Differential Effects of Individual Factors on the Developmental Pathways of Depression

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Abstract

This study used growth mixture modeling to investigate the developmental pathways of depressive symptoms across adolescence and emerging adulthood (ages 12-25 years) using a nationally representative sample (*N* = 20,394). Four unique non-linear trajectories were found: low-decreasing (normative), low-increasing, low-high increasing, and high-decreasing. In general, being male (i.e., low-decreasing, high-decreasing) and having high self-esteem (i.e., low-decreasing, low-increasing, high-decreasing) were protective against depressive symptoms. None of the included factors had any effect on the dramatic increase of depressive symptoms within the low-high increasing trajectory. The results suggest that, by taking a person-centered approach that included two developmentally sensitive periods, four distinct trajectories of depressive symptoms, and a unique pattern of risk and protective factors within these trajectories were found. Study findings can inform individualized prevention and intervention initiatives by targeting protective factors that are most likely to have a positive impact on the reported developmental pattern of depressive symptoms.

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Adolescence is an important period of development with many life-long processes being established, particularly key aspects of emotional functioning such as emotion regulation (Rawana et al., 2010; Schulenberg, Sameroff, & Cicchetti, 2004). Emotion regulation has been defined as strategies by which emotions are managed in order to meet an individual's goal in a socially acceptable manner (Gross & Thompson, 2007). As such, these strategies include initiating, maintaining, and modifying the occurrence, intensity, or duration of feelings (Gross & Thompson, 2007). One example of an emotion regulation strategy is cognitive reappraisal, defined as thinking about a situation in a way to alter its emotional influence (Gullone et al., 2010). For instance, Sarah, who is happy in her current romantic relationship, is confronted by her romantic partner, who expresses his wish to end the relationship. Utilizing a cognitive reappraisal strategy, Sarah could use the opportunity to express concern and compassion for her partner and discuss reasons for her partner's wish to end the relationship, as opposed to being emotionally overwhelmed and distressed. This strategy may then encourage more dialogue, and they may engage in mutually-satisfying next steps. In general, emotion regulation is particularly important because it is implicated in many psychological disorders (Kring & Sloan, 2009). Vine and Aldao (2014) have found that emotion regulation deficits were the key underlying factors in symptoms of depression and other psychopathologies. Relatedly, emotion regulation is becoming a target for treatment transdiagnostically (Kring & Sloan, 2009; Neacsiu, Eberle, Kramer, Wiesmann, & Linehan, 2014).

Depressive symptoms increase among both boys and girls during mid-adolescence, although girls show higher levels of depressive symptoms throughout adolescence (Van Oort, Greaves-Lord, Verhulst, Ormel, & Huizink, 2009). In addition, adolescent depressive symptoms

often recur in adulthood (Kessler, 2011), and are commonly associated with difficulties in emotion regulation (Betts Gullone, & Allen, 2009). For example, a meta-analysis found that maladaptive or ineffective emotion regulation strategy use (i.e., emotion dysregulation) was associated with depressive symptoms (Aldao, Nolen-Hoeksema & Schweizer, 2010). The effect was generally stronger among adults than youth, indicating a possible developmental trajectory, or time-dependent pathway for the development of depressive symptoms. Further, data from the limited number of longitudinal studies indicate that maladaptive emotion regulation strategy used in childhood and adolescence tends to predict the experience of depressive symptoms later on. To this end, an experience-sampling-method study found that among adults effective emotion regulation strategy use predicts mood improvement, while ineffective emotion regulation strategy use predicts depressive symptoms (Heiy & Cheavens, 2014). Accordingly, the current study will take a developmental approach to the investigation of emotion regulation, as measured by depressive symptoms, throughout adolescence and emerging adulthood. Potentially influential variables from within the individual will be examined, as these factors are important for development within these periods of life (Arnett, 2012; Grusinger & Blatt, 1994).

Emotion Regulation

Emotion regulation has been described by Gross and Thompson (2007) as the effortful or automatic employment of strategies, such as cognitive reappraisal or expressive suppression, which results in a general increase or decrease of either positive or negative emotion, depending on the goals of the individual at the time. Gross and Thompson (2007) also note that multiple emotion regulation strategies may be invoked simultaneously and exert influence on each other. Likewise, emotion dysregulation is a result of strategy use that is inappropriate or ineffective in modifying emotional experience to the current goals of the individual. Emotion dysregulation

may lead to psychopathology, and in particular, depression (Aldao, Nolen-Hoeksema & Schweizer, 2010; Betts Gullone, & Allen, 2009; Heiy & Cheavens, 2014; Neacsiu et al., 2014). To this end, depression is associated with high levels of negative affect, low levels of positive affect, and altered emotional processing (e.g., Watson, Clark, & Carey, 1988). Altered emotion regulation strategy use has even been identified as a trait-like marker for depression among adults (Kanske, Heissler, Schönfelder, & Wessa, 2012). For these reasons, depressive symptoms are understood as a key indicator of underlying emotion dysregulation.

Development of emotion regulation can be specifically conceptualized by the transactional developmental ice-cream-cone-in-a-can model (Sameroff, 2010; Sameroff & Fiese, 2000). In this model, emotions are mainly regulated by others initially, but are increasingly self-regulated as time goes on and development progresses. Other-regulation is largely accomplished by the main caregivers, but can be anyone or anything from an individual's environment or context. In relation to Bronfenbrenner's ecological systems theory (1979), other-regulation may come from any of the systems described. Importantly, self-regulation account for the majority of emotion regulation as an individual ages.

Development within Adolescence and Emerging Adulthood

Two developmentally sensitive periods for emotion regulation are adolescence and emerging adulthood (Collins & Steinberg, 2006; Gross & Thompson, 2007; Rutter, 1996; Schulenberg, Sameroff & Cicchetti, 2004). Also during this time, specifically in mid-adolescence, depressive symptoms generally increase and many adolescents experience depressive symptoms that often recur in adulthood (Kessler, 2011). Depressive symptoms in adolescence are commonly associated with difficulties in emotion regulation (Betts Gullone, & Allen, 2009). Adolescence is broadly defined as occurring between the ages of 10 and 18 years

(Sawyer et al., 2012). There are many transitions and developmental goals in adolescence, including puberty and biological maturation, cognitive development, adaptive emotion regulation, identity forming , increased responsibility, balancing independence and interdependence, and forming romantic relationships (Collins & Steinberg, 2006; Sawyer et al., 2012). Emerging adulthood is a relatively newer period of development identified mainly in industrialized developed countries, and is defined as occurring between the ages of 18 and 25 years or 18 to 29 years (Arnett, 2012). Transitions and developmental goals are similar to that of adolescence and include neurological changes in frontal lobe, limbic and dopamine systems, and associated cognitive development (i.e., perspective taking, adaptive emotion regulation), experimentation with roles, experiencing vocational achievement, living independently, and continuing to develop romantic relationships (Arnett, 2012; Rutter, 1996; Schulenberg, Sameroff, Cicchetti, 2004). The end of required education combined with a general lack of personal commitment and the freedom to experiment with roles sometimes results in uncertainty and an overall feeling of floundering among emerging adults (Schulenberg, Sameroff & Cicchetti, 2004). Successful management throughout both of these developmental periods depends, in part, on the resources and strengths of the individual within their developmental context (Schulenberg, Sameroff & Cicchetti, 2004). The present study focuses on these two developmental periods because of their sensitivity to emotion regulation and depression, as well as their potential continuing impact into adulthood.

Development through adolescence and emerging adulthood can be theoretically conceptualized using Bronfenbrenner's (1979) ecological systems model. In this transactional model, the individual (e.g., genetics, biology, temperament) is at the center and environmental influences are circled around the individual in four increasingly removed and overarching layers,

the microsystem, mesosystem, exosystem, and macrosystem. The microsystem refers to the direct relationships between an individual (including biological factors) and family members, peers, teachers, siblings, religious leaders, and neighbours. The mesosystem refers to relations between institutions within the microsystem, including that between school and church. The exosystem refers to the larger social setting, including mass media and local politics. Finally, the mesosystem refers to cultural influences, specifically, the ideologies, attitudes, and beliefs of the culture. Bronfenbrenner also added the chronosystem to account for sociohistorical variables and changes over time within and between each of the ecological levels (Bronfenbrenner, 1986). Indeed, the development of an individual progresses by building upon itself, or scaffolding (Schulenberg, Sameroff & Cicchetti, 2004). As such, difficulties with emotion regulation, or the experience of depressive symptoms, in adolescence and emerging adulthood may persist into adulthood.

Emotion dysregulation in adolescence can transfer into emerging adulthood, with consequences such as psychopathology, problematic relationships, and low academic success, (Berzin, 2010; Chaiton et al., 2013; Kessler, 2011). These outcomes are important for many reasons. In terms of psychopathology, emotion dysregulation is common to many disorders, and specifically to all mood disorders (Aldao, Nolen-Hoeksema & Schweizer, 2010). Relationships are a primary support throughout the many changes in emerging adulthood and disruptions in relationships could impede the developmental goals of emerging adulthood and beyond (Kessler, 2011). Finally, low academic success could seriously limit future vocational opportunities, in addition to having other consequences (Kessler, 2011). Although difficulties from emerging adulthood are likely to carry into adulthood, this is not always the case and trajectories may change at any time as they are only probabilistic (Schulenberg, Sameroff & Cicchetti, 2004).

There is increasing interest in person-centered statistical methods that integrate a transactional developmental framework, incorporating many different variables over time, allowing for individual differences to be identified. One such method is growth mixture modeling (GMM), which allows for the investigation of both baseline data and changes over time for population subgroups (Field, 2009,Muthén& Muthén, 2000). These and similar techniques have been applied to the investigation of depressive symptoms over time in community samples. Results generally indicate the existence of three or four statistically different trajectories of development with the majority of individuals displaying low depressive symptoms across adolescence or emerging adulthood (e.g., Costello et al., 2008; Frye & Liem, 2011; Mezulis, Salk, Hyde, Priess-Groben, & Simonson, 2014). In addition, three other trajectories have been identified in most or some studies: a low-to-high group (Costello et al., 2008; Dekker et al., 2007; Frye & Liem, 2011; Mezulis et al., 2014), a consistently high group (Frye & Liem, 2011; Sterba et al., 2007), and a high-to-low group (Costello et al., 2008; Frye & Liem, 2011; Mezulis et al., 2014). The study by Costello and colleagues (2008) did incorporate both adolescence and emerging adulthood in the analyses, however, the second half of emerging adulthood (i.e., 22-25) was combined into one time point due to missing data. It is unknown if and how the reported results would change if that time point was expanded.

There are no known studies that have examined developmental pathways of depression symptoms across both adolescence and emerging adulthood (i.e., age 10-25). It is important to include both developmentally sensitive periods in the same analyses in order to better understand and more accurately represent the nature and degree of changes in depressive symptoms that occur during this pivotal time. As reviewed above, current research indicates that symptoms of depression in adolescence may carry over into emerging adulthood, and that symptoms of

depression in emerging adulthood may carry over into adulthood. The elevated risk of depression throughout adolescence and emerging adulthood coupled with the potential continuity of depressive symptoms emphasizes the need to investigate these two developmental periods longitudinally within the same study.

The current study aimed to replicate and expand upon previous research on the developmental pathways of depression, a key indicator of emotion dysregulation, across both adolescence and emerging adulthood using a nationally representative sample. Furthermore, the current study aimed to investigate individual differences by testing whether different trajectories of depressive symptoms existed in the sample, and, if so, whether risk and protective factors differed within each of the trajectories found (Lerner, 2004). The current study will focus on factors from within Bronfenbrenner's (1979) individual system, as these are most salient to the individual.

Developmental Influences: Risk and Protective Factors

The development of emotion regulation across adolescence and emerging adulthood is influenced by both risk and protective factors. As the current study examines the interplay of both risk and protective factors, the theoretical foundations for both are described below, followed by the theoretical foundations for studying the interplay between the two.

Risk factors, or variables that increase the probability of a maladaptive or negative outcome, may also be referred to as vulnerabilities and may be biological, psychological, cognitive, or environmental in nature. The developmental psychopathology framework, certainly the most widely known and applied framework for understanding psychopathology, provides a comprehensive explanation of risk factors (Cicchetti & Cohen, 1995). Cicchetti and Cohen (1995) propose that development scaffolds itself over time, resulting in the effects of early

experience being carried forward. Accordingly, early intervention and prevention are emphasized because previous maladaptive responses place restraints on future adaptation, and the longer a maladaptive trajectory continues, the more difficult it will be to change. Applying this to depression, a risk is something that occurs and increases the chance of emotion dysregulation and therefore depressive symptoms. However, development involves more than the accumulation of risk factors. Protective factors are also salient and influential.

Developmental psychopathology outlines the importance of protective factors, and defines them as variables that increase the probability of a positive or adaptive outcome (Cicchetti & Cohen, 1995). Further emphasizing this concept, positive youth development (PYD) focuses on protective factors and provides a comprehensive framework for understanding them. As described by Lerner, Almerigi, Theokas, and Lerner (2005), PYD takes a strengths-based approach that seeks to identify personal assets that can be developed to promote positive development (Duckworth, Steen, & Seligman, 2005). Under this model, when difficulties are experienced, negative or maladaptive outcomes are only one of many potential outcomes that may alternatively be neutral or even positive. This theory is focused on optimizing individual or ecological resources and promoting mutually beneficial relationships to overcome adversities as opposed to developmental psychopathology's focus on repairing individuals (Cicchetti & Cohen, 1995; Lerner, Almerigi, Theokas & Lerner, 2005). Protective factors include resiliency factors, and may also be referred to as strengths. Strengths have been defined as characteristics and competencies that are valued by both the individual and their society (Rawana & Brownlee, 2009). Specifically, characteristics refer to the fundamental qualities of an individual, whereas competencies refer to purposefully developed skills (Rawana & Brownlee, 2009). In terms of depression, strengths support appropriate emotion regulation development irrespective of the

presence of risks, thereby decreasing the likelihood of experiencing depressive symptoms. However, both developmental psychopathology and positive youth development are insufficient on their own as an individual both experiences and accumulates both strengths and risks throughout development. The relative contributory value and interaction between these influences must be accounted for.

To address this, an integrated approach has been developed by Schwartz, Pantin, Coatsworth, and Szapocznik (2007). This framework generally combines both developmental psychopathology and positive youth development, as the conceptual bases of both theoretical orientations are quite similar. It is important to observe both risks and strengths together in order to capture the interaction between them. Specific strengths may have more or less of a buffering action depending on the specific risks present and the overall context of the individual. Accordingly, this theory integrates the principle of differential susceptibility (Bakermans-Kranenburg & van Ijzendoorn, 2011). Not all instances described as risks will act as risks for every individual and this is likely because their specific strengths prevent or counteract any negative influence of the instance. Research using this approach seeks to identify conditions under which strengths may become more or less effective, or alternatively, conditions under which risk factors truly confer risk (Schwartz et al., 2007). This approach may help individuals adapt best within their current risk factors by promoting the most impactful strengths. The advantage of this theory is that it integrates a more dynamic and ecologically valid model of risks and protective factors and accounts for individual differences (Lerner, 2004). In addition, it acknowledges that there are additional individual and contextual variables that may moderate the impact of a risk. For these reasons the integrated approach is the preferred theoretical framework, over developmental psychopathology and positive youth development, of the proposed study.

Influences on Depressive Symptoms

Some studies have investigated the roles of risk and protective variables on the trajectory of depressive symptoms (e.g., Costello et al., 2008; Frye & Liem, 2011; Mezulis, Salk, Hyde, Priess-Groben, & Simonson, 2014), however, the following variables have yet to be investigated simultaneously. It is important to examine these specific variables simultaneously because, although each have previously been implicated in depressive symptoms, there is the potential for unique interactional effects among them, as the integrated approach outlines. In addition, these interactional effects may differ across trajectories.

Biological Sex

From around adolescence onward, there is a large discrepancy between boys and girls in the experience of depression and depressive symptoms in general, with girls reporting approximately twice that of boys (e.g., Rawana & Morgan, 2013; Bearman & Stice, 2008). To account for this, a 'gender additive model' has been proposed, with gender referring to biological sex (Bearman & Stice, 2008). This model states that some risks are gender-invariant, being experienced the same by both boys and girls, but also that some risks are girl-specific. In this way, girls are exposed to more risk than boys, resulting in the overrepresentation of girls. Along the same line, there may be gender-specific emotion regulation strategies that are less effective for girls, more effective for boys, or some combination. For example, when researching emotion regulation strategies, girls are more likely to engage in rumination and boys are more likely to engage in catastrophizing, while both are equally as likely to engage in self-blame (Rawana, 2013). Taken together, the suggestion is that either rumination is less effective at regulating emotion among girls when compared to the use of catastrophizing among boys, or that self-blame is less effective for girls than for boys, or a combination of the two.

Importantly, empirical evidence for this model is conflicting, implying that this model is either incorrect or requires some changes in order to sufficiently account for the dynamic developmental processes involved. This model states that there is a sex difference but does not account for differential underlying developmental processes between the sexes. In order to be most useful and informative, the model should incorporate mechanisms of action as well. Unfortunately, an alternative has not yet been accepted by the field. Empirical findings will continue to accumulate and likely inform future modification by investigating the role of biological sex in emotion dysregulation as well as the role of biological sex as it interacts with risk and protective factors.

Pubertal Timing

Another biological variable of interest is pubertal timing. Puberty occurs in adolescence and induces substantial changes in an individual's life that may play into the sex differences seen in depressive symptoms. Current research indicates that both early and late pubertal timing, in comparison to one's peers, has been linked to difficulty adjusting, including the experience of depressive symptoms (Susman & Rogol, 2004). Additional experiences vary by biological sex. Specifically, among girls, early puberty is associated with greater internalizing symptoms during adolescence (Black & Klein, 2012; Crockett, Carlo, Wolff, & Hope, 2013), although this is not always found (Gaysina, Richards, Kuh, & Hardy, 2015; Opoliner, Carwile, Blacker, Fitzmaurice, & Austin, 2014). Among boys, early puberty is associated with increased prosocial behavior at home (Carlo, Crockett, Wolf, & Beal, 2012). Late pubertal timing among boys has been linked to substance abuse disorders and disruptive behavior in emerging adulthood (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2014), as well as affective symptoms in adulthood (Gaysina, Richards, Kuh, & Hardy, 2015). In addition, early or late maturation may affect the socialization

of the individual (Carlo et al., 2012), which may have an effect of their self-esteem. Given the social and emotional effects of puberty, and its potential interaction with biological sex, it is worthwhile to further investigate the influence of pubertal timing on depressive symptoms across development.

Self-Esteem

Self-esteem is another important individual factor throughout development, defined as what a person thinks and how a person feels about themselves (Rosenberg, Schooler, & Schoenbach, 1989), which generally acts as a protective factor (Orth, Robins, & Widaman, 2012). There are sex differences within the self-esteem research with findings indicating that girls report lower levels than boys (Puskar et al., 2010), and age effects with research findings showing these sex differences decrease with age (Kling, Shibley Hyde, Showeres, & Boswell, 1999). In addition, early pubertal timing has been linked to lower self-esteem among girls than boys (Williams & Currie, 2000), although the research has also shown null findings (Benoit, Lacourse, & Claes, 2013). Among boys, late pubertal timing is generally associated with lower self-esteem compared to girls (Brack, Orr, & Ingersoll, 1988), although this is less well studied. Thus, it is important to study the relationship between biological sex, pubertal timing, and self-esteem in order to elucidate these relationships.

Current Study

The current study took a person-centered approach to investigate emotion dysregulation, as measured by depressive symptoms, throughout adolescence (i.e., 12-18 years old) and emerging adulthood (i.e., 18-25 years old). The study has three aims. First, the study investigated whether there are different developmental trajectories of depressive symptoms across time. Second, the influence of biological sex, pubertal timing, self-esteem, the interaction between

biological sex and pubertal timing, and the interaction between biological sex and self-esteem on depressive symptoms within each trajectory was investigated. Third, changes in the influence of the risk and protective factors over time within each trajectory was investigated. These aims were addressed using a large national database, which provided longitudinal data for a host of variables across the adolescent and emerging adult periods, making it ideal for an investigation of depressive symptoms across a large span of early development. Specifically, the current study employed Growth Mixture Modeling (GMM), an MLM technique, to model the different trajectories and the risk and protective factors within each trajectory. This is one of the first studies to directly investigate boys and to include participants followed across these two stages that are particularly important for emotion dysregulation. The study also incorporates a developmental and ecological perspective, while integrating developmental psychopathology, positive youth development, and differential susceptibility, in order to further understand the dynamic processes involved.

Hypotheses

There are three general research questions driving the current study: 1) do depressive symptoms develop differently for certain groups across adolescence and emerging adulthood?; 2) what factors, if any, act as risk and protective factors within each identified group?; and 3) do the risk and protective factors maintain their influence over time within each group? Based on the reviewed literature, it is hypothesized that a minimum of three distinct trajectories will be identified in the current study. It is specifically hypothesized that among boys, pubertal timing that is perceived as coinciding with peers, and high self-esteem would protect against depressive symptoms. Similarly, among girls, pubertal timing that is perceived as early or late as compared to peers, and low self-esteem were hypothesized to act as risk factors for depressive symptoms.

The interaction between these variables as well as their specific influence over time is considerably less well studied. Current literature does not point to a clear hypothesis; however, it does indicate the need for the current study.

Methods

Participants

The current study used a sub-sample from the National Longitudinal Study of Adolescent to Adult Health (Add Health; see Harris et al. 2009 for a complete description of the sample and research design). Add Health is a US-based large and comprehensive investigation into adolescent health and development that includes over 20,000 participants chosen from 80 high schools and 52 middle schools with unequal probability of selection (i.e., systematic sampling methods and implicit stratification) to ensure representativeness of US schools with respect to region of country, urbanicity, school size, school type, and ethnicity. The majority of participants entered the study at 12 to 17 years of age, and the study has now completed four waves of data collection spanning 14 years, with an approximately 80% response rate from wave 1 participants at each subsequent wave. Specifically, wave 1 included 20, 745 participants, wave 2 included 14,738 participants, wave 3 included 15,197 participants, and wave 4 included 15,701 participants. Add Health provides data on a number of diverse developmental domains, including biological sex, pubertal timing, and self-esteem, among many others.

Measures

Add Health collects data on a number of diverse developmental domains. The outcome of interest will be self-reported depressive symptoms (CES-D; Radloff, 1977). The current study will also include data on the following domains: biological sex, pubertal timing, self-esteem,

perceived parental closeness, and perceived sibling closeness. All data is self-reported by the youth.

Depression. Depression was measured at each of the four waves using the nine-item short form of the Center for Epidemiologic Studies Depression Scale (CES-D; Needham, 2007). Each item is rated on a four-point likert-type scale that measures the frequency of experiencing each item, ranging from 0 (*never or rarely*) to 3 (*most of the time or all of the time*). Clinical indicator cutoff scores for this shortened measure are 11 or higher for females and 10 or higher for males (Primack, Swanier, Georgiopoulos, Land, & Fine, 2009). Previously reported reliability alphas across all waves range from 0.80 to 0.83 (Brummett, Babyak, Williams & Harris, 2014; Needham, 2007), indicating good internal consistency.

Biological sex. Biological sex was determined by asking the participant what their biological sex is in wave one of data collection. The two options were male or female. In the current sample, 49.3% were male.

Pubertal timing. Pubertal timing was measured at wave one with one item, "How advanced is your physical development compared to other (girls/boys) your age?" that is rated on a 5-point likert-type scale, from 1 (*I look younger than most*) to 5 (*I look older than most*; Cavanagh, Riegle-Crumb, & Crosnoe, 2007; Schreck, Burek, Stewart, & Miller, 2007). This item has been used previously to determine pubertal timing relative to same-age peers within the Add Health database with the average rating being 3.27 (Schreck et al., 2007). This finding suggests that it is a valid measure of relative pubertal timing as, in line with expectations, most participants considered themselves on par with their peers. Similarly, the mean pubertal timing score in the current sample was 3.19.

Self-esteem. Self-esteem was measured at the first three waves using four items. This measure was not present in the fourth wave of data collection. Each item was rated on a 5-point likert-type scale that measures the degree of agreement with each item, ranging from 1 (*strongly disagree*) to 5 (*strongly agree*; Costello, 2008; Rawana, 2013). This measure has good internal consistency, as indicated by previously reported reliability alphas of 0.86 (Galliher, Rostosky, & Hughes, 2004; Rawana, 2013) and 0.85 (Costello, 2008). The group mean self-esteem score in the current sample was 4.15, and scale reliability was $\alpha = .83$.

Data Analysis

The Add Health database was transformed from wave-based to age-based to better represent developmental change (e.g., Costello et al., 2008) in SPSS v.22. The identified domains (i.e., biological sex, pubertal timing, and self-esteem) were investigated as predictors of depressive symptoms for participants aged 12-25 with data present for biological sex, pubertal timing, and at least one data point for depressive symptoms and self-esteem. A total of 20,394 participants met this criteria and were included in all analyses. However, the transformation into age-based data resulted in significant missing data. Ages were combined into two-year spans (i.e., age 12-13, 14-15, 16-17, 18-19, 20-21, 22-23, 24-25) in order to reduce missing data (Costello et al., 2008). Descriptive and correlational analyses were completed using SPSS v.22.

An unconditional growth model was constructed to determine the shape of the overall trajectory to be used in subsequent analyses. Growth mixture modeling (GMM) techniques were then used in order to both represent linear and non-linear change at baseline and across time (Bauer, 2003,Muthén & Muthén, 2000). Specifically, an unconditional growth mixture model (GMM) addressed the first aim of the proposed study by producing group-based trajectories of depressive symptoms over time. The second aim was addressed using a conditional GMM that

incorporated biological sex, pubertal timing, self-esteem, and the interaction of biological sex by each predictor (i.e., pubertal timing, self-esteem) within each trajectory. The third aim was addressed by investigating the influence of the above-mentioned risk and protective factors on the growth factors of each trajectory as identified by the conditional GMM. This exposed any differential risk and protective factors associated with group membership to each trajectory. All growth and growth mixture model analyses were undertaken using Mplus 7.0 statistical software (Muthén & Muthén, 1998-2012). Robust (full information) maximum likelihood (RML) estimation for missing data was used in order to account for any multivariate non-normality that may have been present in the data.

Results

Relationships Among Predictors

A correlation matrix was run in order to assess the level of multicollinearity among the predictors across all participants. Correlations ranged from $r = .002$, $p > .05$ (i.e., between pubertal timing and self-esteem) to $r = .140$, $p > .01$ (i.e., between gender and self-esteem), suggesting that multicollinearity was not an issue as all correlations were smaller than .70 (Tabachnick & Fiddell, 2001).

Unconditional Growth Model of Depressive Symptoms

Plotting the mean depression score over time revealed a trajectory of increasing depressive symptoms after age 12-13 that peaked at age 16-17, then decreased until age 20-21 when scores remained similar until age 24-25 (see Figure 1). This pattern suggested that a quadratic (i.e., age^2) or cubic (i.e., age^3) growth function may fit the data better than a linear (i.e., age) one. Testing these three models revealed the grand mean depression score across all waves

of data collection to be 5.32, suggesting that, on average, participants reported low rates of depressive symptoms. The cubic growth model could not be applied due to nonconvergence after multiple adjustments to improve model fit, and it was deemed inappropriate for the data. Deviance statistics for the linear (D = 245081) and quadratic (D = 244738) functions revealed that quadratic growth fit the data best (ΔD = 343, df = 1, $p < .001$). Therefore, the quadratic growth function was retained in subsequent analyses and is presented in Figure 2. The unconditional quadratic growth model's estimated linear slope was 0.264 (SE = 0.040, $p < .001$), indicating that depression scores were increasing after age 12-13, and the estimate quadratic slope was -0.088 (SE = 0.006, $p < .001$), indicating that this increase slowed over time and depression symptoms began to decrease after age 16-17 until age 24-25. However, the intercept, linear slope, and quadratic slope all have significant standard deviations at 3.31, 1.00, and 0.18, respectively. This suggests that a growth mixture model is appropriate (Jung & Wickrama, 2008).

Unconditional Growth Mixture Model of Depressive Symptoms

Previous research indicates that three or four classes might be most appropriate. An unconditional growth mixture model with two-, three-, four-, and five-classes were fit to the data in order to determine the number of trajectories that best represented the data (Jung & Wickrama, 2008; Muthén & Muthén, 2000). Growth factors (i.e., the intercept, linear slope, and quadratic slope) were unconstrained between classes, such that each class freely estimated these parameters independently. Deviance statistics are not an appropriate tool to compare model fit between different numbers of categorical latent classes, as the likelihood ratio comparisons no longer follow a chi-squared distribution (Tofighi & Enders, 2008), so alternative evaluative measures are reported (Nylund, Asparouhov, & Muthén, 2007; Muthén & Muthén, 2000). Model

fit statistics for two, three, and four class models are presented in Table 1. A five-class model was not identified and therefore did not fit the data. Importantly, the maximum loglikelihood increases with the number of classes, the Bayesian Information Criteria (BIC) decreases, and both the Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMRT) and Lo-Mendell-Rubin adjusted likelihood ratio test (LMRT) are significant ($p < .001$), all of which indicated that a four-class growth mixture model fits the data best (Costello et al., 2008; Muthén & Muthén, 2000). Tests of means difference across trajectories were all significant ($p < .001$). Therefore, the four-class quadratic growth mixture model was retained for subsequent analyses.

Figure 3 presents the four trajectories, referred to as low-decreasing, moderate-decreasing, low-high increasing, and high-decreasing. However, not all posterior probabilities for class membership are above .700 (Costello et al., 2008). The probability of being correctly classified into one of the four trajectories was below this cutoff for two trajectories. Literature suggests that model fit may be improved with the addition of covariates, and that was the next step (Muthén & Muthén, 2000).

Conditional Growth Mixture Model of Depressive Symptoms

In order to improve model fit, a conditional growth mixture model was constructed (Curran & Hussong, 2003). Biological sex, perceived pubertal timing, self-esteem, the interaction between biological sex and perceived pubertal timing, and the interaction between biological sex and self-esteem were added into the four-class quadratic growth mixture model as time-invariant covariates. Each of the covariate's interaction with time (i.e., age or linear slope and age^2 or quadratic slope) was also evaluated. As can be seen in Table 1, model fit indices and estimated posterior class membership probabilities improved (i.e., .681 to .824). Figure 4 illustrates this final model with four trajectories. Importantly, the moderate-decreasing trajectory

changed shape under these conditions, becoming a low-increasing trajectory. Regressions were unconstrained between classes such that each of the four classes independently estimated the influence of each of the predictors on its intercept, linear slope, and quadratic slope. This allowed any potential class-specific differentiation to be detected. Accordingly, the results are presented by class below as well as in Table 2.

Low-decreasing (normative) trajectory. Participants within this trajectory reported an initial mean depression score of 5.1 at age 12-13 that decreased over time to approximately 2.6 at age 24-25. R-square results indicated that the covariates accounted for 19.5% (age 20-21, $p <$ .001) to 78.2% (age 24-25, $p = .036$) of the variance in depression scores. The covariates significantly accounted for the intercept only (36%, $p < .001$). Biological sex was significant on the intercept (-0.676, $p < .001$), linear slope (0.268, $p = .003$), and quadratic slope (-0.035, $p =$ .006). Self-esteem was significant on the intercept (-3.504, $p < .001$) and linear slope (0.399, $p =$ .011). The interaction between biological sex and self-esteem was also significant on the intercept (0.831, $p = .008$) and linear slope (-0.420, $p = .032$). Perceived pubertal timing and the interaction between biological sex and perceived pubertal timing were not significant predictors within this class. These results indicate that initially, at age 12-13, being male and higher self-esteem are associated with lower depression scores, but being male with higher self-esteem is associated with greater depression scores. Over time the pattern is similar, as being male and having higher self-esteem are both associated with an accelerated decrease in depression scores, while being male with higher self-esteem is associated with a slower decrease. However, the protective effect of being male appears to somewhat decrease.

Low-increasing trajectory. Participants within this trajectory reported an initial mean depression score of 5.8 at age 12-13 that increased over time to approximately 7.6 at age 24-25.

R-square results indicated that the covariates accounted for 50.2% (age 16-17, $p < .001$) to 84.2% (age 24-25, $p = .002$) of the variance in depression scores. The covariates explained a significant ($p < .01$) amount of variance in all three growth factors, specifically, 38.8% of the intercept, 33.1% of the linear slope, and 35.1% of the quadratic slope. Self-esteem had a significant effect on intercept (-3.285, $p < .001$), linear slope (-1.027, $p = 0.410$), and quadratic slope (.021, $p = .007$). The interaction between biological sex and self-esteem was significant only on the intercept (-0.912, $p = 0.049$). Biological sex, perceived puberty status, and the interaction between biological sex and perceived pubertal timing were all non-significant predictors within this class. These results indicate that, initially, at age 12-13, having higher self-esteem and being male with higher self-esteem was associated with lower rates of depression symptoms. Over time, self-esteem predicts a slower increase in depression symptoms.

Low-high increasing trajectory. Participants within this trajectory reported an initial mean depression score of 4.1 at age 12-13 that increased over time to approximately 15.8 at age 24-25, indicating that, over time, these individuals began experiencing clinically significant levels of depressive symptoms (Primack et al., 2008). R-square results indicated that the covariates accounted for 28.4% (age 18-19, $p < .001$) to 86.2% (age 24-25, $p < .001$) of the variance in depression scores. The variance explained by the covariates within each of the growth factors were all non-significant. The only significant effect found in this trajectory was that of biological sex on the intercept (-2.112, $p = .019$). This indicates that initially, at age 12-13, being male was associated lower rates of depression symptoms. However, the protective effect of being male did not hold over time.

High-decreasing trajectory. Participants within this trajectory reported an initial mean depression score of 13.9 at age 12-13, indicating clinically significant depressive symptoms

(Primack et al., 2008), that decreased over time to approximately 2.9 at age 24-25. R-square results indicated that the covariates accounted for 30.2% (age 20-21, $p < .001$) to 83.4% (age 12-13, $p < .001$) of the variance in depression scores. The covariates explained a significant ($p < .001$) amount of variance in all three growth factors, specifically, 75.8% of the intercept, 72.7% of the linear slope, and 56.2% of the quadratic slope. Biological sex had a large significant effect on intercept (-5.920, $p < .001$), linear slope (3.037, $p = .004$), and quadratic slope (-0.321, $p = .021$). Self-esteem had a significant effect on intercept (-3.750, $p < .0001$) and quadratic slope (0.119, $p < .001$). Perceived puberty status, the interaction between biological sex and perceived pubertal timing, and the interaction between biological sex and self-esteem were all non-significant predictors within this class. These results indicate that initially, at age 12-13, being male and having high self-esteem was associated with lower rates of depression symptoms. Over time, being male and having high self-esteem predicts a faster decline in depression scores, although the effect from being male decreases somewhat.

Discussion

Study Objectives

The overall objective of the present study was to investigate the developmental trajectories of emotion dysregulation, as measured by depressive symptoms, throughout adolescence (i.e., 12-18 years old) and emerging adulthood (i.e., 18-25 years old) using a nationally representative community sample. First, this study investigated whether there are different developmental trajectories of depressive symptoms across time. Second, this study investigated whether biological sex, pubertal timing, self-esteem, the interaction between biological sex and pubertal timing, and the interaction between biological sex and self-esteem acted as risk or protective factors within each of the identified trajectories. Third, the current

study examined the effects of these risk and protective factors over time within each trajectory. It was hypothesized that three to four different trajectories of depressive symptoms across early development would be found, and that being male, average pubertal timing, and high self-esteem would be protective against depressive symptoms in all trajectories, while being a girl, pubertal timing that is not aligned with peers (i.e., early or late), and low self-esteem would confer risk. The interaction effects between biological sex and either pubertal timing or self-esteem, as well as the change in the influence of identified risk and protective factors, were not specifically hypothesized; however, research indicates that a relationship may exist and so they were included.

Similar studies have typically focused on only one developmental period, either adolescence or emerging adulthood. One notable exception is a study by Costello and colleagues (2008) that did include both adolescence and emerging adulthood. This study is most comparable to the current study in terms of sample and statistical analyses, although there remain substantial differences. In their study, only three waves of Add Health data collection could be included and emerging adulthood was largely represented by two time points due to missing data. In contrast, the current study includes an additional wave of data collection and expands emerging adulthood into three separate time points. The increased number of data points within each time point and the expanded number of time points provided an opportunity to more accurately model the development of depressive symptoms. By including both developmentally sensitive periods, the current study bridges and expands upon prior research to provide a more comprehensive view of how depressive symptoms develop and change over time using a novel and particularly suitable statistical approach.

Summary of Results

Overall, rates of depressive symptoms across adolescence and emerging adulthood were found to be low. Over time, depressive symptoms present a non-linear (i.e., quadratic) developmental pathway with depression scores increasing after age 12-13 until age 16-17, after which they begin decreasing until age 24-25. This trend is in line with previous literature describing adolescence as a particularly vulnerable period for depressive symptoms (e.g., Kessler, 2011). However, depression scores were highly varied at every time point, which suggested that a single trajectory may not be accurately representing the developmental pathway of depression. With this in mind, a person-centered statistical approach (i.e., GMM) was adopted in order to identify unique groups (i.e., classes or trajectories) of individuals whose depression scores change across time similarly within their group but differently than any other group identified (Muthén & Muthén, 2000). Essentially, this approach facilitated a more accurate representation of the developmental pathways of depression.

The first hypothesis of identifying three or four unique trajectories was supported. Within the current sample, four distinct patterns of depressive symptoms were identified, each characterized by non-linear change over time. This compliments previous research finding four trajectories of depressive symptoms across adolescence or emerging adulthood (e.g., Costello et al., 2008; Frye & Liem, 2011; Mezulis et al., 2014). These findings expand on previous research by indicating that four trajectories are representative of the developmental pathways when both developmentally sensitive periods are included in analyses. Based on their shape, these four trajectories are referred to as low-decreasing (normative), low-increasing, low-high increasing, and high-decreasing (Figure 3). These results confirm the findings of Costello and colleagues (2008), who found similar trajectories using Add Health data from the first three waves of data collection. The current study replicates and extends these results to include an additional wave of

data collection and expanding the developmental period of emerging adulthood by continuing the 2-year intervals up to age 25 as opposed to a 4-year interval for the second half of emerging adulthood (i.e., age 22-25). In addition, the current study used a different statistical procedure.

The second hypothesis was generally supported although there were both general and trajectory-specific exceptions. All of the risk and protective factors included in the current study were important, however, only some directly affected when and how depressive symptoms changed (i.e., risk and protective factors; see Figure 4 for trajectories incorporating these factors). Pubertal timing was neither a risk nor a protective factor within any of the four trajectories. This finding is surprising given the large body of research linking early or late pubertal timing to depressive symptoms (e.g., Susman & Rogol, 2004). Biological sex, self-esteem, the interaction between biological sex and pubertal timing, and the interaction between biological sex and self-esteem, as well as the interaction between each of these with time, were found to be important factors. The relationships between these factors and depression scores are different within each of the four trajectories, and these relationships change over time.

The dynamic nature of the risk and protective factors speaks to the third research question. Most of the included risk and protective factors were significant throughout adolescence but did not impact change in depressive symptoms throughout emerging adulthood. This is not surprising as depressive symptoms in adolescence have been very well studied, whereas depressive symptoms in emerging adulthood have not, likely due to the relatively novel inclusion of this as a unique developmental period in research (Arnett, 2012).

Individuals belonging to different trajectories were differentially susceptible to the included risk and protective factors. This is a novel contribution to the literature, as the majority of similar studies focus on identifying factors that influence membership into one trajectory over

another (e.g., Costello et al., 2008). The focus instead on identifying risk and protective factors within each trajectory can go beyond identification and aid in preventative and intervention initiatives that are more appropriate for an individual given their clinical presentation. Accordingly, the results are discussed below by trajectory.

Depressive Symptom Trajectories

Low-decreasing (normative) trajectory. The majority of participants, 68%, were classified as belonging to this trajectory. Participants within this trajectory reported an initial mean depression score of 5.1 at age 12-13 that decreased over time, with a slight acceleration, to approximately 2.6 at age 24-25. These youth experienced a low rate of depressive symptoms. Almost no individual within this trajectory reported experiencing clinically significant depressive symptoms at any time. The findings below must be interpreted with this in mind, as risk factors do not imply a risk for experiencing a clinically significant amount of depressive symptoms. Initially, at age 12-13, being male and having higher self-esteem are protective against depressive symptoms, but being male with higher self-esteem is a risk factor for higher depression scores. Throughout adolescence and emerging adulthood the pattern is similar, as being male and having higher self-esteem both continue to be protective, while being male with higher self-esteem is a risk factor, in that depression scores decrease less rapidly as they enter emerging adulthood. In addition, the protective effect of being male appears to somewhat decrease over time, into emerging adulthood.

Current research aligns with the finding that being male (e.g., Bearman & Stice, 2008; Rawana & Morgan, 2013) and having high self-esteem (Orth et al., 2012) are protective against depressive symptoms. The finding that being male with high self-esteem is a risk factor is novel and unexpected given the protective influence of each factor on their own, as described above.

Additionally, the lack of effect of pubertal timing, including its interaction with biological sex, adds to the mixed results of current research, which states the early pubertal timing may be more detrimental to females and late pubertal timing may be more detrimental to males. (Benoit et al., 2013; Brack et al., 1988; Crockett et al., 2013; Gaysina et al., 2015; Susman & Rogol, 2004; Williams & Curie, 2000). However, at least one study suggests that the consequences of non-aligned pubertal timing may be expressed by externalizing rather than internalizing symptoms among males (Graber et al., 2014).

Low-increasing trajectory. Participants within this trajectory (20%) reported an initial mean depression score of 5.8 at age 12-13 that increased over time, at a slightly decelerated rate, to approximately 7.6 at age 24-25. These youth experienced low to moderate rates of depressive symptoms. Few individuals within this trajectory reported experiencing clinically significant depressive symptoms at any time. The findings below must be interpreted with this in mind, as risk factors do not necessarily imply a risk for experiencing clinically significant depressive symptoms. Initially, at age 12-13, having higher self-esteem and being male with higher self-esteem are protective factors. Moving through adolescence and emerging adulthood, only self-esteem is protective against increasing depression symptoms.

Current literature is again in line with the finding that being male (e.g., Bearman & Stice, 2008; Rawana & Morgan, 2013) and having high self-esteem (Orth et al., 2012) are protective against depressive symptoms. Similar to the Low-decreasing trajectory, the lack of effect of pubertal timing, including its interaction with biological sex, is not aligned with current research stating that early or late pubertal timing is a risk factor for depressive symptoms (Susman & Rogol, 2004).

This trajectory was the least stable, with the majority of misclassified youth instead belonging to the low-decreasing (normative) trajectory. Looking at Figure 4, it is clear that these two trajectories are most similar. The risk and protective factors within this trajectory are also similar to the pattern found for the low-decreasing (normative) trajectory. Taken together, the misclassification may be expected. However, they remained unique and differentiable trajectories in light of such similarities. It will be important to further investigate this trajectory, as its shape indicates that depressive symptoms may continue to increase into adulthood. Indeed, this would be in line with research indicating that depressive symptoms may carry over from adolescence into emerging adulthood and into adulthood (Kessler, 2011).

Low-high increasing trajectory. Participants within this trajectory (5%) reported an initial mean depression score of 4.1 at age 12-13 that increased over time, although more slowly throughout emerging adulthood, to approximately 15.8 at age 24-25. Although these individuals experienced low to moderate levels of depressive symptoms throughout adolescence, depressive symptoms became clinically significant in emerging adulthood (Primack et al., 2008). Initially, at age 12-13, being male was protective against depressive symptoms. However, this protective effect did not continue throughout adolescence or emerging adulthood.

Current literature is again in line with the finding that being male (e.g., Bearman & Stice, 2008; Rawana & Morgan, 2013) is protective against depressive symptoms. The lack of effect from all other factors, and the lack of effect of any factor as these youth develop is unexpected. Perhaps the lack of findings is a consequence of the majority of studies focusing on either a mainly normative sample or a diagnostic sample that includes either adolescents (e.g., Mezulis et al., 2014), or adults, as emerging adulthood has only recently become an accepted developmental period (Arnett, 2012). This group of youth may not have been previously identified. Nonetheless,

this trajectory requires further study as these youth are experiencing an alarmingly quick increase in depression symptoms that are in the clinical range by the time they reach emerging adulthood.

High-decreasing trajectory. Participants within this trajectory (8%) reported an initial mean depression score of 13.9 at age 12-13, indicative of clinically significant depressive symptoms (Primack et al., 2008), that decreased at an accelerated rate over time to approximately 2.9 at age 24-25. Initially, at age 12-13, being male and having high self-esteem are protective against depression symptoms, although it is unlikely that the protective effect results in clinically non-significant symptomatology. Across development, being male and having high self-esteem continue to be protective, predicting a faster decline in depression scores. In emerging adulthood, though, the protective effect from being male decreases somewhat.

Similar to other trajectories, current research aligns with the finding that being male (e.g., Bearman & Stice, 2008; Rawana & Morgan, 2013) and having high self-esteem (Orth et al., 2012) are protective against depressive symptoms. However, the lack of effect of pubertal timing, including its interaction with biological sex, is not aligned with current research stating that early or late pubertal timing is a risk factor for depressive symptoms (Susman & Rogol, 2004). This is particularly unexpected given that these youth experience the most depressive scores around the typical age for puberty (i.e., age 12-13; Sawyer et al., 2006; Susman & Rogol, 2004). As this group of youth start out with such elevated depression symptoms, it would be important to investigate their experiences in childhood.

Clinical Implications

The differences between the four trajectories emphasize the need for person-centered analyses that incorporate individual differences, as well as more individualized treatments. The

inclusion of biological sex, pubertal timing, self-esteem, the interaction between biological sex and pubertal timing, and the interaction between biological sex and self-esteem together were shown to be important contributors to depression scores at every time point. But the specific relationships within each of the four developmental trajectories were different.

The protective effect of being male was strongly supported in the current study. Having a biological sex of male was associated with lower depressive symptoms at age 12-13 within all four trajectories, although for the low-increasing this was through its interaction with pubertal timing rather than a direct effect. This is in line with a vast amount of research indicating a gender difference in depressive symptoms beginning in early adolescence (e.g., Aldao et al., 2008; Kessler, 2011). This protective effect of being male was strongest in the high-decreasing trajectory, where it continued to have a significant effect across time as well. This is in contrast to the other three trajectories, where the protective effect of being male decreases over time. It is important that clinicians keep this in mind. Evidence justifies an increase in resources invested in adolescent females, but there is little evidence to justify the same in emerging adulthood. Although there may be biological sex differences in the rates of depression, being male does not always act as a protective factor, especially in emerging adulthood (e.g., Aldao et al., 2008; Kessler, 2011).

It is worthwhile to note that the three trajectories with low initial status had an intercept that was within two depression score points, meaning that adolescents that have significantly different outcomes in emerging adulthood look very similar at age 12-13.This is similar to the findings reported by Costello and colleagues (2008). One of these trajectories went on to display clinically significant depressive symptoms throughout emerging adulthood (Primack et al., 2008), with biological sex affecting only the initial status at age 12-13, and no factors affecting

the quickly increasing symptomatology. This supports previous research indicating that depressive symptoms in adolescence can result in psychopathology in emerging adulthood (Aldao et al., 2008; Kessler, 2011). It also emphasizes the need to identify variables that differentiate these three groups at age 12-13 in order to correctly identify these youth and implement trajectory-appropriate preventative or intervention strategies (Costello et al., 2008).

Limitations and Future Research

The current study is not without limitations. The Add Health study includes a nationally representative sample and truncated measures that may not be representative of specific populations or factors. However, this limitation was outweighed by advantages of using the Add Health database, including the large sample size, longitudinal design that spanned the developmental periods of interest, and wide variety of variables measured similarly at each time point. Classification accuracy for the low-increasing trajectory was below the suggested .700 cutoff (i.e., .681). Given the similarity in shape to the low-decreasing (normative) trajectory, this is expected; however, it does suggest that the model could be improved. In the current study, model fit was improved with the addition of risk and protective factors, and future research may aim to further improve the model by including additional factors. The factors chosen, while supported by research, are not exhaustive and many additional factors may be included in order to improve understanding of these developmental trajectories. Future research is encouraged to include additional time-invariant and time-varying covariates, or risk and protective factors, in order to more fully understand the relationships involved. Finally, growth mixture modeling is a relatively new statistical approach that brings some controversy (Jung & Wickrama, 2008). Using Mplus software (Muthén & Muthén, 1998-2012); however, the method overcomes much of the criticisms as the software generates multiple distinct indices (e.g., MLL, BIC, VMLT, etc.)

that may be used to describe and evaluate the fit of a model to the data, and the risk of the model describing only a subset or small portion of the available data (i.e, local maxima) is reduced by beginning the statistical analyses at many different points in the data (i.e., utilizing random starting values) and requiring replication of results, or convergence. However, nonconvergence was experienced in the current study and prevented two analyses from continuing. Future studies may aim to overcome this with additional model specification changes.

Conclusion

In total, 92% of adolescents and 95% or emerging adults reported experiencing rates of depressive symptoms that were not clinically significant. A person-centered approach elicited a more individualized understanding of the different patterns in which youth experience depressive symptoms. By including two developmentally sensitive periods for depression, the findings illustrate patterns of change across these ages, and specifically, that individuals experiencing clinically significant depressive symptoms in adolescence (8%) are, on average, not the individuals experiencing clinically significant depressive symptoms in emerging adulthood (5%). Rates are higher in adolescence with even the normative trajectory (68%) experienced higher rates of depressive symptoms in adolescence when compared to emerging adulthood. However, the opposite occurred as well, although to a lesser extent, with 25% of youth experiencing more depressive symptoms in emerging adulthood. This bridges and supports previous research identifying adolescence as a particularly vulnerable period for depression, as well as the potential for depressive symptoms to carry over from adolescence into emerging adulthood (Costello et al., 2008; Kessler, 2011). The risk of clinically significant depression is differentially distributed in adolescence (i.e., 12-18) and emerging adulthood (i.e., 18-25), supporting the differentiation of adolescence and emerging adulthood as two different developmental periods (Arnett, 2012).

The finding of four unique trajectories of depression provides a more detailed understanding of the developmental pathways of depression throughout adolescence and emerging adulthood. The examination of research-supported risk and protective factors separately within each of these four trajectories further improves our understanding of when depression develops, for whom, and the changes experienced over time. Such information can be applied to develop individualize prevention and intervention strategies, thereby increasing the appropriateness and potential effectiveness of targeted issues or strengths.

References

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

Arnett, J. J. (2012). New horizons in emerging and young adulthood. In A. Booth, S. L. Brown, N. S. Landale, W. D. Manning, & S. M. McHale (Eds.), *Early adulthood in a family context* (pp. 231-244). New York: Springer.

Bakerman-Kranenburg, M. & Van Ijzendoorn, M. (2011). Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Developmental Psychology, 23,* 39-52.

Bauer, D. J. (2003). Estimating multilevel linear models as structural equation models. *Journal of Educational and Behavioral Statistics, 28,* 135-167.

Bearman, S. K., & Stice, E. (2008). Testing a gender additive model: The role of body image in adolescent depression. *Journal of Abnormal Child Psychology, 36,* 1251-1263.

Benoit, A., Lacourse, E., & Claes, M. (2013). Pubertal timing and depressive symptoms in late adolescence: The moderating role of individual, peer, and parental factors. *Development and Psychopathology, 25,* 455-471.

Berger, K.S. (2012). *The developing person through childhood* (6th ed.). New York, NY: Worth Publishers.

Berzin, S. C. (2010). Vulnerability in the transition to adulthood: Defining risk based on youth profiles. *Children and Youth Services Review, 32,* 487-495.

Betts, J., Gullone, E., & Allen, J. S. (2009). An examination of emotion regulation, temperament, and parenting style as potential predictors of adolescent depression risk status: A correlational study. *British Journal of Developmental Psychology, 27,* 473-485.

Black, S. R., & Klein, D. N. (2012). Early menarcheal age and risk for later depressive symptomatology: the role of childhood depressive symptoms. *Journal of Youth and Adolescence, 41,* 1142-1150.

Brack, C. J., Orr, D. P., & Ingersoll, G. (1988). Pubertal maturation and adolescent self-esteem. *Journal of Adolescent Health Care, 9*, 280–285.

Branje, S. J., Hale III, W. W., Frijns, T., & Meeus, W. H. (2010). Longitudinal associations between perceived parent-child relationship quality and depressive symptoms in adolescence. *Journal of Abnormal Child Psychology, 38*, 751-763.

Bronfenbrenner, U. (1979). *The ecological of human development: Experiments by nature and design.* Cambridge, MA: Harvard University Press.

Bronfenbrenner, U. (1986). Recent advances in research on the ecology of human development. In R. K. Silbereisen, K. Eyferth, G. Rudinger (Eds.). *Development as action in context:*

Brummett, B. H., Babyak, M. A., Williams, R. B., Harris, K. M., Jiang, R., Kraus, W. E., ... & Siegler, I. C. (2014). A putatively functional polymorphism in the HTR2C gene is associated with depressive symptoms in white females reporting significant life stress. *PloS one*, *9*: e114451.

Carlo, G., Crockett, L. J., Wolff, J. M., & Beal, S. J. (2012). The Role of Emotional Reactivity, Self-regulation, and Puberty in Adolescents' Prosocial Behaviors. *Social Development, 21*, 667-685.

Cavanagh, S.E., Riegle-Crumb, C. & Crosnoe, R. (2007). Puberty and the education of girls. *Social Psychology Quarterly, 70,* 186-198.

Chaiton, M., Contreras, G., Brunet, J., Sabiston, C. M., O'Loughlin, E., Low, N. C. P., Karp, I., Barnett, T. A., & O'Loughlin, J. (2013). Heterogeneity of depressive symptom

trajectories through adolescence: predicting outcomes in young adulthood. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 22*, 96-105.

Cicchetti, D. & Cohen, D. J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti, & D. J. Cohen (Eds.), *Developmental psychopathology: Theory and methods* (pp. 3-20). New York: Wiley.

Collins, W. A., & Steinberg, L. (2006). Adolescent development in interpersonal context. (6th ed.). In N. Eisenberg, W. Damon & R. W. Lerner (Eds.), *Handbook of child psychology* (Vol. 3). (pp. 1003-1067). New York: Wiley.

Costello, D. M., Swendsen, J., Rose, J. S., & Dierker, L. C. (2008). Risk and protective factors associated with trajectories of depressed mood from adolescence to early adulthood. *Journal of consulting and clinical psychology*,*76*, 173.

Crockett, L. J., Carlo, G., Wolff, J. M., & Hope, M. O. (2013). The role of pubertal timing and temperamental vulnerability in adolescents' internalizing symptoms. *Development and psychopathology*, *25*, 377-389.

Crossman, A.F. (2007). The parent-child relationship and substance use: A test of long-term mediating effects of self-esteem using data from the national longitudinal study of adolescent health. *Dissertation Abstracts International Section A: Humanities and Social Sciences*, *68*, 1675.

Duckworth, A. L., Steen, T. A., & Seligman, M. E. P. (2005). Positive psychology in clinical practice. *Annual Review of Clinical Psychology, 1,* 629-651.

Eisenberg, N. (1996). Meta-emotion and the socialization of emotion in the family – A topic whose time has come: Comment on Gotmann et al. (1996). *Journal of Family Psychology, 10*, 269-276.

Field, A. (2009). Discovering statistics using SPSS. (3rd ed.). Thousand Oaks, CA: SAGE Publications Inc.

Gaysina, D., Richards, M., Kuh, D., & Hardy, R. (2015). Pubertal maturation and affective symptoms in adolescence and adulthood: Evidence from a prospective birth cohort. *Development and Psychopathology*, 1-10.

Graber, J. A., Seeley, J. R., Brooks-Gunn, J., & Lewinsohn, P. M. (2004). Is pubertal timing associated with psychopathology in young adulthood?. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*, 718-726.

Gross, J. J. (2013). Emotion regulation: Taking stock and moving forward. *Emotion, 13*, 359-365.

Gross, J.J., & Thompson, R.A. (2007). Emotion regulation: Conceptual foundations. In J.J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3-24). New York: Guilford Press.

Grusinger, S., & Blatt, S. (1994). Individuality and relatedness: Evaluation of a fundamental dialectic. *American Psychologist, 49,* 104-111.

Harris, K.M., C.T. Halpern, E. Whitsel, J. Hussey, J. Tabor, P. Entzel, and J.R. Udry. 2009. The National Longitudinal Study of Adolescent Health: Research Design [WWW document]. URL: http://www.cpc.unc.edu/projects/addhealth/design.

Heiy, J. E., & Cheavens, J. S. (2014). Back to basics: A naturalistic assessment of the experience and regulation of emotion. *Emotion, 14,* 878-897.

Jonassaint, C. R., Ashley-Koch, A., Whitfield, K. E., Hoyle, R. H., Richman, L. S., Siegler, I. C., . . . Williams, R. (2012). The serotonin transporter gene polymorphism (5HTTLPR) moderates the effect of adolescent environmental conditions on self-esteem in young

adulthood: A structural equation modeling approach. *Biological Psychology, 91,* 111-119.

Kanske, P., Heissler, J., Schönfelder, S., & Wessa, M. (2012). Neural correlates of emotion regulation deficits in remitted depression: the influence of regulation strategy, habitual regulation use, and emotional valence. *Neuroimage, 61,* 686-693.

Kessler, R. C. (2012). The costs of depression. *The Psychiatric Clinics of North America, 35,* 1-14.

Kling, K. C., Hyde, J. S., Showers, C. J., & Buswell, B. N. (1999). Gender differences in self-esteem: a meta-analysis. *Psychological Bulletin, 125,* 470-500.

Kramer, L. (2014). Learning emotional understanding and emotion regulation through sibling interaction. *Early Education and Development, 25,* 160-184.

Kring, A. M., & Sloan, D. M. (Eds.). (2009). *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment.* New York: Guilford Press.

Lerner, R. M. (2004). Diversity in individual <- -> context relations as the basis for positive development across the life span: A developmental systems perspective for theory, research, and application. *Research in Human Development, 1,* 327-346.

Lerner, R. M., Almerigi, J.B., Theokas, C., & Lerner, J. V. (2005). Positive Youth Development: A view of the issues. *Journal of Early Adolescence, 25,* 10-16.

Mezulis, A., Salk, R. H., Hyde, J. S., Priess-Groben, H. A., & Simonson, J. L. (2014). Affective, biological, and cognitive predictors of depressive symptom trajectories in adolescence. *Journal of Abnormal Child Psychology, 42,* 539-550.

Muthén, B., & Muthén, L. K. (2000). Integrating person-centered and variable-centered analyses:

Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical and Experimental Research, 24,* 882-891.

Muthén, L.K. & Muthén, B. (2012). Mplus User's Guide. Seventh Edition. Los Angeles, CA: Muthén & Muthén.

Nagin, D. (2009). *Group-based modeling of development*. Cambridge, MA: Harvard University Press.

Neacsiu, A. D., Eberle, J., Kramer, R., Wiesmann, T., & Linehan, M. M. (2014). Dialectical Behavior Therapy Skills for Transdiagnostic Emotion Dysregulation: A Pilot Randomized Controlled Trial. *Behaviour Research and Therapy, 49,* 40-51.

Needham, B.L. (2007). Gender differences in trajectories of depressive symptomatology and substance use during the transition from adolescence to young adulthood. *Social Sciences & Medicine, 65,* 1166-1179.

Needham, B.L. (2008). Reciprocal relationships between symptoms of depression and parental support during the transition from adolescence to young adulthood. *Journal of Youth and Adolescence, 37,* 893-905.

Nylund, K. L., Asparouhov, T., & Muthén, B. (2007). Deciding on the number of classes in latent class analysis and growth mixture modeling: A Monte Carlo simulation study. *Structural Equation Modeling: A Multidisciplinary Journal,* 14, 535–569.

Oliva, A., & Arranz, E. (2005). Sibling relationships during adolescence. *European Journal of Developmental Psychology, 2,* 253–270.

Opoliner, A., Carwile, J. L., Blacker, D., Fitzmaurice, G. M., & Austin, S. B. (2014). Early and late menarche and risk of depressive symptoms in young adulthood. *Archives of Women's Mental Health, 17,* 511-518.

Orth, U., Robins, R. W., & Widaman, K. F. (2012). Life-span development of self-esteem and its effects on important life outcomes. *Journal of Personality and Social Psychology, 102*, 1271.

Primack, B. A., Swanier, B., Georgiopoulos, A. M., Land, S. R., & Fine, M. J. (2009). Association between media use in adolescence and depression in young adulthood: A longitudinal study. *Archives of General Psychiatry, 66,* 181-188.

Puskar, K. R., Bernardo, L. M., Ren, D., Haley, T. M., Tark, K. H., Switala, J., & Siemon, L. (2010). Self-esteem and optimism in rural youth: Gender differences. *Contemporary Nurse, 34,* 190-198.

Radloff, L.S. 1977. "The CES-D Scale: A self-report depression scale for research in the general public. *Applied Psychological Measurement, 1,* 385-401.

Rawana, J. S., & Morgan, A. S. (2013). Trajectories of depressive symptoms from adolescence to young adulthood: the role of self-esteem and body-related predictors. *Journal of Youth and Adolescence*, *43*, 597-611.

Rawana, J. S. (2013). The relative importance of body change strategies, weight perception, perceived social support, and self-esteem on adolescent depressive symptoms: Longitudinal findings from a national sample. *Journal of Psychosomatic Research*, *75*, 49-54.

Rawana, E., & Brownlee, K. (2009). Making the possible probable: A strength-based assessment and intervention framework for clinical work with parents, children and adolescents. *Families in Society: The Journal of Contemporary Social Services*, *90*, 255-260.

Rawana, J. S., Morgan, A. S., Nguyen, H., & Craig, S. G. (2010). The relation between eating-

and weight-related disturbances and depression in adolescence: A review. *Clinical Child and Family Psychology Review, 13,* 213-230.

Rosenberg, M., Schooler, C., & Schoenbach, C. (1989). Self-esteem and adolescent problems: Modeling reciprocal effects. *American Sociological Review, 54,* 1004-1018.

Roettger, M. E., & Boardman, J. D. (2012). Parental incarceration and gender-based risks for increased body mass index: evidence from the National Longitudinal Study of Adolescent Health in the United States. *American Journal of Epidemiology,* 409.

Rozemberg, L., Avanci, J., Schenker, M., & Pires, T. (2014). Resilience, gender and family during adolescence. *Ciência & Saúde Coletiva, 19,* 673-684.

Rutter, M. (1996). Transitions and turning points in developmental psychopathology: As applied to the age span between childhood and mid-adulthood. *International Journal of Behavioral Development, 19,* 603-626.

Sawyer, S. M., Afifi, R. A., Bearinger, L. H., Blakemore, S. J., Dick, B., Ezeh, A. C., & Patton, G. C. (2012). Adolescence: A foundation for future health. *The Lancet, 379,* 1630 1640.

Sameroff, A. J. (1995). General systems theories and developmental psychopathology. In D. Cicchetti, & D. J. Cohen (Eds.), *Developmental psychopathology: Theory and methods* (pp. 659-695). New York: Wiley.

Sameroff, A. (2010). A unified theory of development: A dialectic integration of nature and nurture. *Child Development, 81,* 6-12.

Sameroff, A. J., & Fiese, B. H. (2000). Transactional regulation: The developmental ecology of early intervention. *Handbook of early childhood intervention, 2,* 135-159.

Schnettler, S., & Steinbach, A. (2011). How do biological and social kinship play out within

families in the US? An evolutionary perspective on perceived parental care and closeness in adolescents. *Zeitschrift für Familienforschung-Journal of Family Research, 23,* 173-195.

Schreck, C. J., Burek, M. W., Stewart, E. A., & Miller, J. M. (2007). Distress and violent victimization among young adolescents: Early puberty and the social interactionist explanation. *Journal of Research in Crime and Delinquency, 44,* 381-405.

Schulenberg, J. E., Sameroff, A. J., & Cicchetti, D. (2004). The transition to adulthood as a critical juncture in the course of psychopathology and mental health. *Development and Psychopathology, 16,* 799-806.

Schwartz, S. J., Pantin, H., Coatsworth, J. D., & Szapocznik, J. (2007). Addressing the challenges and opportunities for today's youth: Toward an integrative model and its implications for research and intervention. *The Journal of Primary Prevention, 28,* 117-144.

Scott, M. E., Booth, A., King, V., & Johnson, D. R. (2007). Postdivorce father-adolescent closeness. *Journal of Marriage and Family, 69,* 1194-1209.

Slomkowski, C., & Manke, B. (2004). Sibling relationships during childhood: Multiple perceptions from multiple perspectives. In R.D. Conger, F.O. Lorenz, & K.A.S. Wickrama (Eds.), Continuity and change in family relationships: Theory, methods, and empirical findings. (pp. 293-318). New Jersey: Lawrence Erlbaum Associates, Inc.

Smojver-Ažić, S., & Bezinović, P. (2011). Sex differences in patterns of relations between family interactions and depressive symptoms in adolescents. *Croatian medical journal,* 52, 469-477.

Stojković, I. (2013). Pubertal timing and self-esteem in adolescents: The mediating role of body

image and social relations. *European Journal of Developmental Psychology, 10,* 359-377.

Tofighi, D. & Enders, C. K. (2008). Identifying the correct number of classes in growth mixture models. *Advances in latent variable mixture models*, (Information Age Publishing, Inc), 317-341.

Van Voorhees, B. W., Paunesku, D., Kuwabara, S. A., Basu, A., Gollan, J., Hankin, B. L., Melkonian, S., & Reinecke, M. (2008). Protective and vulnerability factors predicting new-onset depressive episode in a representative of US adolescents. *Journal of Adolescent Health, 42*, 605-616.

Vine, V., & Aldao, A. (2014). Impaired Emotional Clarity and Psychopathology: A Transdiagnostic Deficit with Symptom-Specific Pathways through Emotion Regulation. *Journal of Social and Clinical Psychology, 33,* 319-342.

Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology, 97,* 346-353.

Videon, T. M. (2002). The effects of parent-adolescent relationships and parental separation on adolescent well-being. *Journal of Marriage and Family*,*64,* 489-503.

Williams, J. M., & Currie, C. (2000). Self-esteem and physical development in early adolescence: Pubertal timing and body image. *Journal of Early Adolescence, 20*, 129–149.

Appendix A: Tables

Table 1.

Model Fit Indices for Latent Class Growth Mixture Models[a]

Number of Classes	MLL[b]	BIC[c]	SABIC[d]	AIC[e]	VLMRT[f]	LMRT[g]	Posterior Probabilities[h]
2	-121197	242594	242530	242435	$p < .001$	$p < .001$	0.951 0.835
3	-120382	241021	240939	240815	$p < .001$	$p < .001$	0.925 0.795 0.756
4	-119500	239318	239217	239064	$p < .001$	$p < .001$	0.774 0.743 0.692 0.660
4 Conditional	-116041	232955	232675	232257	$p = .023$	$p = .024$	0.824 0.815 0.701 0.681

Note. [a]N = 20,394. [b]MLL = Maximum loglikelihood. [c] = Bayesian Information Criteria. [d] = Sample-size adjusted Bayesian Information Criteria. [e] = Akaike Information Criteria. [f] = Vuong-Lo-Mendell-Rubin likelihood ratio test for k-1 classes. [g] = Lo-Mendell-Rubin adjusted likelihood ratio test for k-1 classes.

Table 2.

Models of Depressive Symptoms Based on Covariates[a]

Model			UGM[b]	Conditional GMM[c]			
			quadratic	Low-decreasing	Low-increasing	Low-high increasing	High-decreasing
Outcome	Intercept	γ_{00}	5.735**** (0.059)	5.124*** (0.163)	5.783*** (0.550)	4.139*** (0.695)	13.942*** (1.012)
Initial Status, π_{0i} (intercept)	Sex	γ_{01}		-0.676*** (0.144)	-0.850 (0.552)	-2.112* (0.899)	-5.920*** (1.345)
	Pubertal Timing	γ_{02}		-0.041 (0.114)	0.646 (0.428)	-0.020 (0.592)	0.561 (0.611)
	Self-esteem	γ_{03}		-3.504*** (0.255)	-3.285*** (0.916)	-1.659 (1.687)	-3.750*** (0.347)
	Sex x Pubertal Timing	γ_{012}		- 0.065 (0.132)	-0.912* (0.463)	-0.077 (0.814)	-0.193 (0.829)
	Sex x Self-esteem	γ_{013}		0.831** (0.311)	1.041 (0.970)	0.797 (0.923)	-0.168 (1.267)
Rate of Change, π_{1i} (linear slope)	Age	γ_{10}	0.264*** (0.040)	- .120* (0.109)	0.608* (0.305)	2.663*** (0.404)	-0.534** (0.690)
	Sex x Age	γ_{11}		.268** (0.490)	-0.408 (0.318)	-0.248 (0.556)	3.037** (1.047)
	Pubertal Timing x Age	γ_{12}		- .016* (0.069)	-0.325 (0.268)	0.163 (0.407)	-0.510 (0.472)
	Self-esteem x Age	γ_{13}		- .001 (0.023)	-1.027* (0.503)	-0.158 (0.853)	-0.008 (0.000)
	Sex x Pubertal Timing x Age	γ_{112}		0.024 (0.084)	0.417 (0.299)	-0.339 (0.548)	0.189 (0.650)

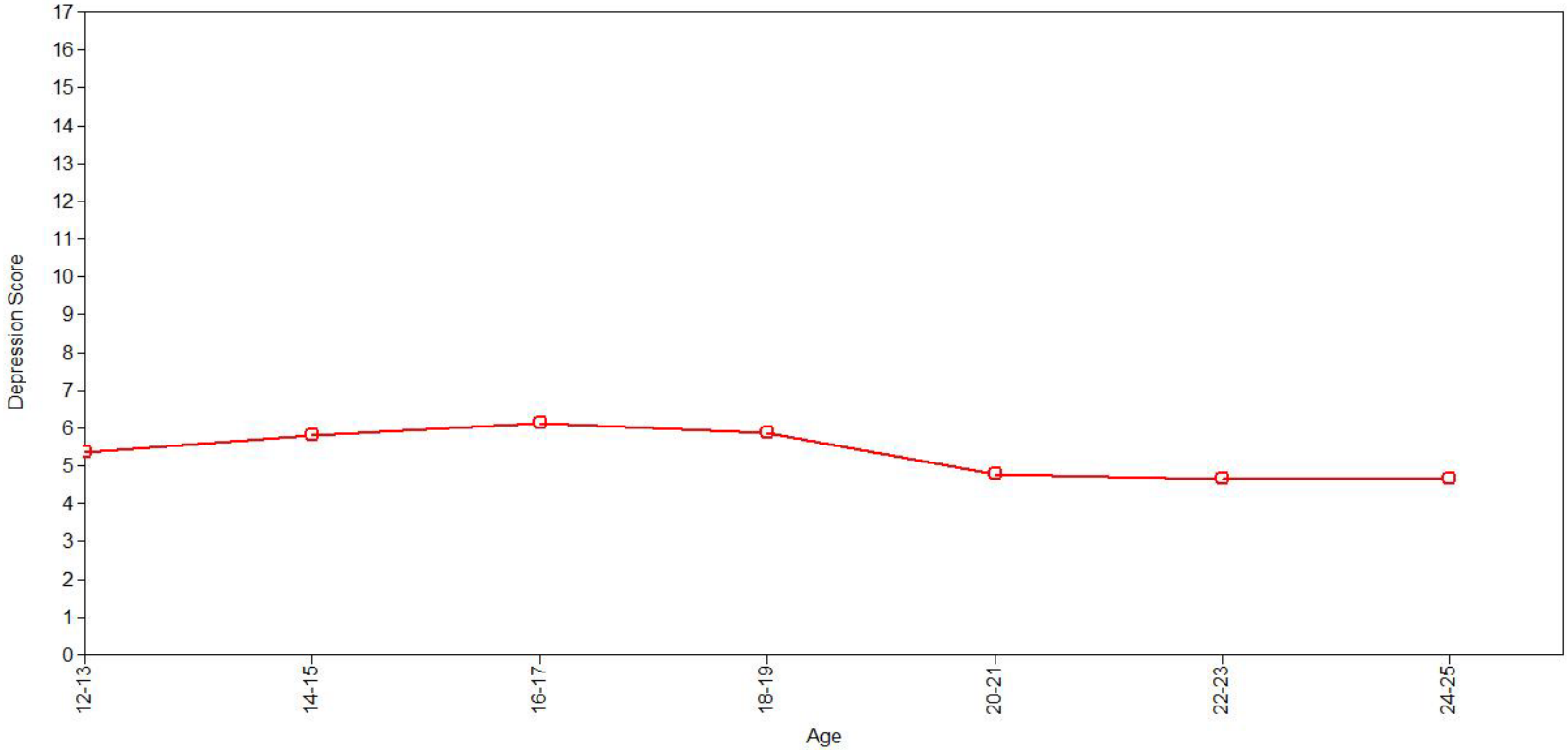
Model			UGM[b]	Conditional GMM[c]			
			quadratic	Low-decreasing	Low-increasing	Low-high increasing	High-decreasing
	Sex x Self-esteem x Age	γ_{113}		- 0.420* (0.196)	-0.043 (0.574)	0.143 (1.142)	-0.482 (1.034)
Rate of Change, π_{2i} (quadratic slope)	Age^2	γ_{20}	-0.088*** (0.006)	-.055** (0.014)	-0.043 (0.038)	-0.117* (0.064)	-0.215 (.030)
	Sex x Age^2	γ_{21}		- 0.035** (0.013)	0.060 (0.043)	0.066 (0.086)	-0.321* (0.139)
	Pubertal Timing x Age^2	γ_{22}		0.002 (0.010)	0.031 (0.036)	-0.023 (0.062)	0.063 (0.064)
	Self-esteem x Age^2	γ_{23}		- 0.001 (0.023)	0.213** (0.078)	0.029 (0.130)	0.119*** (0.016)
	Sex x Pubertal Timing x Age^2	γ_{212}		0.001 (0.012)	-0.052 (0.041)	0.064 (0.082)	-0.029 (0.090)
	Sex x Self-esteem x Age^2	γ_{213}		0.057 (0.029)	-0.046 (0.088)	-0.114 (0.180)	0.065 (0.144)

Note. [a]N = 20,394. [b]UGM = Unconditional Growth Model. [c] = Growth Mixture Model.
*p < .05. **p < .01. ***p < .001.

Appendix B: Figures

Figure 1.

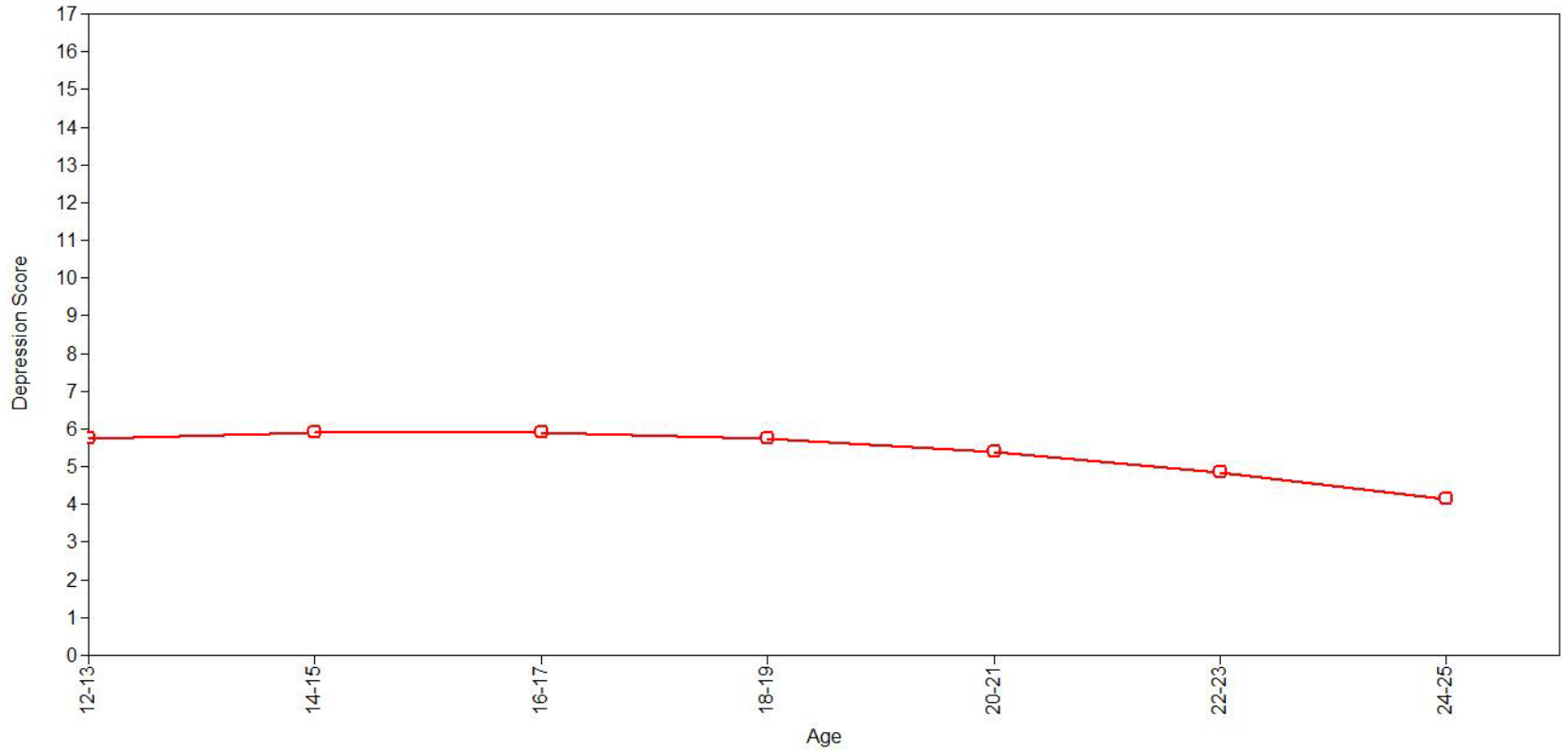
Mean Depression Scores[a] *by Age.*



Note: [a] = Nine-item CES-D (Center for Epidemiologic Studies Depression Scale) score.

Figure 2.

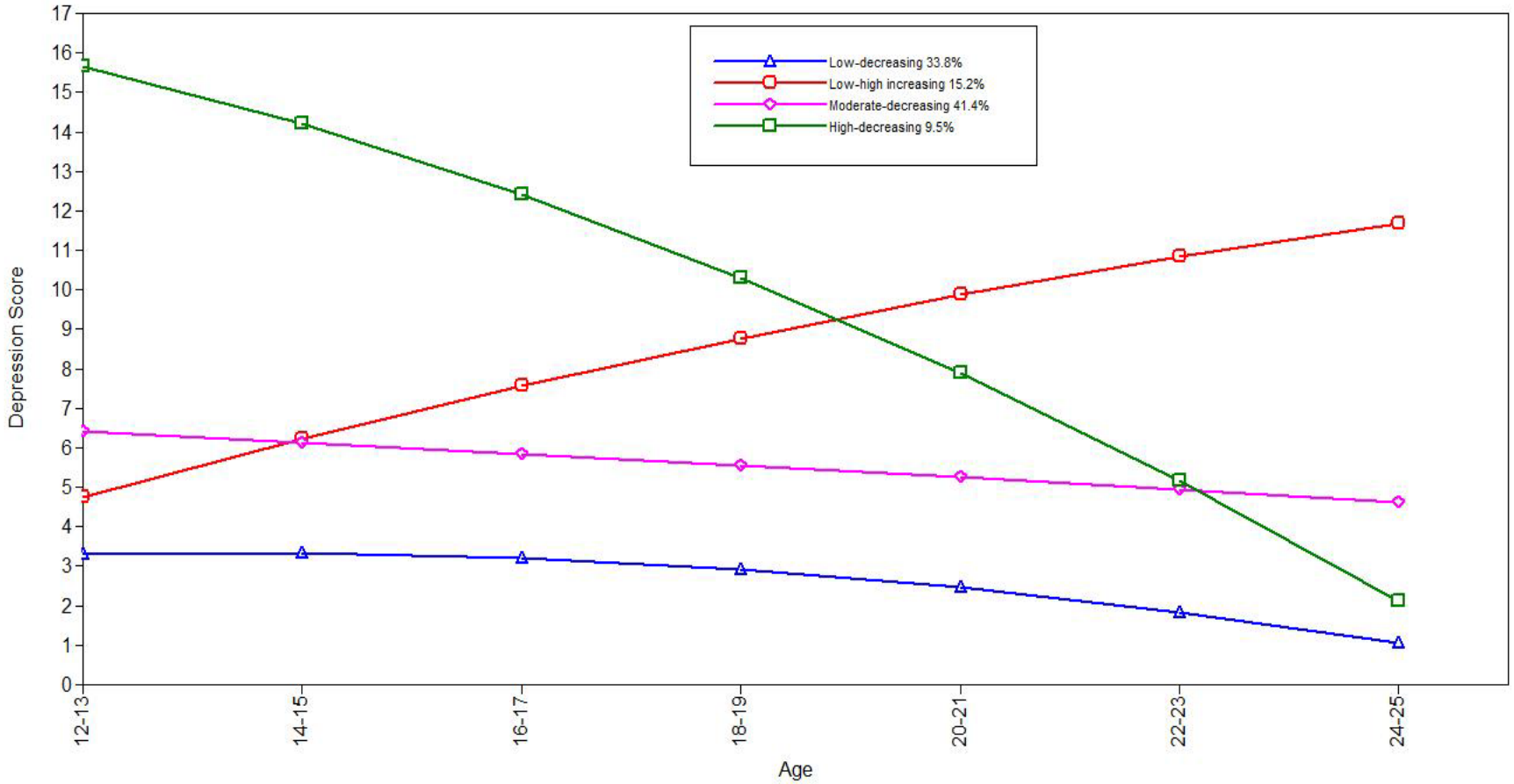
Unconditional Quadratic Growth Model Estimated Mean Depression Scores[a] by Age.



Note: [a] = Nine-item CES-D (Center for Epidemiologic Studies Depression Scale) score.

Figure 3.

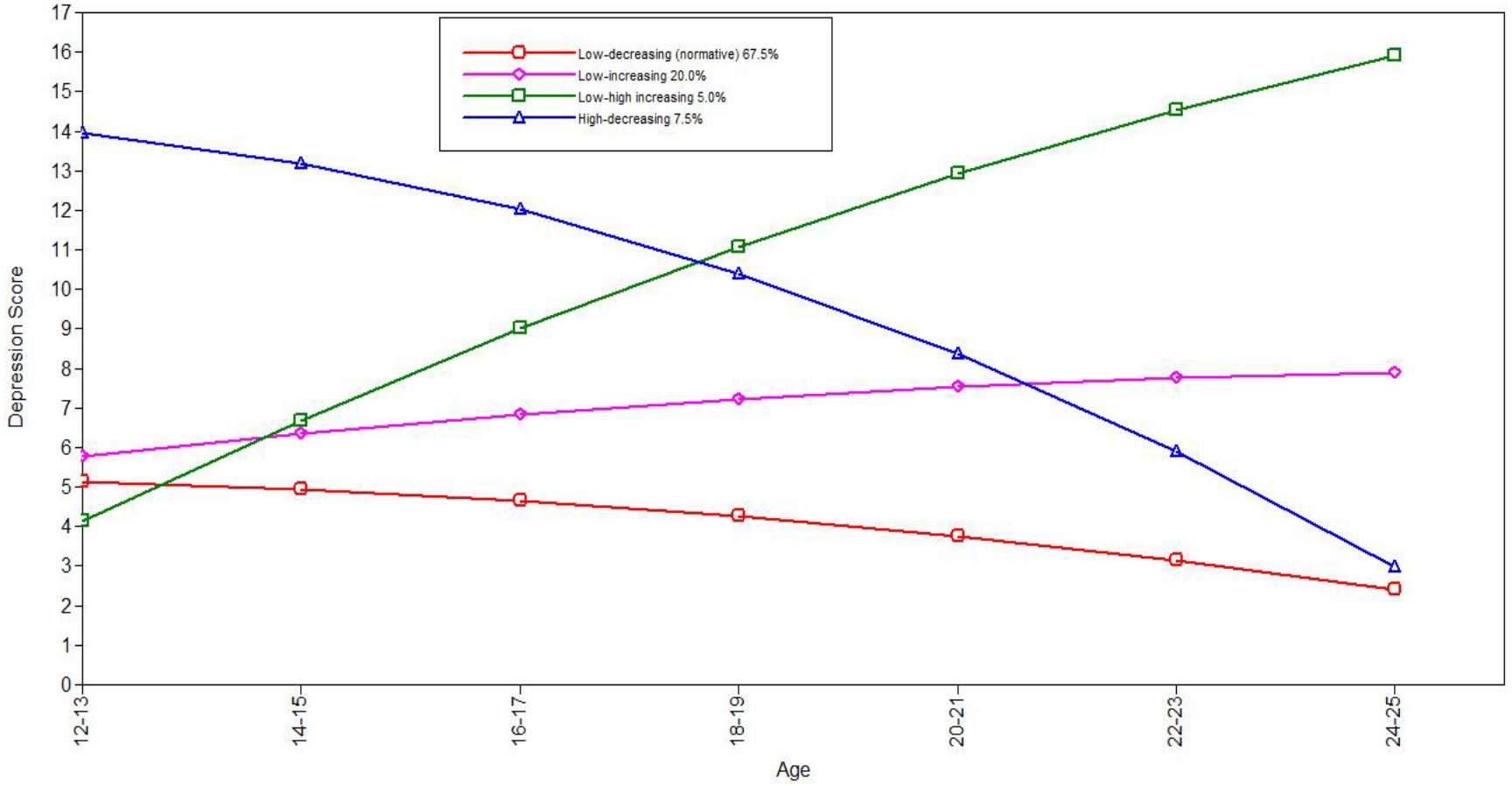
Four-Class Unconditional Growth Mixture Model Estimated Depression Score[a] Trajectories.



Note: [a] = Nine-item CES-D (Center for Epidemiologic Studies Depression Scale) score.

Figure 4.

Conditional Growth Mixture Model Estimated Depression Score[a] Trajectories.



Note: [a] = Nine-item CES-D (Center for Epidemiologic Studies Depression Scale) score.