facial hair growth and voice change in boys, and breast development and menarche in girls. A numerical score is coded for each indicator based on current status (i.e., not yet begun = 1, barely started = 2, definitely underway = 3, completed = 4). Menarche is coded dichotomously (i.e., premenarche = 1, postmenarche = 4). All items are summed and then averaged to obtain

an overall PDS score. Reliability estimates (i.e., coefficient alpha for internal consistency) have been reported to vary between .68 and .83 (Peterson et al., 1988). With respect to criterion validity, the PDS compares fairly well (*r*s = .61 to .67) with physician ratings of Tanner stages (Brooks-Gunn et al., 1987), and interviewer ratings of the child's pubertal status (*r*s = .41 to .79). The PDS has become one of the most widely used measures of pubertal development (Dick, Rose, Pulkkinen, & Kaprio, 2001) and has been used successfully in many studies that address issues of pubertal development. It can be used with adolescents who have or have not already passed puberty milestones (no age restrictions noted).

Petersen et al. (1988) suggest that the availability of such a self-report measure is important for studies where direct measures of puberty may not be possible (e.g., within schools). One advantage it has over other measures is that is allows for the characterization of boys development in addition to girls development. It also allows for consideration of the overall puberty process rather than on change in any one single characteristic. The originating authors of the PDS and their research suggest that young adolescents *are* able to make appropriate distinctions about the changes occurring in their bodies.

Problem Questionnaire

Seiffge-Krenke (1995) describes the Problem Questionnaire (see Appendix F) that was designed to measure adolescents' coping with minor events and everyday stressors across seven developmentally relevant fields. No upper and lower age restrictions have been noted. For the purposes of this study, only the scales for parent and peer stressors will be used. This will consist of a self-report measure containing 20 items that are rated

on a five point Likert-type scale from "highly stressful" to "not stressful at all". The Cronbach's alphas for internal consistency of the parent and peer subscales were reported as .84 and .83, respectively, by the test author.

Positive and Negative Affect Schedule – Children (PANAS-C)

The PANAS-C (Laurent et al., 1999, see Appendix G) is comprised of two scales that measure positive and negative affect. The positive affect scale has 12 items and reflects the extent to which a person feels enthusiastic, active, and alert. The negative affect scale has 15 items and reflects a general dimension of subjective distress and unpleasurable engagement. This version of the PANAS-C was modeled after the adult namesake (Watson et al., 1988) and was modified for use with school children (no age restrictions noted). It is proposed to measure two dominant and relatively independent dimensions of affect that emerged through factor analysis. Terms were specifically chosen for inclusion if they had at least average loadings (i.e., greater than .40) on the relevant factor and weak loadings (i.e., less than .25) on the other factor.

The test authors (Laurent et al., 1999) have reported on the psychometric properties of the scale and suggest that it is a brief, useful measure that can be used to differentiate anxiety from depression in school age children. They suggest that a conceptually appropriate intercorrelation of -.16 was maintained between the PA and NA subscales, and report that internal consistency reliability coefficients of .92 and .89 were found for the NA and PA scales, respectively. Support was also presented for convergent validity. Convergent validity was demonstrated through theoretically relevant and significant correlations with measures of depression and anxiety, Children's Depression Inventory and State-Trait Anxiety Inventory for Children.

The PANAS-C has been used in other studies with school age children (Kiernan, Laurent, Joiner, Catanzaro, & MacLachlan, 2002; Seligson, Huebner, & Valois, 2003) and has been found to perform appropriately. Similar to its adult counterpart, alteration of the time frame with which the child is to respond to the scale items does not alter the psychometric properties of the scale (Laurent et al., 1999).

Other scales that are based on the tripartite model were also considered for inclusion (e.g., the Mood and Anxiety Symptom Questionnaire [MASQ]). For various and compelling reasons (e.g., no adolescent norms have been developed for MASQ, MASQ is not available in the public domain, little research exists using the MASQ as a measure for NA/PA with adolescent student samples [Watson et al., 1995], substantial research exists using the PANAS-C with various adolescent samples [Laurent, Catanzaro, & Joiner, 2004]) it was determined that the PANAS-C would be most suitable for use in this study.

Children's Social Desirability Scale

Any research project that asks individuals to provide personal information (even if responses are not identifiable) may result in response biases (Carskadon & Acebo, 1993). There is no reason to believe that children would be exempt from this process (Crandall, Crandall, & Katkovsky, 1965). Paulhus (2002) has been instrumental in integrating and defining the constructs that relate to response biases. A response bias is any systematic tendency to answer questionnaire items on some basis that interferes with accurate self-reports. Socially desirable responding (SDR) is one type of response bias and is defined as the tendency to give overly positive self-descriptions. When a response bias is used consistently across time and questionnaires, it is referred to as a response style, however

when a response bias is short-lived and only a temporary distraction, it is noted as a response set.

In this study, given the personal nature of the information sought, we attempted to control for response bias. A questionnaire developed specifically for measuring SDR in children was used: Children's Social Desirability Scale (CSD, Crandall et al., 1965, see Appendix H). This scale was modeled after the Marlowe-Crowne scale (20 items were rephrased and 28 items were newly developed) and adjusted for age appropriate content, language, and experiences. Some of the items are framed in extreme language (e.g., always or never) and some of the items are framed in more occasional language (e.g., sometimes). An alternate format (i.e., Yes/No) exists for children under age 10, but no upper age restrictions are noted. Similar to the scoring criteria used in Crandall et al. (1965), the amount of socially desirable responding was indicated by the number of responses that suggest an undeviating attitude or behavior (i.e., endorsement of items that use extreme language). To minimize the presence of an acquiescent response set, approximately half of the CSD items were keyed in the true direction, and half in the false direction.

In terms of psychometrics, corrected split-half reliability coefficients ranged from .82 to .95 in the original sample of boys and girls in 1965. Test-retest reliabilities after a one-month interval revealed correlations of .90 in the original sample.

Finger Length Ratio

Photocopies of participants' right and left hands were obtained using a portable copier. Pen marks on the second and fourth digits at the basal crease assisted in measuring finger length consistently. Every fifth participants' hand was also measured

twice with a digital calliper to ensure accurate measurement. Participants who had injuries to second or fourth digits were excluded from statistical analyses using this variable.

Finger Ridge Count

Prints of the first and fifth digit (i.e., thumb and "pinky") for each hand were obtained from each participant. These digits were chosen for finger ridge counts because the middle fingers (i.e., index, middle, ring) have a higher incidence of arch patterns that result in a ridge count of zero (Sanders & Kadam, 2001). Ink pads were used to press marks of both fingers onto a sheet of paper. The Henry classification system as described by Holt (1968) was used to count finger ridges. Asymmetry was denoted if the total ridges for one hand (i.e., both thumb and "pinky") exceed the total for the other hand by at least two (as recommended by Sanders & Kadam, 2001).

Data Reduction and Statistical Analyses

To address the association between depressive scores and rumination (Hypothesis 1) and low PA and rumination (Hypothesis 3), two two-tailed Pearson product-moment correlation coefficients were conducted. One-way ANOVAs were used to evaluate whether sex differences existed in rumination, depressive symptoms, and affect scores. Two ANOVAs were used to test if different dominant coping styles (rumination and distraction) were used by boys and girls (Hypothesis 2). Regression analyses were conducted to determine if pubertal status was a better predictor of sex differences in depressive scores than chronological age (Hypothesis 4). The criterion variable was depressive score, the predictor variable was age, the proposed mediator variable was pubertal status, and the proposed moderator variable was sex. A hierarchical regression

was conducted to determine if indirect measures of prenatal hormone exposure were significantly related to depressive symptoms after controlling for age, sex, and pubertal status (Hypothesis 5). Multiple regressions were used to determine the contribution of parent and peer stress to depressive symptoms (supplementary Hypothesis 6).

Results

Descriptive Information

Prior to running the above analyses, the data were screened for univariate outliers using the criteria of a standardized score > $\pm$ 3.0 (Tabachnick & Fidell, 2001). Fourteen univariate outliers were identified and replaced with the next most extreme score in the distribution. Specifically, three scores from the CRSS distraction subscale were changed, one score from the PDS was changed, three scores from the PQ peer subscale were changed, four scores from the CDI were changed, and three finger ridge count scores were changed. Screening for multivariate outliers through Mahalanobis distance analyses resulted in only one participant being flagged. The scores for this participant were not altered because the variables which contributed to the scatter outside the centroid area were not main variables in the analyses. If further analyses are done with this data set, this approach should be reconsidered in terms of limiting generalizability of results for those particular subscales. Data were also evaluated for violation of multivariate assumptions as outlined by Tabachnick and Fidell (2001).

Missing values analysis revealed finger ridge count to be the only variable with more than 5% of the values missing (i.e., 26 and 27%, left and right fifth digits, respectively). Other variables in the data set contained between 0 and 4% missing values. Pairwise deletion was used as the analytic strategy to handle missing data (Tabachnick &

Fidell, 2001). Table 2 displays the means and standard deviations of the raw scores for all variables used in the analyses. Table 3 displays correlations across variables used in the analyses.

The measure of social desirability was normally distributed with mean and median values approximately equal and a skewness statistic of .54. However, the correlation matrix indicates that this variable was significantly related to some of the other variables being evaluated. Therefore social desirability was entered as a covariate in the ANOVA analyses for Hypothesis 2, and also controlled for in step 1 of the multiple regressions for Hypothesis 6. There were no sex differences on social desirability, t (209) = 1.23, $p > .05$.

Main Analyses

Hypothesis 1

The CRSS was used to evaluate coping style in the adolescent sample. It was proposed to be an unconfounded measure of rumination and distraction by the test authors. In other evaluations by the primary investigator (Welsh & Mazmanian, 2007) the CRSS has been shown to demonstrate acceptable indices of reliability (i.e., internal consistency's of .90 for the rumination subscale and .83 for the distraction subscale). The correlations with other variables in this study (see Table 3) are also supportive of concurrent validity of the CRSS.

Higher scores on CDI total scores were significantly associated with higher scores on ruminative coping ($r = .37, p < .01$). When the group was split by sex, this significant relationship was maintained by both sexes ($r = .42, p < .01$ for females and $r = .27, p < .01$ for males). When NA scores from the PANAS-C was used to represent depressive

Table 2

Means and Standard Deviations of Variables (Raw scores)

Variable	Subscale	n	Mean	SD
Handedness		210	4.32	0.94
CRSS	Rumination	212	5.66	2.03
	Distraction	212	6.48	1.64
Total		212	12.14	3.05
PDS		204	3.01	0.58
PQ	Parent	213	23.36	8.56
	Peer	210	20.59	7.92
Total		210	43.91	14.34
PANAS-C	Positive	211	42.75	10.00
	Negative	212	34.16	13.68
Total		211	76.86	13.74
RCMAS	Physiological anxiety	208	3.67	3.36
	Worry/oversensitivity	208	4.02	3.11
	Social concerns/concentration	208	2.65	1.75
	Lie	208	2.52	2.12
Total		208	10.20	6.01
CDI	A (negative mood)	211	2.55	2.24
	B (interpersonal problems)	211	0.89	1.06
	C (ineffectiveness)	211	1.95	1.91
	D (anhedonia)	211	3.28	2.88
	E (negative self-esteem)	211	1.56	1.88
Total		211	10.17	7.80
CSDS		211	16.53	7.56
Digit Length (scan)	Right 2	213	69.96	5.13
	Right 4	213	72.82	5.53
	Left 2	213	69.73	5.27
	Left 4	213	73.00	5.78
Ridge Count	Right 1	205	21.95	11.19
	Right 5	155	12.48	6.25
	Left 1	205	19.03	10.68
	Left 5	157	12.65	6.85

Note. SD = standard deviation; CRSS = Children's Response Styles Scale; PDS = Pubertal Development Scale; PQ = Problem Questionnaire; PANAS-C = Positive and Negative Affect Schedule – Children; RCMAS = Revised Children's Manifest Anxiety Scale; CDI = Children's Depression Inventory; CSDS = Children's Social Desirability Scale

Table 3

Correlation Matrix for all Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Age	-																		
2. Handedness	-.036	-																	
3. CRSS rum	.128	.009	-																
4. CRSS dis	.088	-.020	.376**	-															
5. CRSS tot	.133	-.005	.867**	.788**	-														
6. PDS	.494**	-.033	.262**	.062	.206**	-													
7. PQ par	-.056	-.011	.324**	-.078	.174*	.084	-												
8. PQ peer	-.019	-.004	.332**	-.077	.180**	-.068	.504**	-											
9. PQ tot	-.043	-.015	.385**	-.080	.213**	.026	.875**	.851**	-										
10. PANAS-C pos	.097	-.097	-.111	.291**	.082	-.054	-.227**	-.135	-.201**	-									
11. PANAS-C neg	.036	-.027	.401**	.020	.277**	.143*	.444**	.319**	.447**	-.360**	-								
12. PANAS-C tot	.108	-.101	.322**	.230**	.338**	.102	.277**	.216**	.298**	.369**	.734**	-							
13. RCMAS tot	.053	-.123	.526**	-.053	.323**	.041	.496**	.550**	.596**	-.239**	.535**	.365**	-						
14. CDI tot	-.061	-.065	.373**	-.145*	.171*	.110	.496**	.431**	.533**	-.345**	.494**	.246**	.696**	-					
15. CSDS	.012	-.036	-.188**	.174*	-.031	-.070	-.324**	-.096	-.245**	.108	-.210**	-.136*	-.293**	-.287**	-				
16. 2D:4D right	.040	.108	.083	-.100	.002	.115	-.078	.027	-.020	.004	-.024	-.019	.028	.046	-.024	-			
17. 2D:4D left	.047	-.007	.173*	-.075	.075	.131	.048	.134	.109	-.072	.097	.043	.162*	.146*	-.033	.722**	-		
18. FRC right	-.186*	-.076	-.151	-.047	-.122	-.103	-.002	.022	.014	-.070	.002	-.046	-.069	-.037	.201*	-.019	-.035	-	
19. FRC left	-.147	-.051	-.092	.000	-.060	-.044	.005	.042	.036	.028	-.009	.008	-.088	-.014	.187*	.049	.006	.770**	-

** significant at $p < .01$
* significant at $p < .05$

Note. CRSS rum = Children's Response Styles Scale Rumination Subscale; CRSS dis = Children's Response Styles Scale Distraction Subscale; CRSS tot = Children's Response Styles Scale Total; PDS = Pubertal Development Scale; PQ par = Problem Questionnaire Parent Subscale; PQ peer = Problem Questionnaire Peer Subscale; PQ tot = Problem Questionnaire Total; PANAS-C pos = Positive and Negative Affect Schedule – Children Positive Subscale; PANAS-C neg = Positive and Negative Affect Schedule – Children Negative Subscale; PANAS-C tot = Positive and Negative Affect Schedule – Children Total; RCMAS tot = Revised Children's Manifest Anxiety ScaleTotal; CDI tot = Children's Depression Inventory Total; CSDS = Children's Social Desirability Scale; 2D:4D right = Digit Ratio Right Hand; 2D:4D left = Digit Ratio Left Hand; FRC right = Finger Ridge Count Right Hand; FRC left = Finger Ridge Count Left Hand

symptoms, the significant relationship was maintained for the whole sample ($r = .40, p < .01$), as well as females ($r = .38, p < .01$) and males ($r = .36, p < .01$). Two one-way ANOVAs indicated there were significant sex differences in depressive scores. Specifically, females ($M = 11.17$, $SD = 8.58$) reported significantly higher depressive symptoms on the CDI than males ($M = 9.04$, $SD = 6.66$), $F(1, 209) = 3.98, p < .05$). Also, females ($M = 36.40$, $SD = 14.09$) reported significantly higher NA scores on the PANAS-C than males ($M = 31.65$, $SD = 12.81$), $F(1, 210) = 6.54, p < .05$).

Hypothesis 2

Females had significantly higher rumination tendencies (CRSSrum) than males ($M = 5.08$, $SD = 1.99$, $M = 6.17$, $SD = 1.93$, respectively), $F(1, 210) = 16.29, p < .05$, partial $\eta^2 = .07$, power = .98. However, males and females did not significantly differ in the use of distraction (CRSSdis) as a coping style, $F(1, 210) = .52, p > .05$, partial $\eta^2 = .02$, power = .11. With respect to total coping scores, females ($M = 12.73$, $SD = 2.73$) had significantly higher scores than males ($M = 11.47$, $SD = 3.26$), $F(1, 210) = 9.20, p < .05$, partial $\eta^2 = .04$, power = .86. When ANCOVAs were conducted with social desirability entered as a covariate for the above analyses, there were no changes in interpretation. That is, after controlling for social desirability, females still had significantly higher rumination tendencies than males ($F(1, 209) = 14.68, p < .05$, partial $\eta^2 = .07$, power = .97), there was no significant sex difference in the use of distraction ($F(1, 209) = .87, p > .05$, partial $\eta^2 = .00$, power = .15), and females had significantly higher total coping scores than males ($F(1, 209) = 8.81, p < .05$, partial $\eta^2 = .04$, power = .84).

Given that the sample also demonstrated sex differences in depressive symptoms and negative affect scores (see Hypothesis 1), the sex differences found here for rumination

may be due to overlapping constructs between rumination and depressive scores (as has occurred in previous studies) despite the effort to eliminate this confound with the new coping styles measure. The correlations in Table 3 demonstrated that CDI scores and PANAS-C NA scores were significantly related to rumination (.373 and .401, respectively). Therefore both depressive scores were also entered as covariates in an ANCOVA (in addition to social desirability) to remove their influence on the sex by rumination relationship. After controlling for all three of these variables, a significant sex difference in rumination remained, $F(1, 205) = 9.76, p < .05$, partial $\eta^2 = .05$, power = .88. As indicated by adjusted means, females ($M = 6.04$) reported higher rumination tendencies than males ($M = 5.25$).

Hypothesis 3

Low positive affect (i.e., PANAS-C PA) was not significantly associated with ruminative coping for the group ($r = -.11, p > .05$) or when the group was split by sex ($r = -.15, p > .05$; $r = -.01, p > .05$; females and males respectively). Not surprisingly, a one way ANOVA indicated there were no significant sex differences in PA scores, $F(1, 209) = 1.95, p > .05$, partial $\eta^2 = .01$, power = .28.

Hypothesis 4

Frazier, Tix, and Barron (2004) outline the analytical process to statistically evaluate potential mediators. As applied in this study, the first step was to use regression to evaluate the relationship between depressive symptoms (the outcome variable) and chronological age (the predictor variable). The second step involved evaluating the relationship between pubertal status (the potential mediator) and age (the predictor variable). The third step was to use regression to evaluate the relationship between pubertal

status (the potential mediator) and depressive symptoms (the outcome variable). The final step evaluated the strength of relation between the predictor and the outcome, both before and after the mediator is added to the model. If a significant reduction of that relation existed, then the mediator would be said to be significant.

The steps outlined in this analytic strategy are cumulative. That is, a significant result is required at step one in order to proceed onto step two, and so on. It is the final step that reveals if a mediated relationship exists between variables. For this sample, age was not found to be a significant predictor of depressive symptoms at step one, whether it was measured by CDI scores (F (1, 209) = .78, $p > .05$) or NA scores (F (1, 210) = .28, $p > .05$). Given this nonsignificant result at step one, the mediational analyses were discontinued at this point.

The second part of this hypothesis evaluated the influence of sex. It was proposed that boys and girls in the early Tanner stages would have lower depressive scores overall, while girls in mid to late Tanner stages would have higher depressive scores than boys. To investigate this, a moderated regression was conducted with CDI scores entered as the outcome variable, both pubertal status and sex were entered on step one (as a predictor variable and potential moderator variable, respectively), and the interaction term was entered on step two. The interaction term was significant for CDI scores, F (3, 198) = 2.71, $p < .05$. That is, the relationship between PDS and CDI was moderated by sex. Means for CDI scores were plotted for both sexes by Tanner status and Figure 1 demonstrates the significant interaction with crossing slopes[8]. A second moderated regression was conducted, as above, but with NA scores entered as the outcome variable. The interaction

[8] A continuous pubertal status variable was used in the moderated regression for the statistical effect, however, to graph the effect by status the data was converted to a categorical view.

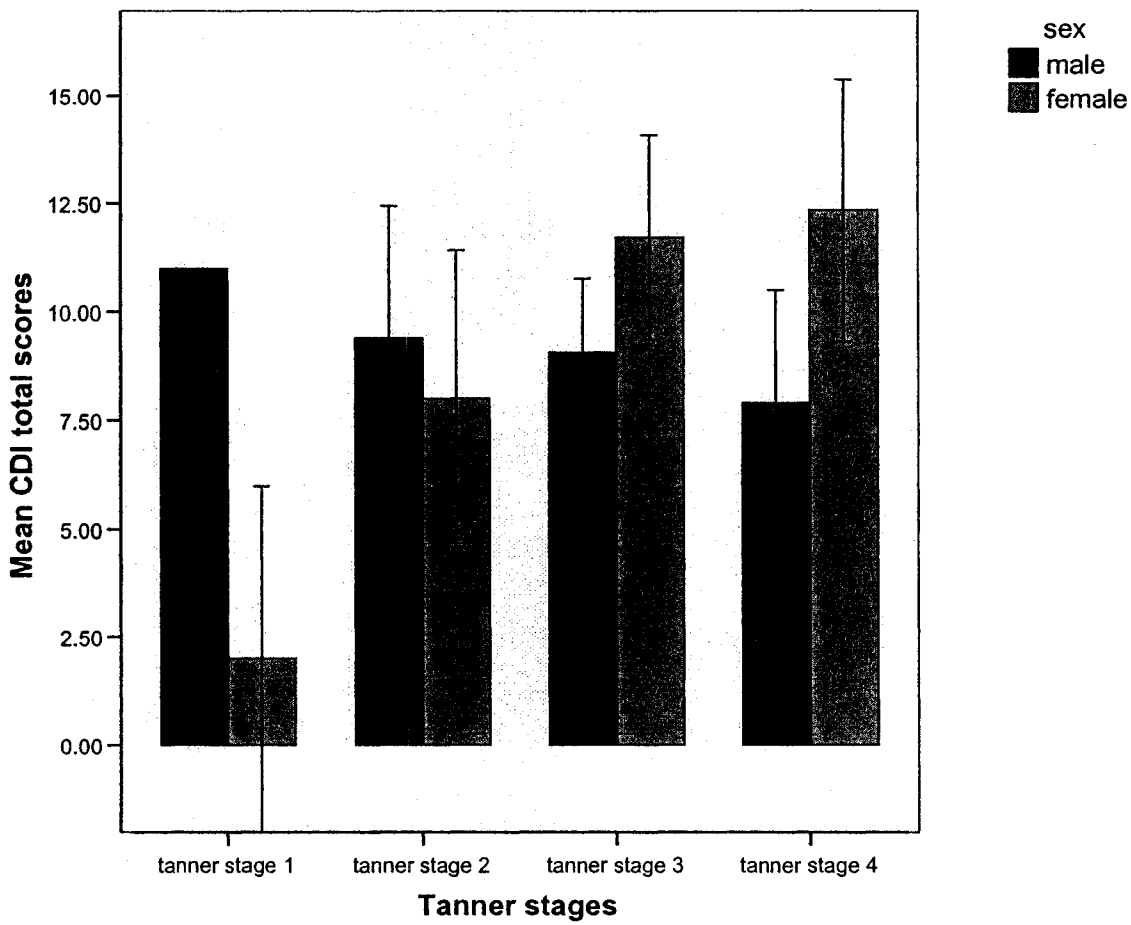

Figure 1. Children's Depression Inventory score x Tanner for males and females.

term was also significant for NA scores, $F(3, 200) = 3.43, p < .05$. Means for NA scores were plotted for both sexes by Tanner stage and Figure 2 demonstrates the significant interaction with crossing slopes. The mean age of females at Tanner stages one through four were: 12.5 ($SD = .71$), 12.73 ($SD = .65$), 13.73 ($SD = 1.56$), and 15.50 ($SD = 1.87$). The mean age of males at Tanner stages one through four were: 14 ($SD = .00$), 13.27 ($SD = .67$), 14.47 ($SD = 1.64$), and 16.30 ($SD = 1.57$).

Given this outcome, the mediational analyses were repeated with male only and female only groups. It still turned out to be the case that age was not a significant predictor of CDI or NA scores for females ($F(1, 103) = .52, p > .05$; $F(1, 103) = .73, p > .05$, respectively). The correlations between age and CDI, and age and NA were not significant for females either (-.063 and -.080, respectively, both nonsignificant). For males the path was slightly different, while the outcome remained the same. That is, age was not a significant predictor of CDI scores ($F(1, 97) = .08, p > .05$), but it was a significant predictor for NA scores ($F(1, 103) = 5.63, p < .05$). The correlations between age and CDI, and age and NA for males paralleled the regression outcome (-.028 and .233, respectively, only the latter was significant with $p < .05$). Still, when age was tested in the stepped method outlined above, the significance did not hold beyond step two. That is, the relationship between age and depressive symptoms was not significant after pubertal status was entered, $F(2, 95) = .28, p > .05$. There was no longer an effect to mediate and therefore pubertal status could not be a statistical mediator, partial or otherwise. Thus again, the mediational analyses were discontinued.

Hypothesis 5

Prior to beginning statistical evaluation of this hypothesis, interrater reliability was

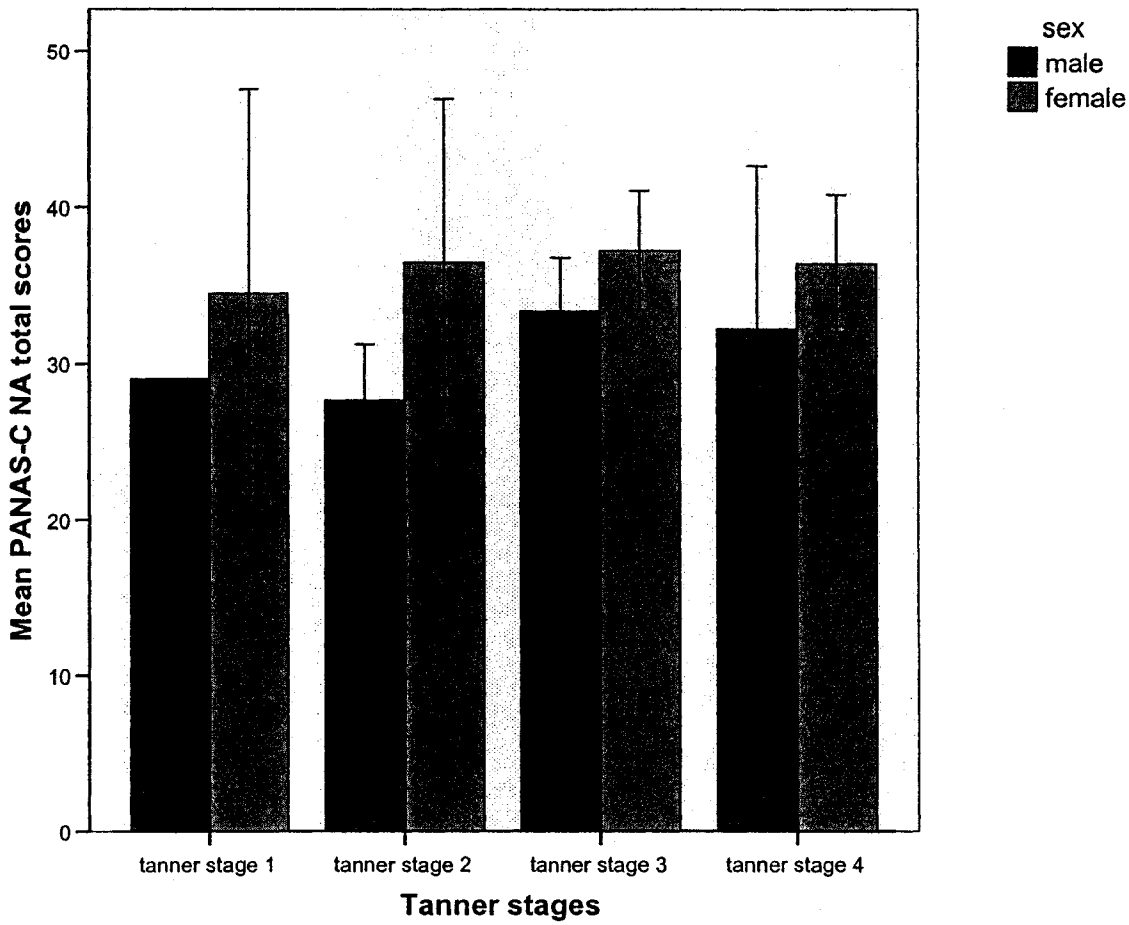

Figure 2. Negative Affect score x Tanner for males and females.

investigated for the digit ratio variable using one-way random effect single measure intraclass correlation coefficients (ICCs). The purpose was to ensure that the digit lengths recorded using the scanned photocopies of hands were equivalent to those taken in real time with the digital calliper. In total, 42 (19.7%) participants provided hand data using both methods. ICC agreements of .96 and .97 were found for the second digits on the right and left hand, respectively, followed by .92 for the fourth digits on both the right and left hands (see Table 4). All ICCs were statistically significant ($p < .01$). The internal consistency estimates (Cronbach's alpha) were sound for all comparisons and ranged from .98 to .99. The finger length variables collected through both methods were deemed acceptable for the purposes of this study. Since photocopy measures (i.e., scans of hands that were later measured with a digital calliper) were available for the full sample, it was this variable that was used in the following analyses.

It was proposed that putative indirect prenatal hormone indicators would be significant predictors of depressive symptoms above and beyond those variables tested in Hypothesis 4. Therefore two series of hierarchical regressions were conducted with depressive scores as the dependent variable (one for CDI and one for NA); age, sex, and pubertal status were entered on step one; and the putative indirect prenatal hormone indicators (i.e., ratio of the second to fourth digit on the right and left hands, ridge count of the first and fifth digit on the right and left hands) were entered on step two. Two separate hierarchical regressions were required for each hormone indicator because some participants were excluded if they had ever broken or fractured their second or fourth finger. Such an event could have potentially altered the bone growth in those digits and presented as a measurement confound affecting the overall digit ratio variable. A broken

Table 4

Interrater Reliability of Digit Ratio Variable

Hand	Digit	n	ICC	Cronbach's α
Right	D2	42	.96	.98
	D4	42	.92	.98
Left	D2	42	.97	.99
	D4	42	.92	.99

Note. ICC = intraclass correlation coefficient; D2 = second digit; D4 = fourth digit. All significant at $p < .01$.

finger would not, however, alter the ridge count pattern since that is defined in utero (Jamison et al., 1993). In total, 16 participants were excluded from the hierarchical regression that analyzed the digit ratio variable. A t test demonstrated that the digit ratio variable means were as expected. That is, males (right and left mean = 0.95, $SD = 0.03$) had significantly lower 2D:4D ratios than females (right and left mean = .97, $SD = .03$), right t (154) = -3.04, $p < .05$ and left t (154) = -3.42, $p < .05$, respectively.

When digit ratios for the right and left hands were entered into step two of the hierarchical regression equation, they were found to be significant predictors of CDI scores, F (5, 182) = 2.31, $p < .05$; but not significant predictors of NA scores, F (5, 184) = 2.16, $p > .05$. For CDI, all variables in the model were able to account for 6% of the variance (R^2 value), and digit ratios were able to account for a unique 1.9% (R^2 change value). The standardized regression coefficients (β) for right and left digit ratios in the CDI model were -.11 and .20, respectively.

When finger ridge counts for the right and left hands were entered into step two of the hierarchical regression equation, they were found not to be significant predictors of CDI scores, F (5, 116) = 1.47, $p > .05$, nor NA scores, F (5, 117) = 1.64, $p > .05$.

For exploratory purposes, the prenatal hormone indicators were tested individually as predictors of depressive scores (CDI and NA scores) without controlling for any variables on step one. Only one was found to be a significant predictor independently. Digit ratio on the left hand was found to significantly predict CDI scores, F (1, 193) = 4.67, $p < .05$.

Supplementary Analyses

Hypothesis 6

To investigate the contribution of parental and peer stress to depressive symptoms, multiple regressions were conducted. CDI, NA, and PA scores were used as outcome variables. Parent and peer variables were entered on separate steps of the regression equation to determine the unique contribution of each variable. Pearson correlations were used to determine the order of entry (i.e., parent stress revealed a higher correlation to all outcomes and therefore was entered first on step one; see Table 5). For CDI scores, both parent and peer stress variables were significant predictors, F (1, 206) = 67.12, $p < .01$, standardized regression coefficients (β) = .50, and F (2, 205) = 41.78, $p < .01$, standardized regression coefficients (β) = .24, respectively. Together, they accounted for 29% of the variance in depressive scores (R^2 value). Individually, parent stress accounted for 24.6% of the variance, while peer stress accounted for an additional 4.4% (R^2 change values). Both parent and peer indicators of stress were significant predictors of NA scores, F (1, 208) = 51.10, $p < .01$, standardized regression coefficients (β) = .44, and F (2, 207) = 27.40, $p < .01$, standardized regression coefficients (β) = .13, respectively. Together, they accounted for 21% of the variance in NA (R^2 value). Individually, parent stress accounted for 19.7% of the variance, while peer stress accounted for an additional 1.2% (R^2 change values). Both parent and peer indicators of stress were significant predictors of PA scores, F (1, 207) = 11.27, $p < .01$, standardized regression coefficients (β) = -.23 and F (2, 206) = 5.68, $p < .01$, standardized regression coefficients (β) = -.03, respectively. Together, they accounted for 5.2% of the variance in PA (R^2 value). Interestingly though, parent stress

Table 5

Correlations Among Parent and Peer Stress and Depressive scores (Children's Depression Inventory, Negative Affect, and Positive Affect)

	CDI	NA	PA
Parent stress	.50 **	.44 **	-.23**
Peer stress	.43 **	.32 **	-.14

Note. CDI = Children's Depression Inventory; NA = Negative Affect; PA = Positive Affect.
** significant at $p < .01$

accounted for the majority of this variance (i.e., 5.1%), while peer stress only accounted for an additional .1% (R^2 change values).

Overall, parent stress levels ($M = 23.42$, $SD = 8.60$) significantly exceeded peer stress levels ($M = 20.59$, $SD = 7.92$), $t\ (209) = 4.98$, $p < .05$. An ANOVA demonstrated that males and females experienced similar stress levels in parent and peer domains, $F\ (1, 211) = .01$, $p > .05$, partial $\eta^2 = .00$, power = .05, and $F\ (1, 208) = .02$, $p > .05$, partial $\eta^2 = .00$, power = .05, respectively.

The above regressions were repeated with social desirability entered on step one. No significant changes in interpretation resulted. That is, parent and peer stress variables were significant predictors of CDI scores ($F\ (2, 205) = 36.71$, $p < .01$ and $F\ (3, 204) = 30.92$, $p < .01$, respectively), NA scores ($F\ (2, 205) = 25.96$, $p < .01$ and $F\ (3, 204) = 18.69$, $p < .01$, respectively), and PA scores ($F\ (2, 205) = 5.73$, $p < .01$ and $F\ (3, 204) = 3.86$, $p < .01$, respectively). The above ANOVA was also repeated with social desirability entered as a covariate (i.e., ANCOVA). The outcome was the same; that is, males and females experienced similar stress levels in parent and peer domains ($F\ (1, 210) = .12$, $p > .05$, partial $\eta^2 = .00$, power = .06, and $F\ (1, 207) = .84$, $p > .05$, partial $\eta^2 = .00$, power = .06, respectively).

All analyses presented in this dissertation were run on the full sample of participants (except where noted). To ensure that exogenous hormonal effects were not driving the significant findings reported, the analyses were repeated with eight participants excluded (seven females, one male). Those eight participants were identified as taking medications that included synthetic hormone or steroid combinations (e.g., oral

contraceptives or antidepressants). There were no significant differences in the statistics reported and all interpretations remained the same after the exclusion of these participants.

Discussion

The results of this study provide partial support for the hypotheses. Specifically: 1) the findings of this research supports RST in youth samples; 2) sex differences were found with respect to depressive symptoms and rumination; 3) some components of the tripartite model of depression seemed to be applicable to adolescents, while others were less so; 4) whether age and pubertal status are robust mediators of depressive symptoms is unclear; 5) the relationship between depressive symptoms and pubertal status was moderated by sex; 6) the 2D:4D variable yielded sex differences as expected, and significantly predicted depressive symptoms; 7) finger ridge count did not significantly predict depressive symptoms; and finally, 8) parent and peer stress levels were similar across sex and both were significant predictors of depressive symptoms in adolescents.

Hypothesis 1 was supported with findings from the Pearson correlations and ANOVAs. Depressive symptoms and rumination were positively and significantly related in this youth sample. This significant relationship held when alternate measures of depressive scores (i.e., CDI and PANAS-C NA) were used. These results are consistent with other findings in the literature based on youth samples (Abela et al., 2002; Schwartz & Koenig, 1996; Ziegert & Kistner, 2002). In fact, the magnitude of the correlation between rumination and CDI scores was stronger in this sample (.37) compared to Ziegert and Kistner's (2002) findings (.25). Given the cross sectional design of this study, we cannot confirm which factor drives this effect; that is, whether youth who ruminate more also end up becoming more depressed or whether depressed youth also end up ruminating more.

Regardless of directionality, this finding supports at least part of the RST (the rumination factor) extension to youth.

The second part of Hypothesis 1 (i.e., sex differences in depressive scores) was also supported. For this particular sample, it seems that females expressed an elevated level of depressive symptoms compared to their male counterparts. This finding has not consistently been found in other studies examining youth samples (Seiffge-Krenke & Klessinger, 2000; Ziegert & Kistner, 2002). The age range for this study was selected precisely because sex differences most commonly emerge between 12 and 17 years; and this is what we found. The significant sex difference in depressive scores found here supports the need to learn more about how well RST "fits" (or does not "fit") for adolescents. So far the data suggest this youth sample is comparable to adult samples (Broderick, 1998; Nolen-Hoeksema, 1998; Spasojevic & Alloy, 2001) with respect to rumination and sex differences in depressive scores.

Hypothesis 2 was partially supported. In line with adult RST work (Nolen-Hoeksema, 1998; Nolen-Hoeksema et al., 1999; Nolen-Hoeksema & Morrow, 1991), the findings from this study indicate that female youth tended to ruminate more than male youth, even after depressive symptoms were controlled for. These results contrast with other studies that have not found a sex difference in ruminative coping (e.g., Abela et al., 2002) in youth samples. However, if one considers a developmental perspective here, these two outcomes are not necessarily in conflict. The different age ranges in this study and the Abela et al. (2002) study (12 to 17 versus 8 to 12, respectively) would seem to suggest that the ruminative sex difference emerges sometime between the ages of 8 and 12 (perhaps on the later end of that range), and is certainly in existence sometime after age 12. The

changing nature of this factor across time indicates the need to have a more comprehensive understanding of how rumination contributes to the development or maintenance of depression. Currently, cross sectional (and one short-term longitudinal) findings are being integrated across studies to inform research hypotheses. However, a single longitudinal study with a larger age bracket could answer these questions more definitively.

Since depressive scores were controlled for in the ANCOVA conducted for this hypothesis, the significant results noted were therefore more likely to be a result of true effects of rumination rather than differences due to measurement or construct overlap. The significant sex difference finding with respect to rumination also supports that the CRSS is a valid indicator of rumination that is not confounded by depression. The Welsh and Mazmanian (2007) study also supports the psychometric stability of the CRSS with respect to internal consistency of each subscale.

The second part of the RST in youth hypothesis was not supported. The alternative coping style included in this study (i.e., distraction) was not discriminative by sex. That is, males and females did not differ in the use of distraction as a coping strategy. Similar to other youth (Ziegert & Kistner, 2002) and adult studies (Nolen-Hoeksema, 1998; Spasojevic & Alloy, 2001), the sex difference in response style was only true for ruminative coping. This hypothesis was generated to evaluate a component of the adult RST that has been reported in other studies (i.e., males use distraction significantly more than females; Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema, 1991). Findings in the current study do not support the distraction hypothesis when applied to a younger sample. This outcome suggests that distraction may be less informative with respect to sex differences in depression when evaluating youth samples. Although the higher mean of

distraction compared to rumination is suggestive of more frequent use as a coping strategy, it seems not to be systematically related to the main variables under investigation here. It may be the case that adolescents tend to use a variety of coping styles and are not entrenched in a pattern. If this is true, then coping style may be more flexible in youth, and perhaps this is protective in its' own right.

Alternative explanations of the current finding must also be considered, however. Past research using distraction subscales has been criticized for poor subscale construction as the driving force behind nonsignificant results. That is, fewer items loading onto the distraction subscale in comparison to the rumination subscale has been cited as a reason for nonsignificant findings. This criticism does not apply here because both distraction and rumination subscales on the CRSS had an equal number of items (i.e., 10 each). Perhaps the strength or efficacy of distraction as a coping style (i.e., how well it works) would be more informative rather than the raw number of distraction techniques employed. New measures would need to be developed to capture this dimension. It is also possible that the combination of coping styles used is important (e.g., having multiple active *and* passive strategies at your disposal to use as you determine) as opposed to relying on one style independently. In this study, females reported using more ways to cope overall than did males. Although distraction was the only alternative coping style included in this study, other coping styles (e.g., problem solving, avoidance) may be used differentially across sex and may also be predictive in depression models. These examples highlight the potential interpretive errors that may be created if a downward extension of an adult theory is not critically or empirically evaluated.

Hypothesis 3 was not supported. Positive affect was not significantly correlated with ruminative coping for either sex. This hypothesis involved merging components from distinct theories of depression (i.e., the rumination factor from RST and the PA factor from the tripartite model). While this result is statistically non-significant, it is nonetheless informative. Positive affect, a significant player in the tripartite model of depression (certainly in adults [Clark et al., 1994], and there is evidence accumulating with adolescents [Laurent & Ettelson, 2001]), was included in this study because a comprehensive understanding of this variable was still lacking in youth samples. It was unclear how this variable was related to another key variable in the depression literature (i.e., rumination) because it has been relatively neglected in research.

This study found that PA symptoms did not differ by sex, nor was it significantly related to a variable that so commonly reveals sex differences. The nonsignificant correlation in this one study is not definitive in ruling out PA from theoretical consideration (especially given the solid empirical grounds it has in the tripartite model from many other studies). It is true, however, that a significant correlation is necessary before other more complex statistical analyses can "rule in" a predictor. Rather, future studies should continue to include this component until a solid empirical foundation of results is revealed. It is possible of course, that PA is an important piece of the depressive adolescent sex difference puzzle, but the PANAS-C PA subscale was not an adequate measure of PA for this sample (i.e., poor construct validity). This point must at least be considered for interpretation until more empirical evaluations can demonstrate otherwise.

Alternatively, some research notes that PA may be a stronger predictor for anxiety in youth than depression (Laurent & Ettelson, 2001). This is exactly the relationship that

was found in this data set. The PA variable did demonstrate significant negative correlations with two of three anxiety subscale scores (i.e., -.12 for RCMAS worry/oversensitivity and -.34 for RCMAS social concerns/concentration). Although not an explicit hypothesis evaluated in this project, this outcome contradicts the distinction in adult samples that low PA is relevant in depression but not anxiety (Clark et al., 1994). Again, this may be another example of how a downward extension of adult theories (e.g., RST and tripartite) to adolescence must be tailored.

The nonsignificant outcome for Hypothesis 3 (i.e., PA was not a significant indicator of depressive symptoms in youth) may be informative in terms of assessing and treating youth for depression. More specifically, knowing if an adolescent is high or low on positive affect will not necessarily help predict who will and will not experience or develop depression. Consider Weiss and Garber's (2003) comment that syndrome differences may exist between adult and youth forms of depression. Loss of interest in pleasure is one criterion for a major depressive episode. Extrapolating the nonsignificant low PA results in this study would seem to suggest that this criterion might not fit the adolescent profile of depression. Assessing for this aspect of depression will not likely inform who is at higher risk; nor will targeting this domain in treatment programs likely directly reduce depressive symptoms (e.g., attempting to increase PA levels through cognitive remediation). PA may, however, be helpful to target in a treatment program for youth with anxiety problems. Given that anxiety is often a "precursor" to depression in adolescents, targeting PA in such a group may indirectly serve as a way to prevent depression by stopping the progression early on.

Hypotheses 1, 2, and 3 all related to the rumination variable. Compared to the adult literature, the role this variable plays in younger populations is less well defined. The measure used in this study that represented rumination was the CRSS rumination subscale. This scale has not been as thoroughly evaluated in terms of psychometrics as other rumination scales (e.g., the Response Styles Questionnaire from Nolen-Hoeksema & Morrow [1991]). However, promising results have been found during initial psychometric queries (Welsh & Mazmanian, 2007; Ziegert & Kistner, 2002). These studies lend empirical support to the strong reliabilities and validities of the scale as two independent factors of coping style - rumination and distraction. Other versions of a rumination subscale have been developed as well (e.g., Children's Response Style Questionnaire, Abela, Rochon, & Vanderbilt, 2000; Coping Across Situations Questionnaire, Sieffge-Krenke, 1995). It is encouraging to see independent sources investigating this process in adolescents.

Together, the results from Hypotheses 1, 2, and 3 inform us about the validity of Nolen Hoeksema and Girgus' (1994) Different Factors Model. Specifically, the data lend support to the interpretation that rumination is a personality risk factor that may lead to depression for girls but not for boys. A cross-sectional study with a larger age span or a longitudinal study would, however, be required to determine if ruminative coping becomes more common in early adolescence (the second component required to confirm a variable as part of this model). As mentioned above, if one combines results from multiple studies, it seems at least likely that this ruminative coping differential emerges sometime between ages 8 and 12. However, this remains to be empirically tested. The data also seem to suggest that distraction is a personality risk factor that does not differentially lead to

depression for either sex. Therefore, these results suggest that a distraction coping style is not defined by the Different Factors model (Nolen-Hoeksema & Girgus, 1994).

Cumulatively, cross sectional and short term longitudinal studies suggest that both onset and maintenance actions for rumination may be important mechanisms for sex differences in depression. Rumination is a personality variable that may be amenable to change. If that coping style can be successfully altered (or avoided in the first place), the risk and prevalence of depression may decrease. Given that rumination is a more passive coping strategy, and the more prevalent one in girls who are at heightened risk, aiming prevention efforts at developing and using alternative coping strategies (including more active strategies) toward this group may be an especially fruitful technique to counteract the onset mechanism of action. It may also be advantageous to offer cognitive remediation aimed at interrupting the rumination cycle to counteract the maintenance mechanism of action (Abela et al., 2002). A repeated measures longitudinal design would be necessary to fully evaluate the validity of such hypotheses and any intervention efforts.

The current research does demonstrate that youth as young as 12 years of age do express depressive symptoms differentially across sex. Girls reported higher depressive scores and tended to use rumination more than boys. Consequently, it may be appropriate to advocate for mental health screening and psychoeducational opportunities to be provided in school programs prior to grade seven for both sexes, but especially girls. This could easily be incorporated into health or social science seminars or awareness weeks. It also seems that PA is less discriminative with respect to sex differences in depressed youth. This may be helpful for adults to know in terms of recognizing that some "typical" signs of depression (e.g., low energy, low excitement, low happiness, loss of pleasure) may not be

evident in youth, but they can still be at risk. In this sense, expanding "our" view of what depression looks like in youth can help "us" be better at identifying those youth who could benefit from support or mental health services. Crucial timing of such opportunities may help avoid the development of a severe and chronic disorder in youth.

The mediational analyses for Hypothesis 4 could not be completed. Following the procedure outlined by Frazier et al. (2004), step one was not significant. That is, chronological age did not demonstrate the expected predictive relationship for depressive scores (whether defined by CDI scores or NA scores). This outcome was surprising given the robust relationship reported in many other studies (Abela et al., 2002; Fleming & Offord, 1990; Hankin & Abramson, 2001). The correlations between age and pubertal status for males ($r = .57, p < .01$) and females ($r = .53, p < .01$) were moderate and significant. It is not clear why age was not a significant predictor. While the whole sample spanned an age range of seven years, over 56% of the sample was either 13 or 14 years old. This distribution may have contributed to a restriction in range with respect to age. The outcome scales used to measure depressive symptoms in this study have been used in other projects. The current sample had similar CDI scores (mean = 10.17, *SD* = 7.80, cf. mean = 8.49-9.84, *SD* = 6.61-7.34 for Abela et al. [2002] and mean = 9.69-9.97, *SD* = 7.91-8.17 for Ziegert & Kistner [2002]) and NA scores (mean = 34.16, *SD* = 13.68, cf. mean = 26.97, *SD* = 10.58 for Laurent et al., [1999]). Therefore it is unlikely that this sample was restricted in range with respect to depressive symptoms. Sample sizes in the studies noted were both smaller and larger than the current study. Regardless of the explanation, this curious outcome did not allow a full comparison between age and pubertal status to be conducted.

In the second part of Hypothesis 4 sex was considered as a moderator of the pubertal status/depression relationship. In this case, significant results were found. That is, sex appeared to moderate the relationship between pubertal status and depression. More specifically, when both sexes were plotted as one group, CDI scores demonstrated an increasing trend as pubertal status increased (see Figure 3), but this trend was nonsignificant. A similar trend existed for NA as pubertal status increased (see Figure 4). Without separating the groups by sex, the significant interaction was masked. When the sample was split by sex the effect for pubertal status was significant. That is, male CDI scores decreased as pubertal status increased and female CDI scores increased as pubertal status increased (see Figure 1). The effect was less dramatic for NA, but still suggested a differential slope in NA scores as pubertal status increased for males (near higher Tanner stages) compared to females (see Figure 2). Overall, it seems that pubertal status has some merit with respect to explaining the sex difference in depression situation. It seems to be especially relevant for females. This finding is not revealed if the sample is considered as a whole. This differential outcome seems to offer support for Nolen-Hoeksema and Girgus' (1994) Different Factors model.

Visual examination of the effect shows a jump in CDI scores between Tanner stages one and two as well as stages two and three for females. It seems logical that these two stages (one and two) may represent critical time periods for intervention. Given that age of progression through stages one and two is not uniform for females (i.e., early vs. late developers), it would make sense to consider the indicators at each of these stages as signals of when intervention may be needed. A repeated measures study with both pre-adolescent and adolescent samples, or a longitudinal study (with the full range of Tanner

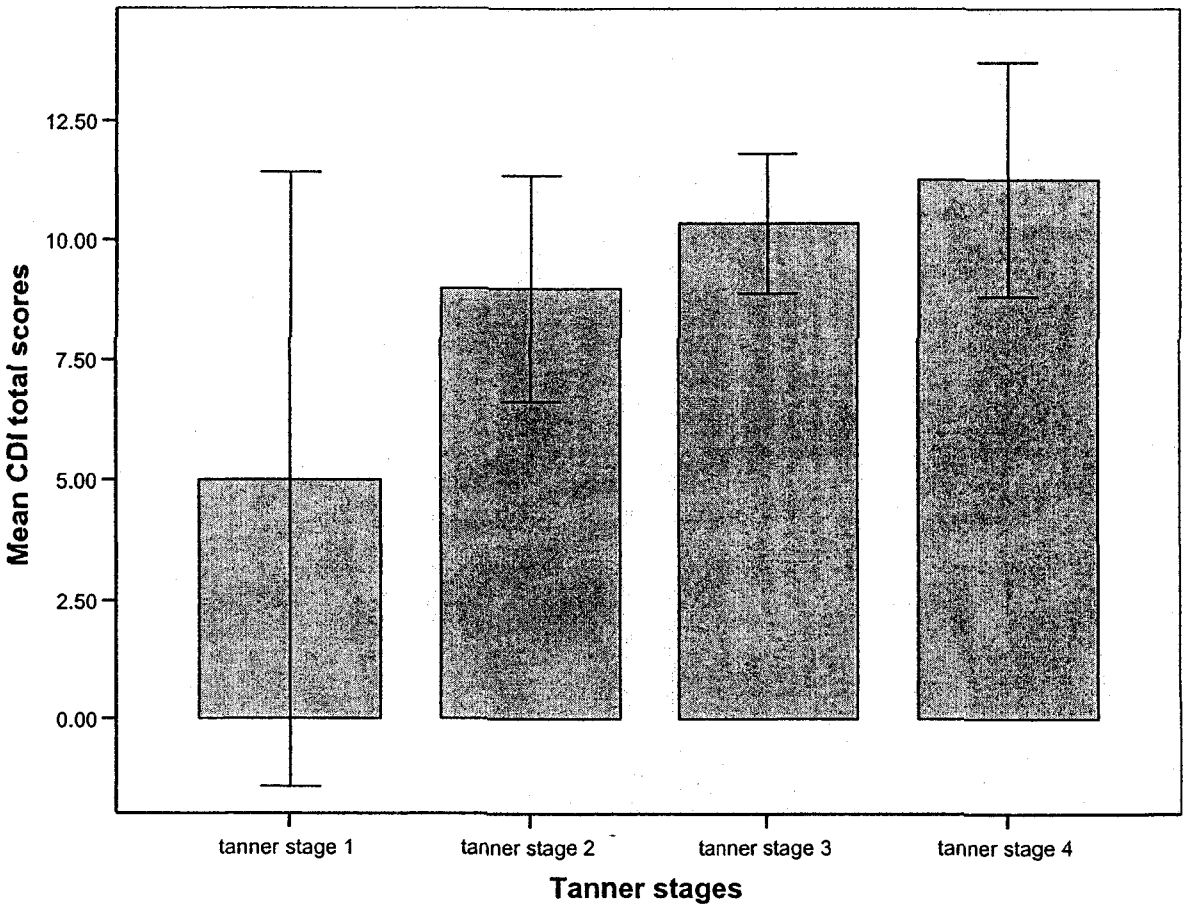

Figure 3. Children's Depression Inventory score x Tanner for full sample.

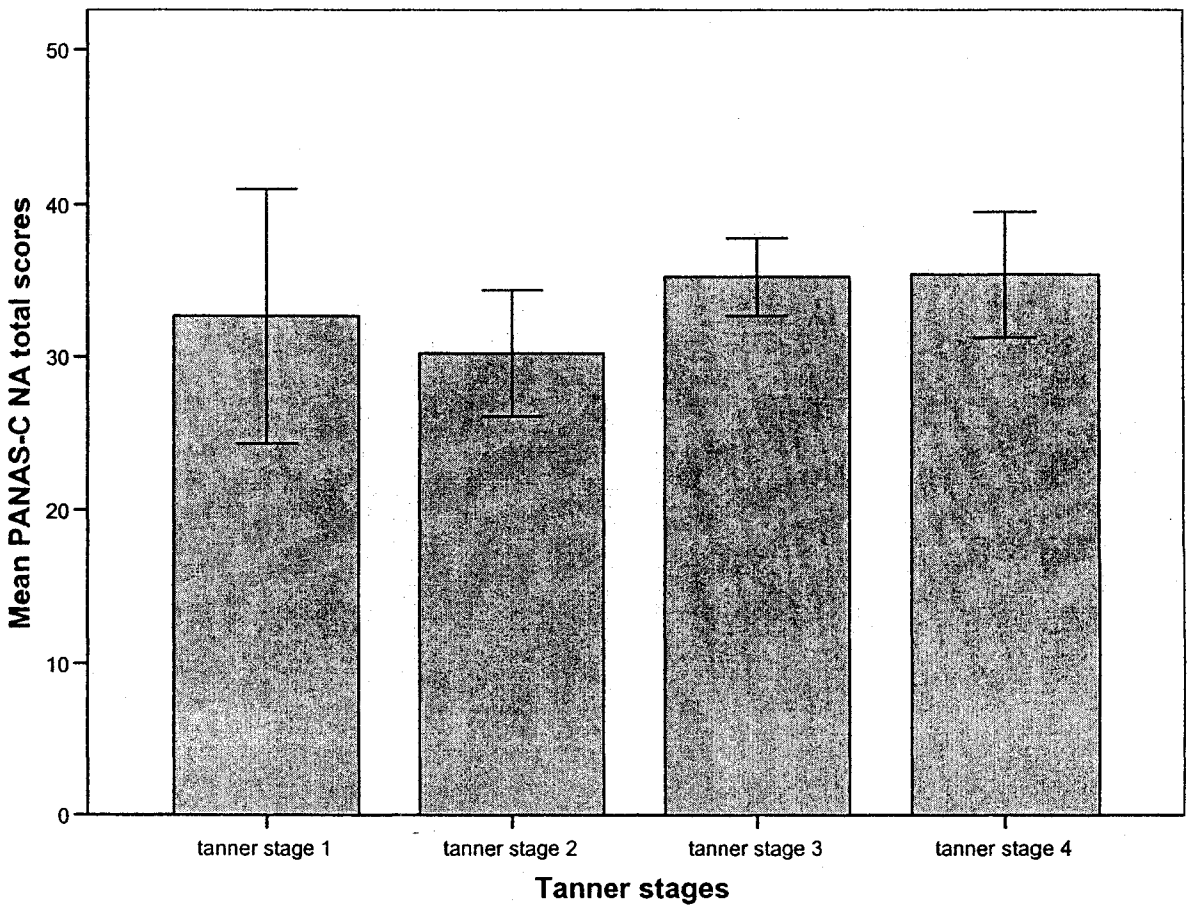

Figure 4. Negative Affect score x Tanner for full sample.

stages with enough participants in each stage) would be needed to narrow down which events associated with these stages are most informative. If one adopts a model of prevention, one could use these indicators as flags to intervene before this level of development is achieved. For example, *before* a female has a growth spurt, begins to show body hair or skin changes, and starts developing breasts would seem to be an ideal time to help avoid the onset of depressive symptoms through psychoeducation. Given results from previous hypotheses in this project, educating youth about coping style and alternative and supportive strategies may be most appropriate during this time frame. The tendency for girls to ruminate is more significantly related to pubertal status than age ($r = .22, p < .05$; $r = .12, p > .05$, respectively), therefore such an approach would seem to be appropriate. While previous research has cited the onset of menarche as key with respect to the initiation of sex differences in adolescent depression (Born et al., 2002), we were unable to adequately test this in our sample due to the small number of premenarchal females (i.e., 15 premenarche girls with CDI scores vs. 92 post menarche girls with CDI scores). We therefore did not have empirical evidence to narrow down the suggested time frame for intervention.

It is possible that use of the PDS in questionnaire form was not as reliable an indicator of pubertal status with this sample (cf. gold standard of physician ratings). In the past Cronbach alpha levels of this questionnaire have ranged from .66 to .81 (Carskadon & Acebo, 1993; Robertson et al., 1992). Although less than ideal, the questionnaire is a format that is socially acceptable in school settings. It would not have been feasible to include physician ratings in this project. While the response bias measure did not indicate that social desirability was a concern with respect to ratings (see Table 3 for the

nonsignificant correlation), it may still be the case that participants were not very good raters of themselves using this instrument for other reasons. Perhaps the adolescents reported their own development relative to some unknown referent. For example, an average girl whose peers are early maturers may rate her own development as *barely started* when in fact, her development is *well under way*. An average girl at the same level of development may rate her development as *completed* if her peers are late maturers. We had no way to control for this possible bias in rating. Most of the sample fell within Tanner stages 2-4 (almost 60% at stage 3), while Tanner stages one and five were virtually unrepresented. Perhaps the distribution for this variable was not dispersed enough to demonstrate a significant effect across pubertal status for the whole sample. It also may be the case that pubertal status per se is not the "active ingredient" in the puberty-depression recipe, but rather that the *adjustment* to pubertal status is the most informative factor. The rate at which pubertal changes occur and the sense of stability surrounding those transitions may be useful to know to understand the nature of this relationship further. This kind of relationship was not measured in this study.

Hypothesis 5 evaluated the value of putative indirect prenatal hormone indicators in the prediction of depressive scores in youth. Past research has reported conflictual findings with respect to digit ratio as a marker of the prenatal environment using adult samples. Few studies have evaluated digit ratio in youth; even fewer have investigated finger ridge count as an indirect hormone indicator. Research investigating the organizational influence of these variables on psychological functioning and behavior is slowly building (Kajantie & Phillips, 2006; Papaioannou et al., 2002; Sloboda et al., 2006), and the current project contributes to that foundation. The methodology that was used to obtain the digit ratio

measures (photocopy scans that were later measured with a digital calliper) was deemed equivalent through ICC analyses to measurements taken in real time with a digital calliper. Through this methodology, empirical support for digit ratio as a significant predictor of depressive scores, as measured by CDI scores, in youth was found.

Ratio of the second to fourth digits has been proposed to be a marker for prenatal testosterone and estrogen (i.e., digit ratio may be a negative correlate of prenatal testosterone and a positive correlate of prenatal estrogen concentrations; Fink, Neave, Laughton, & Manning, 2006; Lutchmaya et al., 2004; Robinson & Manning, 2000). The present study found a significant relationship between the digit ratio variable and depressive scores in youth. However, the interpretation of that finding is less clear. The standardized regression coefficients for the right and left hands were in the opposite direction (i.e., negative and positive, respectively). Thus, the direction of effect is equivocal at this point. It may be that a low digit ratio (i.e., second digit is much shorter than the fourth digit) is important in predicting higher depressive outcomes in adolescents. If one integrates this information with past research on this variable, this outcome may suggest that high prenatal testosterone and low prenatal estrogen are associated with higher CDI scores in adolescence. This interpretation of findings, which is also what Angold et al. (1999) found, would seem to suggest that there may be an organizational effect of high testosterone and low estrogen that may predispose an individual to be more likely to develop depressive symptoms later in life (i.e., adolescence), especially when other factors or stressors (e.g., coping style, puberty) come onto the scene and interact with biological influences. It would also be in line with the hypothesis that some depressed women do not have sufficient estrogen levels and require augmentation strategies to experience treatment

benefits. However, the opposite alternative (i.e., high digit ratio; second digit is longer than the fourth; low prenatal testosterone and high prenatal estrogen levels are implicated) appears just as likely given the current data.

If either of the hormonal interpretations is accurate, there seems to be support for the fetal origins hypothesis of adult disease (Welberg & Secki, 2001). For example, the prenatal environment may somehow predetermine the responsivity of certain hormones that will fluctuate when the initial stages of puberty begin. The morphological changes associated with puberty may be the visible indicators of such a chain of events, but the hormonal alterations may exist for some time before (perhaps even prenatally) the morphological changes surface. When such an interaction occurs, symptoms may build up to be severe enough to meet criteria for a depressive episode. Therefore, the underlying hormones (or their responsivity) may well indirectly affect the outcome through a chain of events (e.g., prenatal/organizational effects, postnatal/activational effects such as puberty with activating hormones, and environmental effects) that may begin in the womb and eventually lead to a depressive outcome. If this is the case, it may offer support for Nolen-Hoeksema and Girgus' (1994) Interaction model. However, since no three-way interactions were done with this data we can't confirm this hypothesis.

Given that there are varying hormone levels present within the same pubertal stage, and similar hormone levels at contiguous pubertal stages, studies investigating the activational effects of hormones in real time (i.e., direct measures taken during adolescence) are necessary to parse out the details of the proposed chain of events. If multiple hormonal measures could be taken before, during, and after pubertal changes (i.e., span across a number of years), it may be possible to identify the developmental path and

critical period of when these hormonal changes (which may be partially determined in utero) have an immediate influence on psychosocial adjustment. While the cost of such a research project would be large, the data collected would be greatly informative.

Across studies, the effect of hormonal contribution has been small but, nonetheless, stable. The percent of variance accounted for in this study (i.e., 1.9%) is lower than the 4% reported by Brooks-Gunn et al. (1994), but nonetheless significant. Clearly there are other significant pieces to the puzzle as well. Some have been evaluated and reviewed in this project. Other sources are beginning to evaluate the link between hormonal functions and personality characteristics as well. Fink et al. (2006) found that males with low 2D:4D ratios tend to have higher scores on sensation seeking, thus supporting an organizational interpretation for at least one personality characteristic. While not a prespecified hypothesis in the current project, 2D:4D did turn out to be a significant predictor of rumination (another aspect of personality), $F(2, 193) = 3.68, p < .05$. These findings demonstrate the need to cast a wide/comprehensive net to decipher the whole complicated picture of sex differences in depression.

To further explore the value of the 2D:4D variable, right and left indicators were compared. Although not hypothesized a priori, it seems to be the case that left 2D:4D was a significant predictor on its own for CDI scores, but right 2D:4D was not. Why the left hand features are more predictive than the right is indeed curious. In a related study with a similar finding, Manning (2002) reported a significant relation between 2D:4D and finger ridge count in very low birth weight children in the left hand but not the right. Intuitively, this finding contradicts the suggestion that sexually dimorphic traits are expressed more strongly on the right side (Tanner, 1978) and the association with testosterone is strongest

in the right hand. In a 1998 study by Manning et al., they found opposite results from ours (i.e., right was significant and left was not), and they were unable to explain the differential effect other than to refer to possible laterality effects (i.e., lower pattern intensity in left hand). Aside from a possible type II error as an explanation for the current left sided outcome, these spurious findings remain unclear. Replication of this finding with other samples may help to increase our understanding of this result.

Some caution must be offered with respect to the interpretation of the digit ratio variable. Manning et al. (2005) note concern that photocopy measures may conflate (but not invalidate) sex differences in this ratio. More specifically, they suggest that variation in the shape of the finger tips may pose a problem when placed on the glass plate due to reflection of light during the photocopying process. There appears to be a negative association between the number and type of sex chromosome and size of finger tip fat-pad which translates into fat-pad size being a sexually dimorphic trait. It is possible that three-dimensional variation in fat-pad size and shape may result in small distortions of the two-dimensional photocopy image through increased curvature that appears to make the digit longer than it truly is. If this hypothesis is true, then the data from this sample may be biased in these respects. The high ICCs found across methodologies reduce the likelihood of this possibility confounding results. At the same time, a similar critique could be offered with respect to the methodology applied in real time calliper measures. Although reflected light may not be the source of error, pressure at the end of the finger tip when the measure is being taken may not be uniform. Although not necessarily likely to result in a sexually dimorphic difference, inconsistent error may still be introduced. To rule these influences

out in any study, it would be most advantageous to have a full set of digit ratio data from both photocopy and real time digital calliper methods.

The suggestion has been made that the 2D:4D ratio may correlate with patterns of dermatoglyphic ridges (Manning, 2002). Thus, finger ridge count may be a secondary indicator of the prenatal environment. Manning et al. (1998) report that the development of the epidermal ridges on the digits and testosterone concentrations are positively correlated in adult males and are likely correlated with fetal concentrations (also supported by Manning et al., 2000). Finger ridge count, however, was not found to be a significant predictor for any kind of depressive scores (CDI nor NA scores) in this sample of youth using hierarchical regression techniques. We have no evidence to support the extension of this claim in adolescents. Recall that finger ridge count was the variable with the most missing data. In fact, too much missing data to replace with regular estimation techniques. The analyses for this variable, therefore, had a smaller sample size on which to draw. In comparison to the other independent variables included in the study, there was likely less power to detect a significant effect for finger ridge count if it existed. We therefore cannot make strong conclusions about this variable as a prenatal hormone indicator or, even if it doesn't represent the prenatal environment, as a predictor of depressive scores.

As an alternative to an indirect hormone indicator, finger ridge count may be a visual indicator of chromosomal abnormalities. Polani (1981) found that total finger ridge count was lower than expected in males with XXY structures and higher than expected in females with X structures. It is therefore possible that the speed of prenatal maturation is delayed in the former and accelerated in the latter, and finger ridge count may be an indicator of this (i.e., a prenatally determined index of maturational rate; Jamison et al.,

1993). It is also possible that modifiers on the X chromosome are responsible for sex-related differences in continuous variation seen in the 2D:4D ratio (Manning, 2002). If this is the case, genetics may be the very first link in the chain of indirect events that potentiates risk for depressive symptomatology later in life. Research evaluating heritability of this 2D:4D variable is ongoing with Manning's Jamaican Symmetry Project and may prove informative.

Despite the findings noted in this project, it is worth finding more concrete and reliable ways to investigate the influence of indirect hormone indicators on sex differences in depression. It is fairly well established in animal research that prenatal stress is associated with increased behavioral disturbance (e.g., Schneider, Roughton, Koehler, & Lubach, 1999; Weinstock, 2001). Extending this work to humans with prenatal anxiety, O'Connor, et al. (2005) suggest that there might be lasting effects of HPA axis functioning in children. That is, a 10-year follow-up study by these authors notes that maternal prenatal anxiety may be a mechanism for an increased vulnerability to psychopathology in children and adolescents. They examined the long-term association between maternal prenatal anxiety (direct measures collected during pregnancy) and wet measures of diurnal cortisol in the children at age 10 years. Findings indicate that anxiety/stress in the prenatal environment accentuates activation of the HPA axis leading to a release of cortisol, which in turn, influences the developing fetal HPA axis. They also have data that indicate this effect is maintained into late childhood. The HPA axis seems to be a primary mediating mechanism in much of this line of research (Glover & O'Connor, 2005). In the study referred to here, the evidence is suggestive of a prenatal hormone vulnerability to anxiety. They could not, however, rule out the influence of current maternal anxiety levels. Given

the significant overlap and association between depressive and anxiety symptoms and prevalence rates, it is not a far leap to generate similar hypotheses about the HPA axis and depression in adolescence. In summary, it seems possible that a prenatal hormonal component (likely for anxiety and maybe for depression) may produce a subtle, but long term effect. Such a pattern is worth evaluating if it could avoid the development of life long mental health issues.

Hypothesis 6 was partially supported. Males and females between ages 12 and 17 experienced similar levels of stress; and they experienced stress from both parent and peer sources. Both sources of stress significantly predicted depressive outcomes defined by CDI, NA, and PA scores. The percentage of variance accounted for by these relationships is substantial, although less so for PA. More specifically, the positive relationship between these variables and CDI and NA indicates that as stress in both domains increase, so do depressive symptoms. The negative relationship between these variables and PA indicates that as stress in both domains increases PA decreases. In these analyses then, the components of the tripartite model are demonstrating expected relationships (i.e., high NA and low PA scores coincide with high CDI scores).

While it is true that male and female adolescents experienced similar levels of stress from these domains, their reactions to the stress may have been different. While reaction to parent and peer stress specifically was not measured in this study, coping style in general was. Results from Hypothesis 2 indicate significant sex differences in coping style. Extrapolation of that finding here may suggest that male and female adolescents react to and cope with parent and peer stress differently despite the indication that they both experience similar levels of stress. Even though levels of stress are similar, we have

evidence to suggest that adolescent females may ruminate about stress originating from both parents and peers more so than their male counterparts. This interpretation is an extension of the Different Factors mode noted by Nolen-Hoeksema (1994) and discussed in detail under Hypothesis 2.

Despite the commonly held notion that peer influence during adolescence overrides parental influence (perhaps to the exclusion of the latter), the data here tells us that both factors are important with respect to depressive symptomatology. The clinical significance of this result suggests that both domains (parent and peer stress) may be relevant, and should at the very least be considered, in a prevention or treatment context. If parent and peer stress levels can be lowered (or at least responded to in more proactive ways) then depressive scores may also decrease and PA scores may increase. This research supports the link between stressful life events (in parent and peer domains) and depression in adolescents. It also reinforces the idea that the reactions people have to such stressful life events (i.e., coping style) are important mechanisms to evaluate.

Strengths and Limitations

The research design employed by Seiffge-Krenke and Stemmler (2002) served as a framework for the current study. In this project, however, we used scales that offered more sophisticated and comprehensive psychometric properties when they existed. Most of the scales that were included had previously been used with Canadian adolescents. We also were able to include multiple measures for depressive symptoms that allowed us to comparatively evaluate how such symptoms are defined[9]. This design allowed us to have

[9] The Likert-style response options for the PANAS-C were printed in the opposite format as in the original scale (i.e., from 5 to 1 rather than 1 to 5), however the label applied to each response matched with the original and the key provided. While some could speculate that this reflection may have contributed to some inconsistency in scores, it seems likely that this difference produced little effect. The NA and PA variables

more confidence that the final results were due to the proposed constructs being tested rather than measurement error or poor construct validity. A cross-sectional group of adolescents was accessed through multiple schools in the local district and was considered to be a representative sample of Canadian adolescents attending school. The sample was however, nonclinical, and interpretations must be made within that context.

Although there are advantages to using self-report questionnaires (e.g., acceptable in a school setting, time efficient, demonstrated reliability and validity), there are also some interpretive drawbacks. Self-report methodologies really assess the individual's perception of their situation at the time, as opposed to accurate reporting of clinical symptoms. Caution is warranted not to extend the conclusions from this study to the development of clinical depression in all adolescents. A larger scale study that includes clinical interviews as the measure of depressive symptoms would be required to inform if it is appropriate to generalize the findings from this study.

Although we included race as a demographic variable, some participants had difficulty responding to this item, as reflected in the high percentage of "other" noted (almost 17%). Just over 75% of the sample identified themselves as Caucasian, and almost 6% were of Aboriginal/First Nations decent. Although the sample does demonstrate some ethnic diversity, the exact nature is less well defined.

As mentioned in the introduction, the organizational influence of hormones is likely important with respect to the sex differences debate. Practical limitations inherent in accessing adolescents in their school setting did not allow direct hormone indicators to be collected in this study. We therefore had to rely on indirect organizational indices. One

---

performed consistently as expected compared to other indicators of depressive symptoms, the means and *SD*s were comparable to other adolescent samples (Laurent et al. 1999), and internal consistency values were high (i.e., Cronbach's alphas of .91 and .92 for PA and NA subscales).

could argue that activational hormonal effects could have an equal or more powerful influence on this outcome than organizational effects. A comparative evaluation between activational and organizational influences within one study could provide a stronger test of this hypothesis. We are therefore limited by the absence of this comparison in the conclusions we can draw about hormonal influences from our findings. Related to this, we were also unable to adequately test the importance of menarchal events in relation to sex differences in depression. A sample with more premenarchal females would allow a more powerful statistical evaluation of that hypothesis.

The finger ridge count variable was not as easy to collect as the other variables. It was difficult to get clear "readable" prints for some participants despite repeated attempts made by the primary investigator. This led to a significant portion of missing data. Conducting the study from a standard laboratory with additional materials available may improve the chances of getting better quality prints for more participants. The reduction in cases available for the finger ridge count analyses likely contributed to a decrease in power to detect significant effects for this variable if they existed.

This study was not designed as an experimental study. It would not have been possible to manipulate some of the variables under investigation (i.e., we cannot alter what the prenatal environment was like, we cannot randomly vary parent and peer stressors). Therefore, all results were informed by correlational analyses. As such, no firm conclusions can be drawn. Replication of the findings using refined methods will instill confidence in the results.

Directions for Future Study

Throughout this investigation some questions posed in the beginning of this document have been addressed. At the same time, more questions have been raised. Since this was not a longitudinal study, we have been unable to investigate the continuity of depression in youth over time. Insight into the "real time" progression would be an informative, but formidable undertaking to tackle. At least one short-term longitudinal study has been conducted (Abela et al., 2002). The next logical step then is to expand time between measures and include more opportunities for measurements. Research in the area of anxiety disorders does indicate that many youth with depression have experienced a past history of anxiety problems (Collins et al., 2004; Dahl & Ryan, 1996). This may be a fruitful avenue to explore further with respect to targets for prevention. That is, identify those adolescents who experience anxiety problems and direct prevention efforts there so those preadolescents don't go on to develop depressive episodes.

This project included components from competing theories as main variables (e.g., ruminative coping, putative prenatal hormone indicators). Future studies might consider including alternative measures of the same components to overcome some of the limitations encountered in this study. For example, organizational hormone effects were evaluated in this study. It would be interesting to include both organizational and activational measures in one study to investigate the current hypotheses through both means. Wet hormone indicators could serve as a more direct measure of real time hormone fluctuation and enable a comparison against organizational hormone effects. To rule out any confounds with respect to the organizational 2D:4D variable discussed above, it would

be ideal to have scan and calliper measures for the full sample. That way analyses could be conducted with measures from both methods.

A search for additional mediating factors (e.g., hopelessness, low self-esteem, locus of control) could also be informative in terms of identifying what additional characteristics continue to put girls at increased risk for depression. With respect to rumination, Muris, Roelofs, Meesters, and Boomsma (2004) have noted that worry may play a bigger role in predicting depression symptoms in youth than rumination. When both rumination and worry (related but distinct constructs) scales were included in their study, rumination no longer accounted for a significant proportion of variance in depressive symptoms. If a prospective design could be utilized across a large enough age span, a developmental sequence may be exposed that progresses from worry to rumination to depression. Measures of alternative coping styles (e.g., avoidant, active vs. passive) could also be included to further elucidate which aspects of coping contribute to such an effect or protect against such an effect. An alternative way to explore the sex differences in depression among youth may be to investigate the factors of resiliency that protect against the development of ruminative coping styles. Insight into why or how some people avoid the development of such a coping style may inform treatment approaches.

An ethnically diverse sample could be accessed to determine if the developmental sequence is equally applicable to adolescents from varying backgrounds. As well, a multi-informant approach could address the limitations inherent in self-report studies. That is, if scores from clinician rated measures, parent or teacher report, and self-report all support the same interpretation, more confidence in those results would be warranted. While the

literature on this topic is growing at a rapid rate, much remains to be explored and replicated before firm conclusions can be drawn.

Conclusion

It was the intent of this paper to integrate findings from each component of the biopsychosocial model. As a whole, this research project has clarified some points of inquiry that relate to sex differences in adolescents. Some components of the RST and tripartite models are applicable to adolescents. Specifically, rumination and NA seem to be important factors while distraction and PA do not. This may have implications for diagnostic criteria that are not applicable to youth (i.e., loss of interest or pleasure) given possible syndrome differences in the disorder itself. Sex appears to be a moderator of the relationship between pubertal status and depressive symptoms. Pubertal status seems to be a more important indicator for females than males; as pubertal status increases in females so do depressive symptoms. With respect to putative indirect markers of prenatal hormones, 2D:4D seems to be more relevant to depressive symptoms than does finger ridge count. Thus, organizational effects of prenatal hormones may represent the first link in a chain of events that eventually predisposes an individual to experience depressive symptoms. Both parent and peer stress domains contribute to the depressive experience for adolescents. As a whole, the hypotheses tended to reveal support for Nolen-Hoeksema and Girgus' (1994) Different Factors model. Further investigation would be required before support can be noted for the Interaction model (Nolen-Hoeksema & Girgus, 1994). Other points remained less clear after examination. For example, the typically robust relationship between chronological age and depressive symptoms was not apparent in this sample, nor was it clear why left 2D:4D was a stronger predictor than right 2D:4D.

This research has identified key variables that should be targeted in prevention or intervention programs (i.e., rumination, parent and peer stressors) and suggested approximate time frames of when such efforts may be most beneficial (i.e., before growth spurts, body hair or skin changes surface, breast development or menarche begins). Even so, it is important to highlight that depression is highly treatable (in children, adolescents, and adults), however, many children and adolescents go without treatment for depression (Collins et al., 2004). Although treatment protocols can be tailored to better address the adolescent experience of depression, barriers to seeking such treatment may still exist and should be evaluated.

Although direct inference from organizational effects (prenatal hormone indicators) to activational effects (current mental health functioning) is not possible given the design of this study, it is worthwhile considering the possible extrapolation of findings. That is, organizational hormone effects may provide a window into what activational hormone effects will surface during adolescence. Although Lutchmaya et al. (2004) have made initial efforts in this direction (i.e., they evaluated this relationship from prenatal exposure to infancy), the possibility over a longer period remains to be explored.

It is important to keep in mind that none of the results rule out the contribution of other factors to the development of depressive symptoms in adolescence. In fact, the data here support the proposal that a biopsychosocial model is *necessary* to fully appreciate the factors involved. This multimodal understanding continues to grow. When single factor designs are utilized, only a limited interpretation is possible (Kendler, Gardner, & Prescott, 2002). Without inclusion of multiple factors, it is impossible to evaluate the relative contribution of competing factors. Only through studies that comparatively investigate

competing models can one achieve a comprehensive understanding. Besides rumination, other cognitive models were not included in the current study simply to keep the questionnaire package to a manageable size given time constraints. As a whole in the literature, cognitive models of depression have consistently received support in their role of depression in youth (Abramson et al., 2002; Cicchetti & Toth, 1998; Flett, Madorsky, Hewitt, Heisel, 2002). It was the aim of this dissertation to focus on models that have been less consistently reviewed and supported. Although not discussed as part of this research study, other cognitive factors are relevant and should be considered an important piece of the picture.

References

Abela, J., Brozina, K., & Haigh, E. (2002). An examination of the response styles theory of depression in third- and seventh-grade children: A short-term longitudinal study. *Journal of Abnormal Child Psychology, 30*, 515-527.

Abela, J., Rochon, A., & Vanderbilt, E. (2000). *The Children's Response Style Questionnaire (Unpublished questionnaire)*. Montreal, Canada: McGill University.

Abramson, L., Alloy, L., Hankin, B., Haeffel, G., MacCoon, D., & Gibb, B. (2002). Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In I. Gotlib & C. Hammen (Eds.), *Handbook of depression* (pp. 268-294). New York: The Guildford Press.

Ahokas, A., Kaukoranta, J., Wahlbeck, K., & Aito, M. (2001). Estrogen deficiency in severe postpartum depression: Successful treatment with sublingual physiologic 17beta-estadiol: A preliminary study. *Clinical Psychiatry, 62*, 332-336.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, Fourth edition, Text revision*. Washington, DC: American Psychiatric Assoication.

Andrews, M., & Matthews, S. (2004). Programming of the hypothalamo-pituitary-adrenal axis: Serotonergic involvement. *Stress, 7*, 15-27.

Angold, A., Costello, E., Erkanli, A., & Worthman, C. (1999). Pubertal changes in hormone levels and depression in girls. *Psychological Medicine, 29*, 1043-1053.

Angold, A., Costello, E., & Worthman, C. (1998). Puberty and depression: The roles of age, pubertal status, and pubertal timing. *Psychological Medicine, 28*, 51-61.

Bailey, A., & Hurd, P. (2005). Finger length ratio (2D:4D) correlates with physical aggression in men but nor in women. *Biological Psychology, 68*, 215-222.

Beall, A. E., Eagly, A. H., & Sternberg, R. J. (2004). Introduction. In A. H. Eagly & A. E. Beall & R. J. Sternberg (Eds.), *The psychology of gender* (second ed., pp. 1-8). New York, NY: The Guildford Press.

Birkhauser, M. (2002). Depresion, menopause and estrogens: Is there a correlation? *Maturitas, 41*(suppl 1), S3-S8.

Born, L., Shea, A., & Steiner, M. (2002). The roots of depression in adolescent girls: Is menarche the key? *Current Psychiatry Reports, 4*, 449-460.

Broderick, P. (1998). Early adolescent gender differences in the use of ruminative and distracting coping strategies. *Journal of Early Adolescence, 18*, 173-191.

Brooks-Gunn, J., Graber, J., & Paikoff, R. (1994). Studying links between hormones and negative affect: Models and measures. *Journal of Research on Adolescence, 4*, 469-486.

Brooks-Gunn, J., Warren, M., Rosso, J., & Gargiulo, J. (1987). Validity of self-report measures of girls' pubertal status. *Child Development, 58*, 829-841.

Butler, L. D., & Nolen-Hoeksema, S. (1994). Gender differences in responses to depressed mood in a college sample. *Sex Roles, 30*, 331-346.

Carskadon, M., & Acebo, C. (1993). A self-administered rating scale for pubertal development. *Journal of Adolescent Health, 14*, 190-195.

Cicchetti, D., & Toth, S. (1998). The development of depression in children and adolescents. *American Psychologist, 53*, 221-241.

Clark, L., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*, 316-336.

Clark, L., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology, 103*, 103-116.

Cohen-Bendahan, C., van de Beek, C., & Berenbaum, S. (2005). Prenatal sex hormone effects on child and adult sex-typed behavior: Methods and findings. *Neuroscience and Biobehavioral Reviews, 29*, 353-384.

Coleman, J. (1989). The focal theory of adolescence: A psychological perspective. In K. Hurrelmann & U. Engel (Eds.), *The social world of adolescents: International perspectives* (pp. 43-56). Berlin: Walter de Gruyter & Co.

Coleman, L., & Coleman, J. (2002). The measurement of puberty: A review. *Journal of Adolescence, 25*, 535-550.

Collins, K., Westra, H., Dozois, D., & Burns, D. (2004). Gaps in accessing treatment for anxiety and depression: Challenges for the delivery of care. *Clinical Psychology Review, 24*, 583-616.

Compas, B., Davis, G., Forsythe, C., & Wagner, B. (1987). Assessment of major and daily stressful events during adolescence: The Adolescent Perceived Events Scale. *Journal of Consulting and Clinical Psychology, 55*, 534-541.

Compas, B., & Oppedisano, G. (2000). Mixed anxiety/depression in childhood and adolescence. In A. Sameroff & M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 531-548). New York: Kluwer.

Crandall, V., Crandall, V., & Katkovsky, W. (1965). A children's social desirability questionnaire. *Journal of Consulting Psychology, 29*, 37-36.

Cyranowski, J., Frank, E., Young, E., & Shear, K. (2000). Adolescent onset of gender difference in lifetime rates of major depression: A theoretical model. *Archives of General Psychiatry, 57*, 21-27.

Dahl, R., & Ryan, N. (1996). The psychobiology of adolescent depression. In D. Cicchetti & S. Toth (Eds.), *Adolescence: Opportunities and challenges* (pp. 197-232). Rochester, NY: University of Rochester Press.

de Matos, M., Barrett, P., Dadds, M., & Shortt, A. (2003). Anxiety, depression, and peer relationships during adolescence: Results from the Portuguese national health behaviour in school-aged children survey. *European Journal of Psychology of Education, 18*, 3-12.

Dick, D., Rose, R., Pulkkinen, L., & Kaprio, J. (2001). Measuring puberty and understanding its impact: A longitudinal study of adolescent twins. *Journal of Youth and Adolescence, 30*, 385-399.

Dorn, L., Dahl, R., Williamson, D., Birmaher, B., Axelson, D., Perel, J., Stull, S., & Ryan, N. (2003). Developmental markers in adolescence: Implications for studies of pubertal processes. *Journal of Youth and Adolescence, 32*, 315-324.

Dorn, L. D., Susman, E., Nottelmann, E., Inoff-Germain, G., & Chrousos, G. (1990). Perceptions of puberty: Adolescent, parent, and health care personnel. *Developmental Psychology, 26*, 322-329.

Epperson, C., Wisner, K., & Yamamoto, B. (1999). Gonadal steroids in the treatment of mood disorders. *Psychosomatic Medicine, 61*, 676-697.

Fallon, E., & Hausenblas, H. (2005). Media images of the "ideal" female body: Can acute exercise moderate their psychological impact? *Body Image, 2*, 62-73.

Fergusson, D., Wanner, B., Vitaro, F., Horwood, L., & Swain-Campbell, N. (2003). Deviant peer affiliations and depression: Confounding or causation? *Journal of Abnormal Child Psychology, 31*, 605-618.

Fink, B., Manning, J., Neave, N., & Grammer, K. (2004). Second to fourth digit ratio and facial asymmetry. *Evolution and Human Behavior, 25*, 125-132.

Fink, B., Neave, N., Laughton, K., & Manning, J. (2006). Second to fourth digit ratio and sensation seeking. *Personality and Individual Differences, 41*, 1253-1262.

Fink, B., Neave, N., & Manning, J. (2003). Second to fourth digit ratio, body mass index, waist-to-hip ratio, and waist-to-chest ratio: Their relationships in heterosexual men and women. *Annals of Human Biology, 30*, 728-738.

Fleming, J., & Offord, D. (1990). Epidemiology of childhood depressive disorders: A critical review. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 571-580.

Flett, G., Madorsky, D., Hewitt, P., & Heisel, M. (2002). Perfectionism cognitions, rumination, and psychological distress. *Journal of Rational-Emotive & Cognitive-Behavior Therapy, 20*, 33-46.

Frazier, P., Tix, A., & Barron, K. (2004). Testing moderator and mediator effects in counseling psychology research. *Journal of Counseling Psychology, 51*, 115-134.

Galambos, N., Leadbeater, B., & Barker, E. (2004). Gender differences in and risk factors for depression in adolescence: A 4-year longitudinal study. *International Journal of Behavioral Development, 28*, 16-25.

Garber, J. (2000). Development and depression. In A. Sameroff & M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 467-490). New York: Kluwer.

Garber, J., & Horowitz, J. (2002). Depression in children. In I. Gotlib & C. Hammen (Eds.), *Handbook of depression* (pp. 510-540). New York: The Guildford Press.

Glover, V., & O'Connor, T. G. (2005). Effects of antenatal maternal stress or anxiety: From fetus to child. In B. Hopkins & S. P. Johnson (Eds.), *Prenatal development of postnatal functions* (pp. 221-245). Westport, Connecticut: Praeger.

Haahr, M (n.d.). True Random Number Service (Version 2.0 beta, Integer Generator) [Computer software]. Retrieved June 22, 2006, from http://www.random.org

Hammen, C. (2003). Interpersonal stress and depression in women. *Journal of Affective Disorders, 74*, 49-57.

Hampson, E., & Moffat, S. D. (2004). The psychobiology of gender: Cognitive effects of reproductive hormones in the adult nervous system. In A. H. Eagly & A. E. Beall & R. J. Sternberg (Eds.), *The Psychology of Gender* (second ed., pp. 38-64). New York, NY: The Guildford Press.

Hankin, B., & Abramson, L. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin, 127*, 773-796.

Hankin, B., Abramson, L., Moffitt, T., Silva, P., McGee, R., & Angell, K. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*, 128-140.

Harris, J. A., Rushton, J. P., Hampson, E., & Jackson, D. (1996). Salivary testosterone and self-report aggressive and pro-social personality characteristics in men and women. *Aggressive Behavior, 22*, 321-331.

Hazell, P., O'Connell, D., Heathcote, D., Robertson, J., & Henry, D. (1995). Efficacy of tricyclic drugs in treating child and adolescent depression: A meta-analysis. *British Medical Journal, 310*(6984), 897-901.

Herault, Y., Fradeau, N., Zakany, J., & Duboule, D. (1997). *Ulnaless* (Ul), a regulatory mutation inducing both loss-of-function and gain-of-function of posterior *Hoxd* genes. *Development, 124*, 3493-3500.

Hines, M. (2004). Androgen, estrogen, and gender. In A. H. Eagly & A. E. Beall & R. J. Sternberg (Eds.), *The psychology of gender* (second ed., pp. 9-37). New York, NY: The Guildford Press.

Holt, S. (1968). *The genetics of dermal ridges*. Springfield, IL: Charles C. Thomas.

Honekopp, J., Bartholdt, L., Beier, L., & Liebert, A. (2007). Second to fourth digit length ratio (2D:4D) and adult sex hormone levels: New data and a meta-analytic review. *Psychoneuroendocrinology, 32*, 313-321.

Ingram, R., Nelson, T., Steidmann, D., and Bistricky, S. (2007). Comparative data on child and adolescent cognitive measures associated with depression. *Journal of Consulting and Clinical Psychology, 75*, 390-403.

Jamison, C. S., Meier, R. J., & Campbell, B. C. (1993). Dermatoglyphic asymmetry and testosterone levels in normal males. *American Journal of Physical Anthropology, 90*, 185-198.

Joffe, H., & Cohen, L. (1998). Estrogen, serotonin, and mood disturbance: Where is the therapeutic bridge? *Biological Psychiatry. Special Issue: A decade of serotonin research: Regulation of affect and eating behavior, 44*, 798-811.

Johnson, F., & Wardle, J. (2005). Dietary restraint, body dissatisfaction, and psychological distress: A prospective analysis. *Journal of Abnormal Psychology, 114*, 119-125.

Kaiser, J., & Gruzelier, J. (1999). The Adolescence Scale (AS-ICSM): A tool for the retrospective assessment of puberty milestones. *Acta Paediatica, Suppl 429*, 64-68.

Kajantie, E., & Phillips, D. (2006). The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology, 31*, 151-178.

Kasch, K., Klein, D., & Lara, M. (2001). A construct validation study of the Response Styles Questionnaire rumination scale in participants with a recent-onset major depressive episode. *Psychological Assessment, 13*, 375-383.

Kashani, J., & Carlson, G. (1987). Seriously depressed preschoolers. *American Journal of Psychiatry, 144*, 348-350.

Kaslow, N., Adamson, L., & Collins, M. (2000). A developmental psychopathology perspective on the cognitive components of child and adolescent depression. In A. Sameroff & M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 491-510). New York: Kluwer.

Kaufman, J., Martin, A., King, R., & Charney, D. (2001). Are child-, adolescent-, and adult-onset depression one and the same disorder? *Biological Psychiatry, 49*, 980-1001.

Kendler, K., Gardner, C., & Prescott, C. (2002). Toward a comprehensive developmental model for major depression in women. *The American Journal of Psychiatry, 159*, 1133-1145.

Kiernan, G., Laurent, J., Joiner, T., Catanzaro, S., & MacLachlan, M. (2001). Cross-cultural examination of the tripartite model with children: Data from the Barretstown studies. *Journal of Personality Assessment, 77*, 359-379.

Kovacs, M. (1992). *Children's depression inventory: CDI manual.* Toronto, ON: Multi-Health Systems, Inc.

Kovacs, M., Devlin, B., Pollock, M., Richards, C., & Mukerji, P. (1997). A controlled family history study of childhood-onset depressive disorder. *Archives of General Psychiatry, 54*, 613-623.

Krahnstover Davison, K., & Susman, E. (2001). Are hormone levels and cognitive ability related during early adolescence? *International Journal of Behavioral Development, 25*, 416-428.

Kumar, C., McIvor, R., Davies, T., Brown, N., Papadopoulos, A., Wieck, A., Checkley, S., Campbell, I., & Marks, M. (2003). Estrogen administration does not reduce the rate of recurrence of affective psychosis after childbirth. *Journal of Clinical Psychiatry, 64*, 112-118.

Laurent, J., Catanzaro, S., & Joiner, T. (2004). Development and preliminary validation of the physiological hyperarousal scale for children. *Psychological Assessment, 16*, 373-380.

Laurent, J., Catanzaro, S., Joiner, T., Rudolph, K., Potter, K., Lambert, S., Osborne, L., & Gathright, T. (1999). A measure of positive and negative affect for children: Scale development and preliminary validation. *Psychological Assessment, 11*, 326-338.

Laurent, J., & Ettelson, R. (2001). An examination of the tripartite model of anxiety and depression and its application to youth. *Clinical Child and Family Psychology Review, 4*, 209-230.

Lee, L. C., & Rebok, G. W. (2002). Anxiety and depression in children: A test of the positive-negative affect model. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*, 419-426.

Lewinsohn, P., Rohde, P., & Seeley, J. (1998). Major depressive disorder in older adolescents: Prevalence, risk factors, and clinical implications. *Clinical Psychology Review, 18*, 765-794.

Lutchmaya, S., Baron-Cohen, S., Raggatt, P., Knickmeyer, R., & Manning, J. (2004). 2nd to 4th digit ratios, fetal testosterone and estradiol. *Early Human Development, 77*, 23-28.

Manning, J. (2002). *Digit ratio: A pointer to fertility, behavior, and health*. New Brunswick, NJ: Rutgers University Press.

Manning, J., Barley, L., Walton, J., Lewis-Jones, D., Trivers, R., Singh, D., Thornhill, R., Rohde, P., Bereczkei, T., Henzi, P., Soler, M., & Szwed, A. (2000). The 2nd:4th digit ratio, sexual dimorphism, population differences, and reproductive success: Evidence for sexually antagonistic genes? *Evolution and Human Behavior, 21*, 163-183.

Manning, J., Baron-Cohen, S., Wheelwright, S., & Sanders, G. (2001). The 2nd to 4th digit ratio and autism. *Developmental Medicine & Child Neurology, 43*, 160-164.

Manning, J., Scutt, D., Wilson, J., & Lewis-Jones, D. (1998). The ratio of 2nd to 4th digit length: A predictor of sperm numbers and concentrations of testosterone, lutenizing hormone and oestrogen. *Human Reproduction, 13*, 3000-3004.

Manning, J., Stewart, A., Bundred, P., & Trivers, R. (2004). Sex and ethnic differences in 2nd to 4th digit ratio of children. *Early Human Development, 80*, 161-168.

Manning, J., & Taylor, R. (2001). Second to fourth digit ratio and male ability in sport: Implications for sexual selection in humans. *Evolution and Human Behavior, 22*, 61-69.

Manning, J. T., Fink, B., Neave, N., & Caswell, N. (2005). Photocopies yield lower digit ratios (2D:4D) than direct finger measurements. *Archives of Sexual Behavior, 34*, 329-333.

Marcotte, D., Fortin, L., Potvin, P., & Papillon, M. (2002). Gender differences in depressive symptoms during adolescence: Role of gender-typed characteristics, self-esteem, body image, stressful life events, and pubertal status. *Journal of Emotional & Behavioral Disorders, 10*, 29-42.

Martin, S., Manning, J., & Dowrick, C. (1999). Fluctuating asymmetry, relative digit length, and depression in men. *Evolution and Human Behavior, 20*, 203-214.

McFarland, K., & Anderson, J. (1980). Factor stability of the Edinburgh Handedness Inventory as a function of test-retest performance, age and sex. *British Journal of Psychology, 71*, 135-142.

McMeekan, E. R. L., & Lishman, W. A. (1975). Retest reliabilities and interrelationship of the Annett Hand Preference Questionnaire and the Edinburgh Handedness Inventory. *British Journal of Psychology, 66*, 53-59.

Mead, L. A., & Hampson, E. (1997). Turning bias in humans is influenced by phase of the menstrual cycle. *Hormones and Behavior, 31*, 65-74.

Migeon, C. J., & Wisniewski, A. B. (1998). Sexual differentiation: From genes to gender. *Hormone Research, 50*, 245-251.

Miller, K., Conney, J., Rasgon, N., Fairbanks, L., & Small, G. (2002). Mood symptoms and cognitive performance in women estrogen users and nonusers and men. *Journal of the American Geriatrics Society, 50*, 1826-1830.

Muris, P., Roelofs, J., Meesters, C., & Boomsma, P. (2004). Rumination and worry in nonclinical adolescents. *Cognitive Therapy and Research, 28*, 539-554.

Muris, P., Van Brakel, A., & Meesters, C. (1998). Coping styles. anxiety, and depression in children. *Psychological Reports, 83*, 1225-1226.

Newcomb, M., Huba, G., & Bentler, P. (1981). A multidimensional assessment of stressful life events among adolescents: Derivation and correlates. *Journal of Health and Social Behavior, 22*, 400-415.

Nolen-Hoeksema, S. (1990). Sex differences in depression during childhood and adolescence. In S. Nolen-Hoeksema (Ed.), *Sex differences in depression* (pp. 178-196). Stanford, CA: Stanford University Press.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569-582.

Nolen-Hoeksema, S. (1998). Ruminative coping with depression. In J. Heckhausen & C. Dweck (Eds.), *Motivation and self-regulation across the life span* (pp. 237-256). New York, NY: Cambridge Press.

Nolen-Hoeksema, S. (2002). Gender differences in depression. In I. Gotlib & C. Hammen (Eds.), *Handbook of depression* (pp. 492-509). New York: The Guildford Press.

Nolen-Hoeksema, S., & Girgus, J. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin, 115*, 424-443.

Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology, 77*, 1061-1072.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Personality and Social Psychology, 61*, 115-121.

Nottelmann, E., Susman, E., Dorn, L., Inoff-Germain, G., Loriaux, L., Cutler, G., & Chrousos, G. (1987). Developmental processes in early adolescence: Relations among chronologic age, pubertal stage, height, weight, and serum levels of gonadotropins, sex steroids, and adrenal androgens. *Journal of Adolescent Health, 8*, 246-260.

Nottelmann, E., Susman, E., Inoff-Germain, G., Cutler, G., Loriaux, L., & Chrousos, G. (1987). Developmental processes in early adolescence: Relationships between adolescent adjustment problems and chronologic age, pubertal stage, and puberty-related serum hormones. *The Journal of Pediatrics, 110*, 473-480.

O'Connor, T. G., Ben-Shlomo, Y., Heron, J., Golding, J., Adams, D., & Glover, V. (2005). Prenatal anxiety predicts individual differences in cortisol in pre-adolescent children. *Biological Psychiatry, 58*, 211-217.

Oinonen, K., & Mazmanian, D. (2002). To what extent do oral contraceptives influence mood and affect? *Journal of Affective Disorders, 70*, 229-240.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia, 9*, 97-113.

Papageorgiou, C., & Wells, A. (2004). Nature, functions, and beliefs about depressive rumination. In C. Papageorgiou & A. Wells (Eds.), *Depressive rumination: Nature, theory and treatment* (pp. 1-20). Chichester, England: John Wiley & Sons Ltd.

Papaioannou, A., Gerozissis, K., Prokopiou, A., Bolaris, S., & Stylianopoulou, F. (2002). Sex differences in the effects of neonatal handling on the animal's response to stress and the vulnerability for depressive behaviour. *Behavioural Brain Research, 129*, 131-139.

Parker, J., & Asher, S. (1987). Peer relations and later personal adjustment: Are low-accepted children at risk? *Psychological Bulletin, 102*, 357-389.

Paulhus, D. (2002). Socially desirable responding: The evolution of a construct. In H. Braun & D. Jackson & D. Wiley (Eds.), *The role of constructs in psychological and educational measurement* (pp. 49-69). Mahwah, NJ: Lawrence Erlbaum Associates, Publishers.

Petersen, A., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence, 17*, 117-133.

Piccinelli, M., & Gomez Homen, F. (1997). Gender differences in the epidemiology of affective disorders and schizophrenia (pp. 3-59). Geneva: World Health Organization.

Piccinelli, M., & Wilkinson, G. (2000). Gender differences in depression: Critical review. *British Journal of Psychiatry, 177*, 486-492.

Polani, P. (1981). Chromosomes and chormosomal mechanisms in the genesis of maldevelopment. In K. J. Connolly & H. F. R. Prechtl (Eds.), *Maturation and Development: Biological and Psychological Perspectives* (pp. 50-72). Lavenham: Suffolk: The Lavenham Press Ltd.

Poulin, M., O'Connell, R., & Freeman, L. (2004). Picture recall skills correlate with 2D:4D ratio in women but not men. *Evolution and Human Behavior, 25*, 174-181.

Putz, D., Gaulin, S., Sporter, R., & McBurney, D. (2004). Sex hormones and finger length: What does 2D:4D indicate? *Evolution and Human Behavior, 25*, 182-199.

Ramirez, J. (2003). Hormones and aggression in childhood and adolescence. *Aggression and Violent Behavior, 8*, 621-644.

Rasgon, N., Altshuler, L., Fairbanks, L., Dunkin, J., Davtyan, C., Elman, S., & Rapkin, A. (2002). Estrogen replacement therapy in the treatment of major depressive disorder in perimenopausal women. *Clinical Psychiatry, 63*(suppl 7), 45-48.

Reise, S., & Henson, J. (2003). A discussion of modern versus traditional psychometrics as applied to personality assessment scales. *Journal of Personality Assessment, 81*, 93-103.

Reynolds, C., & Richmond, B. (1997). *Revised children's manifest anxiety scale: RCMAS manual.* Los Angeles, CA: Western Psychological Services.

Robertson, E., Skinner, M., Love, M., Elder, G., Conger, R., Dubas, J., & Petersen, A. (1992). The Pubertal Development Scale: A rural and suburban comparison. *Journal of Early Adolescence, 12*, 174-186.

Robinson, M., & Alloy, L. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research, 27*, 275-292.

Robinson, S., & Manning, J. (2000). The ratio of 2nd to 4th digit length and male homosexuality. *Evolution and Human Behavior, 21*, 333-345.

Rudolph, K. (2002). Gender differences in emotional responses to interpersonal stress during adolesence. *Journal of Adolescent Health, 30*(4), 3-13.

Rudolph, K., & Asher, S. (2000). Adaptation and maladaptation in the peer system: Developmental processes and outcomes. In A. Sameroff & M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 157-175). New York: Kluwer.

Salmaso, D., & Longoni, A. M. (1985). Problems in the assessment of hand preference. *Cortex, 21*, 533-549.

Sanders, G., & Kadam, A. (2001). Prepubescent children show the adult relationship between dermatoglyphi asymmetry and performance on sexually dimorphic tasks. *Cortex, 37*, 91-100.

Sanders, G., Sjodin, M., & de Chastelaine, M. (2002). On the elusive nature of sex difference in cognition: Hormonal influences contributing to within-sex variation. *Archives of Sexual Behavior, 31*, 145-152.

Schneider, M., Roughton, E. C., Koehler, A. J., & Lubach, G. R. (1999). Growth and development following prenatal stress exposure in primates: An examination of ontogenetic vulnerability. *Child Development, 70*, 263-274.

Schwartz, J., & Koenig, L. (1996). Response styles and negative affects among adolescents. *Cognitive Therapy and Research, 20*, 13-36.

Seiffge-Krenke, I. (1995). *Stress, coping, and relationships in adolescence*. Mahwah, NJ: Lawrence Erlbaum Associates, Publishers.

Seiffge-Krenke, I., & Klessinger, N. (2000). Long-term effects of avoidant coping on adolescents' depressive symptoms. *Journal of Youth and Adolescence, 29*, 617-630.

Seiffge-Krenke, I., & Stemmler, M. (2002). Factors contributing to gender differences in depressive symptoms: A test of three developmental models. *Journal of Youth and Adolescence, 31*, 405-417.

Seligson, J., Huebner, E., & Valois, R. (2003). Preliminary validation of the Brief Multidimensional Students' Life Satisfaction Scale (BMSLSS). *Social Indicators Research, 61*, 121-145.

Silberg, J., Pickles, A., Rutter, M., Hewitt, J., Simonoff, E., Maes, H., Caronneau, R., Murrelle, L., Foley, D., & Eaves, L. (1999). The influence of genetic factors and life stress on depression among adolescent girls. *Archives of General Psychiatry, 56*, 225-232.

Sloboda, D., Newnham, J., Moss, T., & Challis, J. (2006). The fetal hypothalamic-pituitary-adrenal axis: Relevance to developmental origins of health and disease. In P. Gluckman & M. Hanson (Eds.), *Developmental origins of health and disease* (pp. 191-205). New York: Cambridge University Press.

Spasojevic, J., & Alloy, L. (2001). Rumination as a common mechanism relating depressive risk factors to depression. *Emotion, 1*, 25-37.

Stahl, S. (1998). Basic psychopharmacology of antidepressants, part 2: Estrogen as an adjunct to antidepressant treatment. *Journal of Clinical Psychiatry, 59*(suppl 4), 15-24.

Stattin, H., & Magnusson, D. (1990). *Pubertal maturation in female development* (Vol. 2). Hillsdale, NJ: Lawrence Erlbaum Associates.

Steiner, M., Born, L., & Marton, P. (2000). Menarche and mood disorders in adolescence. In M. Steiner & K. Yonkers & E. Eriksson (Eds.), *Mood disorders in women.* London, UK: Martin Dunitz Ltd.

Steiner, M., Dunn, E., & Born, L. (2003). Hormones and mood: From menarche to menopause and beyond. *Journal of Affective Disorders, 74*, 67-83.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using multivariate statistics* (4th ed.). Needham Heights, MA: Allyn & Bacon.

Tanner, J. (1962). *Growth at adolescence.* Oxford, England: Blackwell Scientific Publications.

Tanner, J. (1978). *Foetus into man: Physical growth from conception to maturity.* Cambridge, MA: Harvard University Press.

Thase, M., Jindal, R., & Howland, R. (2002). Biological aspects of depression. In I. Gotlib & C. Hammen (Eds.), *Handbook of depression* (pp. 192-218). New York: The Guildford Press.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27*, 247-259.

van Anders, S., & Hampson, E. (2005). Testing the prenatal androgen hypothesis: Measuring digit ratios, sexual orientation, and spatial abilities in adults. *Hormones and Behavior, 47*, 92-98.

Watson, D., Clark, L., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.

Watson, D., Weber, K., Assenheimer, J., Clark, L., Strauss, M., & McCormick, R. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 3-14.

Weinstock, M. (2001). Alterations induced by gestational stress in brain morphology and behavior of the offspring. *Progress in Neurobiology, 65*, 427-451.

Welberg, L., & Secki, J. (2001). Prenatal stress, glucocorticoids and the programming of the brain. *Journal of Neuroendocrinology, 13*, 13-128.

Welsh, J., & Mazmanian, D. (2007). *Exploring gender differences in depression using the Children's Response Styles Scale.* Poster presented at the annual meeting of the Canadian Psychological Association, Ottawa, ON.

Weiss, B., & Garber, J. (2003). Developmental differences in the phenomenology of depression. *Development and Psychopathology, 15*, 403-430.

Williams, T., Pepitone, M., Christensen, S., Cooke, B., Huberman, A., Breedlove, N., Breedlove, T., Jordan, C., & Breedlove, S. (2000). Finger-length ratios and sexual orientation. *Nature, 404*, 455-456.

Wolk, S., & Weissman, M. (1995). Women and depression: An update. In J. Oldham & M. Riba (Eds.), *American Psychiatric Press Review of Psychiatry* (Vol. 14, pp. 227-259). Washington, DC: American Psychiatric Association.

Ziegert, D., & Kistner, J. (2002). Response styles theory: Downward extension to children. *Journal of Clinical Child and Adolescent Psychology, 31*, 325-334.

Appendix A

Cover Letter & Consent Form for Parents

Dear Parent or Guardian,

I am a psychology professor at Lakehead University and I am writing to request your permission to ask your son or daughter if he or she would like to participate in a study that I am conducting with a doctoral student, Jennifer Welsh. The purpose of this project is to look at changes that occur in moods during the teenage years. In this study your son or daughter will be asked to complete a questionnaire and allow measurements (digit length and finger prints) of his or her hands to be taken. This process should take approximately one hour and will likely be completed during class time.

This study has been approved by the Research Ethics Board of Lakehead University, by the Lakehead Public School Board, and by the school principal. Participants will be informed of the nature of the study and will be asked if they would like to participate. The intent is purely to gain knowledge and there are no known risks associated with this research. Participants will have their names entered in a draw for four $25 prizes.

Participants will be told beforehand that they are volunteers and are free to withdraw at any time without consequence. All responses will remain completely anonymous and will be kept confidential. No names are required on the questionnaires, and the consent forms will be kept separate from questionnaire responses. No one but the researchers will be given access to your son or daughter's responses. Your son or daughter will never be individually identified by his or her responses. The data will be stored in a secure laboratory at the University for at least seven years. Participants are free to inquire about the results once the data have been analyzed.

Please sign below, indicating whether or not you consent to let your son or daughter participate in this study. A signature of permission indicates that you agree to allow your son or daughter to participate in the study as outlined by the conditions above. Should you have any questions, please feel free to contact me. I can be reached at 343-8257, or by e-mail at dmazmani@lakeheadu.ca. Thank you very much for taking the time to consider this request.

Sincerely,

Dwight Mazmanian, Ph.D., C. Psych.
Associate Professor of Psychology

Circle your choice:

I permit / do not permit my son or daughter to participate in the study.

Name of son or daughter (please print): _____

Signature of parent/guardian: _____

Date: _____

Appendix B

Cover Letter & Consent Form for Participants

Dear Participant,

We are from Lakehead University and we would very much appreciate your help in a study we are conducting. It involves completing a questionnaire and allowing measurements of your hands to be taken (digit length and finger prints). The time required to complete this study should be approximately one hour. The purpose of the study is to look at changes that occur in moods during the teenage years.

The study has been approved by the Research Ethics Board of Lakehead University, by the Lakehead Public School Board, and by your principal. The intent is purely to gain knowledge and there are no known risks to you by participating. Your contributions will remain entirely confidential and you are free to withdraw at any time. You are also free to inquire about the results after data analyses have been completed.

If you agree to participate in this study, your name will be entered in a draw for four $25 prizes. The draw will take place when data collection is complete (likely Spring 2006).

Your signature below indicates the following:

1. I understand that I am a volunteer and may withdraw at any time.
2. I understand that there are no known risks to participating in this study.
3. I understand that my responses will remain completely anonymous and confidential.
4. I understand that no one else but the researchers will be given access to my responses, and that I will never be individually identified based on my responses.
5. I understand that data will be stored in a secure laboratory at the University for at least seven years.
6. I understand that I can contact the researchers if I have any questions (contact information will be provided in the debriefing handout).

Please circle your choice below.

I will / will not participate in the study.

Name of participant (please print): _____

Signature of participant: _____

Date: _____

Appendix C

☺ Tell Us a Little About You ☺

Please do not put your name on this page!!

1. Today's Date: _____ 2. Your age: _____ years

3. Your grade: _____ 4. Your gender: ☐ Male ☐ Female

5. Which ethnic or cultural background do you most closely identify with?

☐ Aboriginal/First Nations ☐ African ☐ African American

☐ Asian ☐ Caucasian ☐ Other _____

6. Are you currently taking any medications? This includes "over the counter" medicines as well as prescriptions. Some examples include: Paxil, Prozac, Luvox, Zoloft, Effexor, Celexa, Triphasil, Alesse, Ortho Tri-Cyclen, Flovent, Flonase, Ventolin, Singulair, Allegra, Clarinex.

☐ Yes ☐ No

7. If you said "yes" to number 6, what medications are you taking?

Name(s): _____ Dose (if known): _____

Name(s): _____ Dose (if known): _____

8. To the best of your knowledge, do you have any family members (i.e., biological relatives) who have a mental health problem (e.g., depression, alcoholism, anxiety)?

☐ Yes ☐ No ☐ Maybe/not sure

9. If you said "yes" to number 8, please list the family member and the issue. Please **do not list names**, only their relation to you (e.g., mother, father, sibling, cousin, grandparent).

Member: _____ Issue(s): _____

Member: _____ Issue(s): _____

10. Is there a male adult or male grown-up who lives in your home?

☐ Yes ☐ No

11. If you said "yes" to number 10, please check the one that applies.

☐ Biological father ☐ Step/Foster/Adoptive father

☐ Biological brother ☐ Step/Foster/Adoptive/half brother

☐ Other (e.g., boyfriend, other family members, borders)

12. Who else lives in your home with you? Check all that apply and list how many of each sibling lives with you.

☐ Biological mother ☐ Step/Foster/Adoptive mother

☐ Sister(s) ____ ☐ Brother(s) ____

☐ Step/Foster/Adoptive/Half sister(s) ____ ☐ Step/Foster/Adoptive/Half brother(s) ____

13. Have you ever broken or fractured any fingers? ☐ Yes ☐ No

14. If you said "yes" to number 13, which finger(s)? (e.g., thumb, index, middle, ring, pinky). _____

15. Please use the following scale to indicate which hand you use for each activity:

1	2	3	4	5
left		both		right

Write the number beside each item.

1. Writing _____
2. Throwing _____
3. Using scissors _____
4. Using a toothbrush _____
5. Using a knife without a fork _____
6. Using a spoon _____
7. Striking a match _____

Appendix D

Children's Response Styles Scale (CRSS)

On this sheet is a list of thoughts and behaviors that children and teens sometimes use to respond to life situations. Please indicate *how often* you use each thought or behavior by circling the appropriate number:

1. I think back to other times when I felt this way.

0	1	2	3	4	5	6	7	8	9	10
never										always

2. I think about how I should have done something different.

0	1	2	3	4	5	6	7	8	9	10
never										always

3. I think about something I did a little while ago that was a lot of fun.

0	1	2	3	4	5	6	7	8	9	10
never										always

4. I go away by myself and think about why I feel this way.

0	1	2	3	4	5	6	7	8	9	10
never										always

5. I do something I really like to do.

0	1	2	3	4	5	6	7	8	9	10
never										always

6. I think, "I'll concentrate on something other than how I feel."

0	1	2	3	4	5	6	7	8	9	10
never										always

7. I go someplace alone to think about my feelings.

0	1	2	3	4	5	6	7	8	9	10
never										always

8. I think, "Why can't I stop feeling this way?"

0	1	2	3	4	5	6	7	8	9	10
never										always

9. I think, "I'm going to do something to make myself feel better."

0	1	2	3	4	5	6	7	8	9	10
never										always

10. I do something that has made me feel better in the past.

0	1	2	3	4	5	6	7	8	9	10
never										always

11. I think about other times things didn't go the way I wanted them to.

0	1	2	3	4	5	6	7	8	9	10
never										always

12. I think about fun things.

0	1	2	3	4	5	6	7	8	9	10
never										always

13. I think about what made me feel like this.

0	1	2	3	4	5	6	7	8	9	10
never										always

14. I concentrate on something else that makes me happier.

0	1	2	3	4	5	6	7	8	9	10
never										always

15. I try to take my mind off my feelings by doing something I like.

0	1	2	3	4	5	6	7	8	9	10
never										always

16. I replay in my head what happened.

0	1	2	3	4	5	6	7	8	9	10
never										always

17. I think, "I'm going to go out and have some fun."

0	1	2	3	4	5	6	7	8	9	10
never										always

18. I think about a time when I was feeling much happier.

0	1	2	3	4	5	6	7	8	9	10
never										always

19. I think about my feelings.

0	1	2	3	4	5	6	7	8	9	10
never										always

20. I think about something that just happened, wishing it had gone better.

0	1	2	3	4	5	6	7	8	9	10
never										always

Appendix E

Pubertal Development Scale (PDS)

The next questions are about changes that may be happening to your body. These changes normally happen to different young people at different ages. Since they may have something to do with your sleep patterns, do your best to answer carefully. If you do no understand a question or do not know the answer, just mark "I don't know".

Circle the letter next to your choice.

1. Would you say that your growth in height:
    a) has not yet begun to spurt
    b) has barely started
    c) is definitely underway
    d) seems completed
    e) I don't know

2. And how about the growth of your body hair? ("Body hair" means hair any place other than your head, such as under your arms.) Would you say that your body hair growth:
    a) has not yet begun to grow
    b) has barely started to grow
    c) is definitely underway
    d) seems completed
    e) I don't know

3. Have you noticed any skin changes, especially pimples?
    a) skin has not yet started changing
    b) skin has barely started changing
    c) skin changes are definitely underway
    d) skin changes seem complete
    e) I don't know

Questions 4 and 5 are for BOYS only. Girls continue on to question 6.

4. Have you noticed a deepening of your voice?
    a) voice has not yet started changing
    b) voice has barely started changing
    c) voice changes are definitely underway
    d) voice changes seem complete
    e) I don't know

5. Have you begun to grow hair on your face?
    a) facial hair has not yet started growing
    b) facial hair has barely started growing
    c) facial hair has definitely started growing

d) facial hair growth seems complete
e) I don't know

BOYS you can continue after question 8. Questions 6, 7, and 8 are for GIRLS only.

6. Have you noticed that your breasts have begun to grow?
   a) have not yet started growing
   b) have barely started growing
   c) breast growth is definitely underway
   d) breast growth seems complete
   e) I don't know

7. Have you begun to menstruate (started to have your period)?
   a) yes
   b) no

8. If yes to number 7, how old were you when you started to menstruate?
   Age in years _____

Appendix F

Problem Questionnaire (PQ)

On the following pages you will find a list of worries and difficulties that adolescents of your age have identified as problems. Among these there will certainly be some that apply to you personally and some that hardly apply or not at all apply. Please indicate honestly and spontaneously how stressful the problems are for you.

Use the following scale to respond how you have found each problem to be:

5 4 3 2 1
highly stressful not stressful at all

Problems related to *life with parents at home*

1. My parents show little understanding for my problems in school. 5 4 3 2 1

2. My parents are only interested in my getting good marks in school. 5 4 3 2 1

3. I fight with my parents because my opinions about many things differ from theirs. 5 4 3 2 1

4. My parents think I'm "not all there." 5 4 3 2 1

5. My parents don't let me make my own decisions. 5 4 3 2 1

6. I can't talk with my parents. 5 4 3 2 1

7. My parents don't approve of my friends. 5 4 3 2 1

8. My parents don't have very much time for me. 5 4 3 2 1

9. It's difficult for me to pursue my own interests because I don't want to disappoint my parents. 5 4 3 2 1

10. I wish I wasn't so dependent on home (on my parents). 5 4 3 2 1

Problems related to *relationships with peers*

11. I hardly have any friends. 5 4 3 2 1

12. It's difficult for me to approach others. 5 4 3 2 1

13. I'm having difficulties combining my interests with those of my

friends.	5 4 3 2 1
14. I don't have a real friend with whom I can also talk about personal worries and problems.	5 4 3 2 1
15. Some of my acquaintances are dishonest and underhanded.	5 4 3 2 1
16. Many of my acquaintances are only willing to have superficial contact with me.	5 4 3 2 1
17. I am unsure if the others will accept me.	5 4 3 2 1
18. I don't like the fact that outsiders can't join existing cliques.	5 4 3 2 1
19. My peers are often very stubborn and intolerant toward each other.	5 4 3 2 1
20. I have too little time for my friends.	5 4 3 2 1

Appendix G

Positive and Negative Affect Schedule – Children (PANAS-C)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then circle the appropriate answer next to that word. Indicate to what extent you have felt this way during the past few weeks.

Use the following scale to respond:

1 very slightly or not at all	2 a little	3 moderately	4 quite a bit	5 extremely	
Interested	5	4	3	2	1
Sad	5	4	3	2	1
Frightened	5	4	3	2	1
Excited	5	4	3	2	1
Ashamed	5	4	3	2	1
Upset	5	4	3	2	1
Happy	5	4	3	2	1
Strong	5	4	3	2	1
Nervous	5	4	3	2	1
Guilty	5	4	3	2	1
Energetic	5	4	3	2	1
Scared	5	4	3	2	1
Calm	5	4	3	2	1
Miserable	5	4	3	2	1
Jittery	5	4	3	2	1
Cheerful	5	4	3	2	1
Active	5	4	3	2	1

Proud	5	4	3	2	1
Afraid	5	4	3	2	1
Joyful	5	4	3	2	1
Lonely	5	4	3	2	1
Mad	5	4	3	2	1
Disgusted	5	4	3	2	1
Delighted	5	4	3	2	1
Blue	5	4	3	2	1
Gloomy	5	4	3	2	1
Lively	5	4	3	2	1

Appendix H

Children's Social Desirability Scale (CSDS)

This questionnaire lists a number of experiences that most children have at one time or another. Read each of these carefully. After you have read one, decide whether it does or does not fit you. If it does, put a T (for true) in front of the statement; if it doesn't, put an F (for false) in front of the statement.

____ 1. I always enjoy myself at a party.

____ 2. I tell a little lie sometimes.

____ 3. I never get angry if I have to stop in the middle of something I'm doing to eat dinner, or go to school.

____ 4. Sometimes I don't like to share my things with my friends.

____ 5. I am always respectful of older people.

____ 6. I would never hit a boy or girl who was smaller than me.

____ 7. Sometimes I do not feel like doing what my teachers want me to do.

____ 8. I never act "fresh" or "talk back" to my mother or father.

____ 9. When I make a mistake, I always admit I am wrong.

____ 10. I feel my parents do not always show good judgment.

____ 11. I have never felt like saying unkind things to a person.

____ 12. I always finish all of my homework on time.

____ 13. Sometimes I have felt like throwing or breaking things.

____ 14. I never let someone else get blamed for what I did wrong.

____ 15. Sometimes I say something just to impress my friends.

____ 16. I am always careful about keeping my clothing neat, and my room picked up.

____ 17. I never shout when I feel angry.

____ 18. Sometimes I feel like staying home from school even if I am not sick.

____ 19. Sometimes I wish that my parents didn't check up on me so closely.

____ 20. I always help people who need help.

____ 21. Sometimes I argue with my mother to do something she doesn't want me to do.

____ 22. I never say anything that would make a person feel bad.

____ 23. My teachers always know more about everything than I do.

____ 24. I am always polite, even to people who are not very nice.

____ 25. Sometimes I do things I've been told not to do.

____ 26. I never get angry.

____ 27. I sometimes want to own things just because my friends have them.

____ 28. I always listen to my parents.

____ 29. I never forget to say "please" and "thank you".

____ 30. Sometimes I wish I could just "mess around" instead of having to go to school.

____ 31. I always wash my hands before every meal.

____ 32. Sometimes I dislike helping my parents even though I know they need my help around the house.

____ 33. I never find it hard to make friends.

____ 34. I have never been tempted to break a rule or a law.

____ 35. Sometimes I try to get even when someone does something to me I don't like.

____ 36. I sometimes feel angry when I don't get my way.

____ 37. I always help an injured animal.

____ 38. Sometimes I want to do things my parents think I am too young to do.

____ 39. I sometimes feel like making fun of other people.

____ 40. I have never borrowed anything without asking permission first.

____ 41. Sometimes I get annoyed when someone disturbs something I've been working on.

____ 42. I am always glad to cooperate with others.

____ 43. I never get annoyed when my best friend wants to do something I don't want to do.

____ 44. Sometimes I wish that the other kids would pay more attention to what I say.

____ 45. I always do the right things.

____ 46. Sometimes I don't like to obey my parents.

____ 47. Sometimes I don't like it when another person asks me to do things for him.

____ 48. Sometimes I get mad when people don't do what I want.

Appendix I

Debriefing Handout for Participants

The purpose of this study is to look at changes that occur in moods during the teenage years. We are seeking to build on previous research results that will reveal a more comprehensive picture. We are particularly interested in what factors contribute to an increase in low moods of adolescents during puberty.

The study involved completing a questionnaire package and providing measurements of hand features. Although some questions were of a personal nature, your responses will never be used to identify you. If you are concerned about any of the responses you provided, or with other issues that may have arisen as a result of participating in this study, please feel free to contact Dr. Dwight Mazmanian or Jennifer Welsh at the Department of Psychology, Lakehead University, 955 Oliver Rd., Thunder Bay, Ontario, P7B 5E1, (807) 343-8257, dmazmani@lakeheadu.ca, for referral information. You may also directly contact any of the following people/organizations:

Children's Centre Thunder Bay:	343-5000
Psychologists, Psychiatrists, or other Counsellors:	Yellow pages of phone directory
Guidance Counsellor at your school	
Thunder Bay Regional Health Sciences Centre:	684-6000

Thank you so much for your help!