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Cognitive and interpersonal vulnerabilities to adolescent depression: Classification of risk profiles and implications for personalized prevention

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Abstract

Despite interest in psychosocial vulnerabilities to depression, little is known about reliable and valid individualized risk profiles that can be used to match individuals to evidence-based interventions for depression. This study investigated well-established cognitive and interpersonal vulnerabilities to depression among youth to discern an evidence-based risk classification approach which is being used in a personalized depression prevention randomized clinical trial. Data were drawn from a general community sample of adolescents ($N=467$; ages 10–16, mean 13.14, $SD = 1.62$; 57% females) who were followed prospectively for 3 years. Youth completed measures of cognitive (negative cognitive style, dysfunctional attitudes, rumination) and interpersonal (support and conflict with peers and parents, excessive reassurance seeking, social competence, co-rumination) risks to depression, and then were followed longitudinally for onset of depression. Principal axis factor analyses showed that three latent factors--cognitive vulnerability, interpersonal support, and interpersonal conflict--optimally represented the structure of these risk factors. Clinically practical and meaningful cutoffs, based on tertile cut-off scores on cognitive and interpersonal risk measures, were used to categorize youth into relatively balanced high and low cognitive and interpersonal risk groups. These risk classification groups exhibited validity ($AUC > .70$) by predicting prospective onsets of depressive episodes at 18-months follow-ups. These findings demonstrate a reliable and valid approach to synthesize psychosocial vulnerabilities to depression, specifically cognitive and interpersonal risks. Results are discussed in terms of using these risk classifications profiles to test personalized prevention of depression during adolescence.

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Depression is a common, costly, debilitating disorder. Depression is ranked as the number one cause of disability and will be the second most important disorder in terms of burden of disease by 2020 (WHO, 2001). Prevalence rates of depression surge during adolescence (Hankin et al., 1998). Adolescent-onset depression substantially increases risk for continuity and recurrence of depression into adulthood, and most cases of recurrent depression have initial onset in adolescence (Kim-Cohen et al., 2003). Thus, there is a critical need to focus on prevention in adolescence to reduce the burden of depression (Muñoz, Cuijpers, Smit, Barrera, & Leykin, 2010). Prevention programs have the potential to reach a larger proportion of the population and, if efficacious, can prevent the onset of depression and its associated impairments.

Personalizing preventive interventions to maximize effects

A number of depression prevention programs have been developed and tested with adolescents, primarily based on cognitive-behavioral or interpersonal approaches. Despite several trials demonstrating the efficacy of these preventions, the effect sizes for these prevention programs are small to moderate (Horowitz & Garber, 2006; Merry et al., 2011; Stice, Shaw, Bohon, Marti, & Rohde, 2009). One explanation for the relatively modest impact of depression prevention programs is that these interventions have not been designed for individualization based on known risk factors for depression. In other words, these programs are based on a “one size fits all” approach. This approach assumes, for instance, that cognitive-behavioral prevention programs will be equally effective for youth who are at high cognitive risk for depression as well as those who report low cognitive vulnerabilities.

We are presently conducting a randomized clinical trial (RCT) that is examining an approach to providing personalized depression prevention to adolescents, in line with the recent emphasis on precision medicine for physical and mental health (Hamburg & Collins, 2010; Kapur, Phillips & Insel, 2012). In this RCT, called the Personalized Depression Project (PDP), we match and mismatch youth to two evidence-based depression prevention programs which target different risk factors for depression. Coping with Stress (CWS; Garber et al., 2009), a cognitive-behavioral based prevention program, focuses on reducing negative thinking patterns, such as dysfunctional attitudes, rumination, and negative inferences about stress. Interpersonal Psychotherapy-Adolescent Skills Training (IPT-AST; Young, Mufson, & Schueler, 2016) is an interpersonal program which aims to improve one’s close relationships (e.g., parents and peers), specifically to increase social support and reduce conflict within these relationships. The goal of the PDP study is to examine whether youth who receive a match between risk status and prevention (e.g., high cognitive risk receiving CWS) experience fewer depression symptoms and diagnoses over time as compared to youth who receive non-personalized prevention (e.g., high cognitive risk receiving IPT-AST).

In order to examine the benefits of personalizing depression prevention programs, it is necessary to first establish an evidence-based risk classification approach that can be used to match individuals to these interventions based on their particular psychosocial risks. This current report provides information on the steps taken and evidence garnered in support of the creation and validation of the risk classification approach used in our PDP trial. The

primary goal of developing this classification system was to form relatively balanced risk groups utilizing theoretically-based and empirically-supported cognitive and interpersonal vulnerability measures for matching and mismatching in the PDP trial, with sufficient sample sizes in each of the four classification groups (low/low, high cognitive/low interpersonal, low cognitive/high interpersonal, high/high). In developing this classification system, we prioritized simplicity and practicality so the classification system could be easily translated into clinical practice to inform decisions about which intervention to provide to a given adolescent. Below we articulate the gaps in the literature and the process by which we proceeded to create, test, and validate this classification approach.

Establishing a valid risk group classification for youth depression

Although there is evidence of the importance of both cognitive and interpersonal vulnerabilities for prospectively predicting adolescent depression (Hammen & Shih, 2014; Hankin, Snyder, & Gulley, 2016), further empirical steps are needed before these vulnerabilities can be used to guide decisions about personalized prevention. First, little research has examined the degree of overlap among key cognitive (e.g., dysfunctional attitudes, negative cognitive style, rumination) and interpersonal measures (e.g., parent and peer conflict, low social support, low social competence, excessive reassurance seeking), and how these psychosocial risks can be optimally organized. This is a fundamental question of construct validation, an essential practice for accurate representation and measurement of core latent constructs (Flake, Pek, & Hehman, 2017). The few studies that have examined the latent factor structure of cognitive vulnerabilities suggest that these risks, while moderately inter-correlated, load onto independent factors, and can be differentiated from broad personality risk, such as neuroticism (Adams, Abela, & Hankin, 2007; Hankin, Lakdawalla, Carter, Abela, & Adams, 2007). To our knowledge, no research has addressed the question of factorial independence for interpersonal risks. In addition, no study has examined the degree of overlap, construct validity, and latent structure of multiple cognitive and interpersonal vulnerabilities.

Second, we sought to obtain a set of risk measures that could be used to classify individuals into high and low cognitive and interpersonal risk groups in a relatively simple, efficient manner to increase the clinical utility of these established risk measures. We aimed to have as few measures as possible to optimally characterize the structure of psychosocial risks, which then could be used to create balanced high and low cognitive and interpersonal risk classification groups. A key feature is to have sufficient measurement that conceptually covers the most fundamental aspects of the cognitive and interpersonal factors while minimizing measurement burden for practical clinical use. To achieve our goal of having an efficient battery, we used factor analysis to identify the highest loading measures that would reliably and validly delineate each of the obtained latent factors. Given that we developed this risk group classification system for our PDP trial with the aim of examining the benefits of matching youth to cognitive and interpersonal prevention programs, we were particularly interested in identifying the cognitive and interpersonal vulnerabilities that had the highest factor loadings and were specifically targeted in CWS and IPT-AST, the evidence-based cognitive-behavioral and interpersonal prevention programs we chose to implement in PDP.

Next, we aimed to establish practical, feasible cutoffs for these cognitive and interpersonal measures that could be utilized to classify individuals into high and low cognitive and interpersonal risk. Depending on a clinical scientist's particular purpose, different cutoffs can be generated and validated. For the purposes of personalized prevention in our PDP trial, we were interested in cutoffs that would create relatively equally balanced groups of individuals classified into four cognitive and interpersonal risk groups. We aimed to obtain relatively equally balanced groups so there would be maximal power to test our personalized prevention approach in our PDP trial. If the evidence shows that a relatively equally balanced 2×2 risk classification grouping exists with sufficient high/low individuals forming the "off diagonals", then this would suggest that personalization of depression prevention could occur by matching individuals based on their risk classification profile (e.g., high cognitive and low interpersonal risk) to an intervention that targets these risks (e.g., CWS).

Fourth, we validated these risk classification groups by examining the extent to which these high and low cognitive and interpersonal groups predicted the subsequent onset of depressive episodes in a 3-year multiwave longitudinal design. Past research supports specific, independent cognitive and interpersonal vulnerabilities as predictors of future depression in adolescents (Abela & Hankin, 2008; Hankin, 2012). Yet, no research has explicitly evaluated whether categorized risk, which combines multiple cognitive and interpersonal risks into meaningfully coherent groupings, predicts future onsets of depression during adolescence. As such, this study contributes novel information on how cognitive and interpersonal risks predict later depression. We hypothesized that the high cognitive/high interpersonal group would exhibit the highest prospective incidence of depression onset over time, followed next by each of the two high/low groups, and last by the low cognitive/low interpersonal risk group showing the lowest rates of depression onset.

In summary, this study addressed the following questions: (1) What are the interrelations among the predominantly studied cognitive and interpersonal risks? (2) How many factors provide an optimal structure that best represents these cognitive and interpersonal vulnerabilities, and what are those latent risk factors? (3) Can efficient, practical, transportable, and clinically meaningful cutoffs be instantiated on the main measures that comprise these latent risk factors to create high and low cognitive and interpersonal risk groups, respectively? There are potentially different answers to this question, and the proposed solutions may depend on different rationales and purposes. In the current study, we focused on a solution that would result in a balanced 2×2 classification and was practical and transportable to other settings. (4) What is the validity of these risk groups, particularly with regard to predicting longitudinal outcomes? In addition, we replicated the steps used to create and validate these risk classifications to test for robustness, including reliability and validity. As such, this study addressed key, unresolved questions regarding the classification of risk groups based on cognitive and interpersonal vulnerabilities to depression, with an eye toward translating this knowledge into personalizing depression prevention.

Method

Participants and Procedures

Youth from the general community were recruited at two sites for the Gene, Environment and Mood (GEM) study: University of Denver (DU) and Rutgers University (RU) (see Hankin et al., 2015 for study and sample details). The GEM study was conducted to investigate developmental trajectories and predictors of depression and socio-emotional functioning among youth over time. The present sample consisted of 467 youth who were originally in the 6th (mean age = 12.14, SD = .54), and 9th grade (mean age = 14.79, SD = .46) cohorts from the GEM study. Youth ranged in age from 10 to 16 years (mean = 13.14, SD = 1.62), were 57% female, and identified their ethnicity as 12% Latino and race as 70% Caucasian, 12% African American, 9% Asian/Pacific Islander, and 9% other/multiracial. Median annual family income was \$86,500. Caretakers who provided parent report were 85% mothers. The sample was generally consistent with the ethnic and racial characteristics of the overall population of the United States, although there were relatively fewer Hispanic participants in the GEM study than found in the overall population of the United States.

The caretaker and youth visited the laboratory for an in-depth assessment at baseline and then at 18- and 36-month follow-ups. Caretakers provided informed written consent for their child's participation; youth provided written assent. After the initial baseline assessment, regular phone follow-up (FU) assessments took place every six months for the next three years, for a total of seven repeated measures assessments. Both the caretaker and youth were interviewed with a semi-structured diagnostic interview. At the baseline and 18-month follow-up time points, the cognitive and interpersonal questionnaires were administered. Retention rate from baseline to 36-month follow-up was 93%. The institutional review boards at University of Denver and Rutgers University approved all procedures. Both youth and the caretaker were compensated monetarily for participation.

Measures

Negative cognitive style was assessed using the Adolescent Cognitive Style Questionnaire (ACSQ; Hankin & Abramson, 2002). The ACSQ assesses negative inferences for cause (internal, stable, global), consequence, and the self from Hopelessness Theory (Abramson et al., 1989). Higher scores indicate a more negative cognitive style. The ACSQ is reliable and valid (Hankin, 2008). Internal consistency in this study was .91.

Dysfunctional attitudes were measured via the Children's Dysfunctional Attitudes Scale (CDAS; Abela & Sullivan, 2003). Higher scores indicate higher levels of dysfunctional attitudes. The CDAS is reliable and valid (Abela et al., 2011). Internal consistency in this study was .85.

Rumination was assessed by the ruminative response subscale of the Children's Response Styles Questionnaire (CRSQ; Abela, Vanderbilt, & Rochon, 2004). Higher scores indicate a greater tendency for youths to focus on negative self-meaning and implications as a response tendency when feeling sad or depressed. The CRSQ is reliable and valid (Abela & Hankin, 2011). Internal consistency was .80 in this sample.

Conflict with and social support from parents and peers was measured by the Network of Relationships Inventory (Furman & Buhrmester, 2009), a 13-item self-report measure that assesses different aspects of relationship quality with various peer relationships and parental figures. Based on past research showing that the NRI has a factor structure of two factors for each relationship (support and conflict), we created composite variables of parent conflict and support (mother and father ratings averaged) as well as peer conflict and support (same-sex friend). Internal reliabilities for all variables were above .80.

Excessive reassurance seeking was measured with the Reassurance-Seeking Scale for Children (RSSC; Joiner & Metalsky, 1995). Higher scores indicate higher levels of reassurance seeking. The RSSC is reliable and valid (Abela, Zuroff, Ho, Adams, & Hankin, 2006). In this study, coefficient alpha was .76.

Social competence was assessed via a subscale of the Self-perception Profile for Children (Harter, 1985). This subscale has been used to assess perceived social competence reliably and validly (Cole, 1990), with higher scores indicating poorer social competence. Internal consistency in this sample was .80.

Co-rumination was measured via nine items from a brief co-rumination questionnaire (Hankin, Stone, & Wright, 2010), which was adapted from the original co-rumination measure (based on items in the appendix of Rose, 2002) to assess the extent to which problems are extensively rehashed with friends. It is reliable and valid (Hankin et al., 2010). Internal consistency in this sample was .88.

Depression diagnoses were assessed by trained interviewers who administered the Mood Disorders and Psychosis subsections of the well-validated Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL; Kaufman et al., 1997). Youth and their caretaker were interviewed at baseline and then every 6 months over the 3 years of follow-up to enhance reliability and validity of diagnostic data given the potential of memory and recall biases with retrospective recall over longer periods of time (Compton & Lopez, 2014). No youth was diagnosed with a bipolar spectrum disorder or psychosis. Diagnostic interview inter-rater reliability was good ($\kappa = .91$) based on approximately 20% of interviews being reviewed for reliability. Interviewers utilized both youth- and parent-report on the K-SADS to determine youths' diagnostic status using best estimate diagnostic procedures (Klein, Dougherty & Olino, 2005). Youth were diagnosed with a depressive episode in the past 6 months as ascertained between each K-SADS FU interview if they met criteria for at least depressed mood or anhedonia plus at least two threshold symptoms from DSM-IV's symptom criteria list for Major Depressive Disorder (MDD); the symptoms lasted a minimum of 2 weeks in duration; and youth demonstrated significant distress and/or impairment (i.e., diagnoses of MDD-Definite, MDD-Probable, or minor Depressive Disorder Definite). Hankin et al. (2015) reported the descriptive statistics on depression diagnoses; briefly 31% of the 6th and 9th grade cohorts experienced a clinically significant episode over the 3 year follow-up, with a significant gender difference (36% of girls versus 24% of boys).

Data Analytic Plan

Analyses were conducted using SAS 9.4. For the first question of interrelations among the psychosocial risks, we conducted Pearson correlations. For the second question regarding how many latent factors characterized these psychosocial risks, we used principal axis factor analysis (PAF), with varimax rotation to obtain orthogonal solutions, with all of the cognitive and interpersonal scale score variables. We did the PAFs using baseline data and then again with the 18-month follow-up data to cross validate the solutions and evaluate the replicability of the latent factor pattern in this sample. We chose to use PAF rather than latent class analyses given our goal was to prospectively classify individuals into risk categories and the ease of assigning risk factor status at a screening assessment through a latent class approach has been challenged by researchers (see Bray, Lanza, & Tan, 2015). Third, we determined clinically relevant cut points on the measures comprising those latent factors that best organized cognitive and interpersonal risks. In this step, we aimed to create a relatively equal balance of youth in each cell of our 2×2 risk matrix (i.e., high and low cognitive and interpersonal vulnerabilities, respectively) which was necessary for the PDP design. We chose to utilize cutpoints, rather than a dimensional approach, as cutpoints can be applied quickly and practically to achieve reliable and valid risk group classification. We explored different variations for cut points on the measures that comprise the latent cognitive and interpersonal factors obtained in step 2. Specifically, we evaluated the utility and spread of median splits, quartiles, and tertiles to form high and low risk group classifications. We cross-validated these cut points using the 18-month follow-up data to evaluate whether a relatively equal balance of youth would be observed in each cell of this 2×2 risk matrix.

Finally, we used the baseline classifications, in which youth were categorized into high or low cognitive or interpersonal risk, based on the 2×2 matrix, to evaluate the validity of this categorization system to predict onset of future depressive episodes 18 months later. We used logistic regression to predict the onset of a depressive episode, from baseline to 18-month FU, using the risk groups as our independent predictor variable and history of clinical depression as a covariate. To evaluate the validity of the risk group classification approach, we were primarily interested in the Area Under the Curve (AUC) for this set of independent predictors and prospective depressive episodes as the outcome; we used the convention of an AUC greater than .70 as an “acceptable” criterion for predictive validity based on receiver operating characteristics (Fischer, Bachmann, & Jaeschke, 2003; Pinteá & Moldovan, 2009). Then we used the data from the 18-month FU risk group classifications, keeping the cut points that were established from the baseline data, to predict onset of depressive episodes from the 18-month FU to the 36-month FU, controlling for history of depression, as a replication check on validity of the risk group classifications.

Results

Interrelations among cognitive and interpersonal risks

Table 1 presents the descriptive statistics as well as correlations among the main study variables at the baseline assessment. Three sets of findings emerged. First, consistent with past research, many of the cognitive and interpersonal vulnerabilities were significantly correlated, although the magnitude of effect sizes ranged considerably from minimal to

moderate. Second, also consistent with past work, there were significant gender and age effects. Girls reported more rumination (baseline: $t(470)=2.71, p=.007$; 18-month FU: $t(387)=4.12, p<.001$), co-rumination (baseline: $t(466)=6.39, p<.001$; 18-month FU: $t(380)=6.62, p<.001$), same sex friend support (baseline: $t(464)=8.61, p<.001$; 18-month FU: $t(386)=5.89, p<.001$), and were more likely to experience a depressive episode compared to boys (through 18-month FU: $t(440)=2.38, p=.018$; through 36-month FU: $t(429)=2.91, p=.004$). Older adolescents (9th grade cohort compared to 6th grade cohort) exhibited more negative cognitive style (baseline: $t(470)=4.80, p<.001$; 18-month FU: $t(91)=4.42, p<.001$), dysfunctional attitudes (baseline: $t(469)=3.98, p<.001$; 18-month FU: $t(387)=3.17, p=.002$), rumination (baseline: $t(470)=3.04, p=.003$; 18-month FU: $t(387)=2.90, p=.004$), same sex friend conflict (baseline: $t(464)=2.13, p=.031$; 18-month FU: $t(382)=1.99, p=.048$), lower perceived parent support (baseline: $t(464)=4.02, p<.001$; 18-month FU: $t(380)=2.98, p=.033$), and a greater likelihood of a depressive episode over time (through 18-month FU: $t(440)=4.66, p<.001$; through 36-month FU: $t(429)=2.70, p=.007$). Finally, providing important preliminary validity for these risk factors, the majority of the individual manifest cognitive and interpersonal vulnerabilities were prospectively associated with later onset of depressive episodes over the 3-year follow-up period, although the effect sizes with prospective prediction of later episodes were smaller than effect sizes seen with concurrent risk associations given the expected decrease in effect size at the longitudinal follow-up.

Exploratory factor analyses to discern the best fitting latent structure to organize cognitive and interpersonal vulnerabilities

We used Principal Axis Factor analysis (PAF) with orthogonal (varimax) rotation including all baseline cognitive and interpersonal risk measures to ascertain the best fitting structure of these psychosocial risks. PAF answers how many latent factors are needed to represent these vulnerabilities and what comprises those latent factors. Table 2 shows the results of factor loadings for the three latent factors that provided the best fit to the data, according to traditional estimation criteria: scree test, eigenvalues > 1 , and interpretability of the solutions. The top portion of Table 2 provides results for baseline risk measures, and the bottom portion of the table shows results from measures assessed at 18-month FU.

Examining the factor loadings to obtain a reliable, replicable pattern from both baseline and 18-month FU assessments revealed that all cognitive vulnerability measures (i.e., rumination, dysfunctional attitudes, and negative cognitive style) comprised Factor 1; Factor 2 included interpersonal social support measures; and Factor 3 was composed of all interpersonal conflict measures. Excessive reassurance seeking was the only manifest variable that did not reliably load onto the same factor at both time points, as it was weakly configured to Factor 1 at baseline and then to Factor 3 at 18-months. As the loadings for excessive reassurance were under our .40 loading cutoff and exhibited an inconsistent pattern, we did not include it in our interpretation of the conceptual core of each latent factor. Given that varimax PAF produces an independent set of factors, we wanted to consider the possibility that the three factors could correlate to some extent. An oblimin rotation of the solution for baseline data showed that Factor 1 correlated $-.13$ with Factor 2 and $.39$ with Factor 3; Factor 2 correlated $.06$ with Factor 3; these relations are consistent with the perspective that the three factors are relatively orthogonal of each other.

Next, we conducted these factor analyses again with the manifest measures that loaded highest on each latent factor and were deemed to be conceptually and empirically important to the core latent construct and related to the theorized mechanisms underlying cognitive-behavioral (CWS) and interpersonal (IPT-AST) prevention programs. This step was taken to evaluate a briefer set of risk measures that could represent these three factors to achieve clinical, practical utility with reduced measurement burden. As with the original PAF results, three latent factors were obtained that represented cognitive risk, social support, and interpersonal conflict at baseline and 18-month FU. Table 3 shows these results. The first factor includes manifest measures of cognitive vulnerability, including negative cognitive style, rumination, and dysfunctional attitudes. The second latent factor was composed of parental support and same-sex peer support, and the third latent factor included parent and same-sex peer conflict. These abbreviated set of measures that comprise the three factors are particularly encouraging for practical, efficient, low-burden clinical use relevant for personalized prevention, especially for PDP, given that CWS targets cognitive risks including dysfunctional attitudes, negative cognitive styles and rumination, whereas IPT-AST focuses on skills that decrease parent and peer conflict and increase support in these relationships.

Creating high and low cognitive and interpersonal risk group classifications

These factor analytic results show that manifest cognitive measures loaded onto a single latent factor independent from the two latent factors upon which the manifest interpersonal measures loaded. As such, we proceeded to the third question: Can efficient, clinically meaningful cutoffs be instantiated on the measures that comprise these latent factors to create high and low risk groups based on cognitive and interpersonal vulnerabilities? We included negative cognitive style, dysfunctional attitudes, and rumination as markers of cognitive risk; parent conflict and same-sex friend peer support comprised the indicators of interpersonal risk. We focused on parent conflict and peer support for two reasons. First, it was important that the two interpersonal factors incorporate information from both parent and peer domains, given that both parent and peer relationships are vulnerabilities for adolescent depression (La Greca & Harrison, 2005) and both are targeted in IPT-AST (Young et al., 2016). Second, across the four sets of factor analyses (full and condensed models at baseline and 18-month FU), peer social support had the highest loading on this second factor in three of the four analyses, and parent conflict had the highest loading on the third factor in three of the four analyses.

We then explored different cutoff rules with these cognitive and interpersonal markers to evaluate which would yield the most balanced 2×2 cognitive and interpersonal risk group classifications, a primary aim relevant for our PDP trial. Results based on different, clinically relevant cutoff strategies showed that using a tertile approach to form high- and low-risk groups yielded the most balanced distribution of youth in each cell across the two time points. Tertile cutoffs for the specific measures were as follows: above 29 on rumination, 3.4 on negative cognitive style, 36 on dysfunctional attitudes, and 15.5 for parent conflict, and below 23 on peer social support. More specifically, scoring above the upper third (or lower third for peer social support) on any one cognitive (out of the three) or any one interpersonal risk (at least one from either of the two interpersonal factors) would

qualify the individual to be characterized as at high risk for that particular vulnerability. As shown in Figure 1 (top half for baseline data), cutoffs using tertile splits produced the most balanced distribution among the 2×2 matrix. Importantly with respect to personalized depression risk classifications in PDP based on cognitive and interpersonal risk groups, the proportion of youth who comprised the “off diagonals” (i.e., high cognitive, low interpersonal risk of 20.8%; high interpersonal, low cognitive risk of 22.7%) was relatively equal. Also important for clinical purposes with translational implications for personalized prevention, there were 37.6% at high cognitive and high interpersonal risk, and only 18.8% at low cognitive and low interpersonal risk. In contrast, the median split approach produced many youth at high cognitive and high interpersonal risk (64.9%), and low proportions of the “off diagonals” (high cognitive, low interpersonal risk of 14.8%; high interpersonal, low cognitive risk of 15.2%) and those at low cognitive and interpersonal risk (5.1%). The quartile approach yielded too many youth at low cognitive and low interpersonal risk (30.2%) to be useful for screening for personalized prevention despite reasonable balance in the other three quadrants (high cognitive, low interpersonal risk of 24%; high interpersonal, low cognitive risk of 20.8%; high cognitive, high interpersonal risk of 25%).

With respect to replication of these proportions and the relative balance among the risk groups, analyses using 18-month FU data showed that the tertile approach again provided the best balance with practical, clinical utility (see bottom half of Figure 1). As with the baseline data, the median split approach at the 18-month FU yielded many youth in the high cognitive and high interpersonal risk group, whereas quartiles produced a high proportion of low cognitive and low interpersonal risk.

Last, given age and gender effects in some cognitive and interpersonal risks, we conducted exploratory analyses to examine potential age and gender differences in these risk group classifications. Gender was not significantly linked to group membership ($\chi^2(3)=7.59$). Age was related significantly to group classification ($\chi^2(3)=11.35$). Follow-up analyses showed that the high cognitive and high interpersonal risk group was older ($M=13.16$, $SD=1.54$) relative to the other groups (low/low $M=12.50$, $SD=1.58$; low cognitive/high interpersonal $M=12.70$, $SD=1.66$; high cognitive/low interpersonal $M=12.87$, $SD=1.61$).

Validity of the risk classification groups: Predicting prospective depression episodes

Using tertile splits as the approach to classify youth into high and low cognitive and interpersonal risk groups with relevance for PDP, we then proceeded to evaluate the degree to which these groups demonstrated validity in predicting future episodes of clinical depression as assessed prospectively over an 18-month follow-up. Logistic regressions with risk classification group as the primary independent variable, and using history of clinical depression as a covariate, to predict onset of depressive episode over the 18-month FU resulted in acceptable validity with Area Under the Curve (AUC) = .72. We repeated these analyses with the 18-month FU risk classification grouping data (using the baseline tertile cutoffs on the 18-month FU data) to predict onset of depressive episodes over the next 18 months (i.e., from 18-month FU to 36-month FU), covarying prior history of depression. The AUC was .72, demonstrating acceptable validity. As both sets of analyses exhibited $AUC > .70$, which is a standard criterion for predictive validity (Fischer et al., 2003; Pinte

& Moldovan, 2009), for predicting future depressive episodes over 18 months, these risk classification groupings showed acceptable validity for the key external criterion outcome for which these risk classifications were intended. Figure 2 illustrates the rates of depression onset over 18 months of follow-up (i.e., baseline to 18-month FU and 18-month FU to 36-month FU). Specifically, the high cognitive/high interpersonal risk group exhibited the highest percentage of depression disorder diagnoses; followed by the low interpersonal/high cognitive and then high interpersonal/low cognitive groups; and the low interpersonal/low cognitive risk group experienced the lowest incidence of prospective depressive episodes.

Discussion

A major barrier and challenge to examining personalized interventions is how best to match individuals to an evidence-based intervention based on their individualized, stratified risk profile in a reliable and valid manner that can be translated feasibly, easily, and practically into clinical practice. As such, the goal of the current study was to develop and examine one approach to risk classification using well-studied and empirically-supported individual cognitive and interpersonal vulnerabilities to depression that are targeted via existing depression interventions. This risk group classification facilitates steps toward testing personalized interventions for depression by matching youth in different risk groups to either a cognitive-behavioral or interpersonal evidence-based program. Results of this study provide the first demonstration that such a risk group classification can be achieved with good reliability, validity, and practical clinical utility. We discuss main study findings and implications for personalization of depression intervention among adolescents based on this classification approach.

Principal axis factor analyses using data from both the baseline and 18-month FU assessments showed that three latent factors provided the best solution and provided a simple, conceptually sensible structure to organize multiple cognitive and interpersonal vulnerabilities into fewer latent factors. All of the manifest cognitive risks, including negative cognitive style, dysfunctional attitudes, and rumination, loaded onto the first factor, which reflects cognitive vulnerability to depression. The second factor was composed of interpersonal social support measures, including from parents (mother and father) and same-sex peers, whereas measures tapping interpersonal conflict with parents and same-sex peers comprised the third factor. In sum, results showed that many of the commonly studied cognitive and interpersonal risk factors, from various theoretically distinct conceptual models, can be organized and structured in a simpler manner with good construct validity.

We then sought to obtain a practical and clinically useful approach to indexing risk to depression via cutoffs that create high and low cognitive and interpersonal risk groups for depression propensity. Results showed that using a tertile cut point on the manifest cognitive and interpersonal vulnerability measures enabled us to produce a relatively balanced 2×2 grouping using our baseline data, and this solution was replicated using the 18-month FU data. Scoring in the upper third on at least one of the three cognitive risks, and scoring in the upper third on parent conflict and/or the lower third on peer support (across the two separate interpersonal factors) was found to be efficient for spreading adolescents into the relatively balanced 2×2 groupings. Of importance and favoring the selection of these cut points and

decision rules, especially with regard to personalized prevention for PDP, relatively equal percentages fell into the “off-diagonal” categories of high/low cognitive and interpersonal risk, and fewer fell into the low/low group for whom prevention programs may be less effective (Horowitz & Garber, 2006; Stice et al., 2009). Finally, it is worth noting that gender was not significantly related to the classification of adolescents into these risk groups, whereas age was related such that older youth were more likely to be classified into the high cognitive and high interpersonal risk group. That older age was significantly associated with only this high/high risk group is expected because cognitive risk continues to develop and crystallize into more depressogenic risks as adolescents mature (Hankin et al., 2009), and interpersonal conflict increases while support (especially with parents) tends to decrease with advancing adolescent age (Furman & Buhrmester, 2009).

Validity of these risk classifications was demonstrated by their predicting future onsets of depressive episodes. The risk classification groupings achieved acceptable AUCs (i.e., $> .70$), which is the traditional standard criterion for evaluating positive predictive power of a risk marker in medicine and social science (e.g., Pinteá & Moldovan, 2009). Moreover, the validity and clinical utility of this group-based risk classification approach is supported by the finding that the high/high cognitive and interpersonal vulnerability group was the most likely to experience a future depressive episode, followed next by the “off-diagonal” risk groups who exhibited an intermediate prospective incidence of depression onset, and finally the low cognitive and interpersonal risk group who demonstrated the smallest prospective depression incidence over time. These findings, including $AUC > .70$ and the pattern of which risk groups experienced prospective depressive episodes, was replicated when we conducted the analyses using the 18-month FU risk grouping data to predict onset of depression from the 18-month FU to the 36-month FU. These findings are novel and go beyond prior studies that have documented that individual cognitive and interpersonal risk factors, as examined in a variable centered manner, predict later depression onset (e.g., Hankin et al., 2016; Rudolph, Lansford, & Rodkin, 2016).

A primary motivation for developing these cutoffs, establishing these decision rules, and evaluating the construct validity of our risk classification group approach was to use these risk groups to inform personalization of depression prevention programs. Intervention outcomes may be improved by matching individuals to the intervention that would best suit the individuals’ needs based on their risk profile. Currently, we are conducting a RCT that uses this risk group classification as one approach that uses *a priori* risk information to personalize prevention of depression among adolescents by matching, and mismatching, youth to either CWS or IPT-AST. Thus the key findings of the current study, in which we arrived at a group-based risk classification approach with good construct and predictive validity, provide important fundamental evidence that personalization of intervention, grounded in this risk group classification, may be a viable way to individualize interventions. The cognitive and interpersonal vulnerabilities are relatively independent risks that can be feasibly and efficiently measured and then used to categorize youth into a relatively balanced set via a 2×2 matrix.

These findings are important for our PDP trial and basic knowledge in clinical science. In particular, we found that our practical cutoffs and selection rules could reliably identify the

“off-diagonal” risk groups (i.e., high cognitive/low interpersonal and high interpersonal/low cognitive), and there was a sufficient number of youth that comprised these “off-diagonal” groups. These groups are particularly important for our PDP approach to personalizing depression prevention which we hope will be successful in bending depression trajectories during adolescence, when the rates of depression are known to surge. If this is the case, this classification system can be utilized in clinical practice to identify which youth would benefit from a given preventive intervention, which has the potential to substantially reduce the prevalence and burden of depression. Other future personalization efforts could build on this rationale and logic to test alternative individualization profiles using matching between relevant risks for a particular disorder and appropriate interventions.

We note three primary limitations of this work. These limitations relate to the analytic approach applied and the measurements that were deemed most pertinent to examining our approach to advancing personalization of depression prevention in youth. Other data analytic options (e.g., latent class analysis) and other sources of data (e.g., genomics, biomarkers, performance on behavioral tasks, psychophysiology) could be used to measure and investigate risk profiles. First, results are most specifically relevant for PDP; other cutoffs could be selected, tested, validated, and used for other purposes. Indeed, one of our hopes of demonstrating this risk group classification approach is that others interested in personalizing intervention may follow our logic and steps to create their own risk groups that are most relevant to a different clinical disorder or individualization strategy using our work as a proof of concept. For example, others may decide that a different cutoff, other than scoring in the upper tertile, may be best for their clinical purposes. Additionally, other investigators may not want to combine the two interpersonal dimensions of support and conflict into a single interpersonal risk factor to determine risk group classifications as we did for PDP. Determining susceptibility based on interpersonal conflict and support separately might be important for individualizing interventions as conceptualized via interpersonal formulations. In sum, several other alternative approaches to determining risk group classification exist and can be tested to meet a clinical scientist’s particular interest; the approach with the decisions made in this report are most pertinent to our purposes for PDP, but are not the sole solution.

Second, the practical risk group classification approach used here is not the only option for personalizing intervention. Others have taken complementary approaches to personalizing risk assessment in the treatment and assessment literatures. For instance, DeRubeis and colleagues in their work on the Personalized Advantage Index (PAI) have used machine learning techniques to analyze whether individual differences, assessed before depression treatment initiation in their RCTs for adult major depression, serve as significant post-hoc moderators of treatment efficacy (DeRubeis et al., 2014; Huibers, Cohen, Arntz, Cuijpers, & DeRubeis, 2015). For example, in a RCT comparing CBT and IPT for adult depression, they found an effect size of .51, which translated into a mean BDI difference of 8.9 points, between those who received their optimal treatment based on their PAI versus those who did not (Huibers et al., 2015). These intriguing results demonstrate that treatment outcomes can be improved by matching individuals to the intervention that would have best suited the individual based on their risk profile as determined by the PAI. In addition, other new machine learning and novel statistical methods are being developed and applied to create

and examine evidence-based risk assessment to predict later depression using big data (Goldstein, Navar, Pencina, & Ioannidis, 2017; Iniesta, Stahl, & McGuffin, 2016; Kessler et al., 2016; Niles et al., 2017). We do not see the PAI, alternative machine learning methods, or our approach as opposing or contradictory solutions to tackling the significant problem of identifying risk relevant for individualizing interventions via evidence-based assessment.

Last, we used cut-offs, as opposed to dimensions, to create our risk classification. This decision was made for purely practical and feasibility reasons. We recognize that information is lost when dichotomizing an underlying dimension, and that the cognitive and interpersonal vulnerabilities examined here exist and are structured as continuous dimensions. Indeed, risk can be classified via a dimensional approach. We conducted additional analyses using equations that find the optimal operating point (OOP), which is the optimal threshold cutpoint on the ROC curve. The OOP can be of clinical use for decision-making; it provides geometrical and algebraic means for the classification of the predicted outcome and provides corresponding sensitivity and specificity. Using this approach, risks can be conceived of and assessed using all relevant interpersonal and cognitive dimensions. In this dimensional approach that includes all markers from both interpersonal factors (same-sex friend support and parent conflict) as well as the cognitive factor, we found that prospective depression onset over the subsequent 18 months was predicted with good sensitivity (73.1% and 71.9%, respectively, for baseline to 18-month FU and 18-month FU to 36-month FU) and moderate specificity (57.2%, and 56.2%, respectively) with relative consistency between the two time periods (baseline and 18-month FU)¹. Further analyses examined whether we lost significant amounts of discrimination using our cutpoint approach, as opposed to a dimensional solution with continuous measures of risk. Discrimination quantified by the AUC was .74 (SE=.009) for the dimensional measures of risk prediction of depression onset at the 18-month follow-up, and AUC was .76 for depression onset between 18-month FU and 36-month FU. Statistical comparison of the AUCs using continuous risk measures, in contrast to the AUCs for our cutoff approach, showed a nonsignificant difference for both the 18-month FU ($\chi^2(1) = 1.02, p=0.31$) and 36-month FU ($\chi^2(1) = 1.69, p=0.19$) predictions. In summary, note that the results using a dimensional approach closely match the acceptable AUC found using our cut score approach. Having simple cut scores creates easy-to-understand decision rules that can be applied in a quick, reliable, and valid assessment to achieve risk group classification.

An important result of the risk group classification approach, which categorizes youth into high and low cognitive and interpersonal vulnerability to depression, is that it can be used in our PDP RCT, which seeks to bend depression trajectories significantly more so as compared to a “one-size fits all” approach. Further, we hope that the logic to developing, testing, and validating a risk group classification approach demonstrates useful proof of concept for other clinical scientists interested in forming their own risk groups, based on disorder-relevant risks that match disorder-pertinent interventions.

¹Please contact the first author for specifics of these analyses and results.

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References

- Abela JRZ, Hankin BL. Cognitive vulnerability to depression in children and adolescents: A developmental psychopathology approach. In: Abela JRZ, Hankin BL, editors *Handbook of Child and Adolescent Depression*. New York, NY: Guilford Press; 2008. 35–78.
- Abela JRZ, Hankin BL. Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: A multiwave longitudinal study. *Journal of Abnormal Psychology*. 2011; 120(2):259–271. DOI: 10.1037/a0022796 [PubMed: 21553940]
- Abela JRZ, Stolorow D, Mineka S, Yao S, Zhu XX, Hankin BL. Cognitive vulnerability to depressive symptoms in urban and rural Hunan, China: A multi-wave longitudinal study. *Journal of Abnormal Psychology*. 2011; 120:765–778. [PubMed: 21910514]
- Abela JRZ, Sullivan C. A test of Beck's cognitive diathesis-stress theory of depression in early adolescents. *Journal of Early Adolescence*. 2003; 23(4):384–404.
- Abela JRZ, Vanderbilt E, Rochon A. A test of the integration of the response styles and social support theories of depression in third and seventh grade children. *Journal of Social and Clinical Psychology*. 2004; 23(5):653–674.
- Abela JRZ, Zuroff DC, Ho MHR, Adams P, Hankin BL. Excessive reassurance seeking, hassles, and depressive symptoms in children of affectively ill parents: A multiwave longitudinal study. *Journal of Abnormal Child Psychology*. 2006; 34:171–187. [PubMed: 16555142]
- Abramson LY, Metalsky GI, Alloy LB. Hopelessness depression: A theory-based subtype of depression. *Psychological Review*. 1989; 96(2):358–372.
- Adams P, Abela JRZ, Hankin BL. Factorial categorization of depression-related constructs in early adolescents. *Journal of Cognitive Psychotherapy*. 2007; 21(2):123–139.
- Bray BC, Lanza ST, Tan XM. Eliminating bias in classify-analyze approaches for latent class analysis. *Structural Equation Modeling: A Multidisciplinary Journal*. 2015; 22:1–11. [PubMed: 25614730]
- Cole DA. Relation of social and academic competence to depressive symptoms in childhood. *Journal of Abnormal Psychology*. 1990; 99(4):422–429. [PubMed: 2266218]
- Compton WM, Lopez MF. Accuracy in reporting past psychiatric symptoms: The role of cross-sectional studies in psychiatric research. *JAMA Psychiatry*. 2014; 71(3):233–234. [PubMed: 24401961]
- DeRubeis RJ, Cohen ZD, Forand NR, Fournier JC, Gelfand LA, Lorenzo-Luaces L. The personalized advantage index: Translating research on prediction into individualized treatment recommendations. A demonstration. *PLoS ONE*. 2014; 9(1):1–8.
- Fischer JE, Bachmann LM, Jaeschke R. A readers' guide to the interpretation of diagnostic test properties: Clinical example of sepsis. *Intensive Care Medicine*. 2003; 29:1043–1051. [PubMed: 12734652]
- Flake JK, Pek J, Hehman E. Construct validation in social and personality research: Current practice and recommendations. *Social Psychological and Personality Science*. 2017; doi: 10.1177/1948550617693063
- Furman W, Buhrmester D. Methods and measures: The Network of Relationships Inventory: Behavioral Systems Version. *International Journal of Behavioral Development*. 2009; 33(5):470–478. [PubMed: 20186262]
- Garber J, Clarke G, Weersing VR, Beardslee WR, Brent DA, Gladstone TR, ... Iyengar S. Prevention of depression in at risk-adolescents: A randomized controlled trial. *Journal of the American Medical Association*. 2009; 301:2215–2224. [PubMed: 19491183]
- Goldstein BA, Navar AM, Pencina MJ, Ioannidis J. Opportunities and challenges in developing risk prediction models with electronic health records data: A systematic review. *Journal of the American Medical Informatics Association*. 2017; 24:198–208. [PubMed: 27189013]

- Hamburg MA, Collins FS. The path to personalized medicine. *The New England Journal of Medicine*. 2010; 363(4):301–304. [PubMed: 20551152]
- Hammen CL, Shih J. Depression and interpersonal processes. In: Gotlib IH, Hammen CL, editors *Handbook of depression*. 3. New York, NY: Guilford Press; 2014. 277–295.
- Hankin BL. Cognitive vulnerability-stress model of depression during adolescence: Investigating depressive symptom specificity in a multi-wave prospective study. *Journal of Abnormal Child Psychology*. 2008; 36(7):999–1014. [PubMed: 18437551]
- Hankin BL. Future directions in vulnerability to depression among youth: Integrating risk factors and processes across multiple levels of analysis. *Journal of Clinical Child & Adolescent Psychology*. 2012; 41(5):695–718. [PubMed: 22900513]
- Hankin BL, Abramson LY. Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Clinical Child and Adolescent Psychology*. 2002; 31(4):491–504. [PubMed: 12402568]
- Hankin BL, Abramson LY, Moffitt TE, Silva Pa, McGee R, Angell KE. Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*. 1998; 107:128–140. [PubMed: 9505045]
- Hankin BL, Lakdawalla Z, Carter IL, Abela JRZ, Adams P. Are neuroticism, cognitive vulnerabilities and self-esteem overlapping or distinct risks for depression? Evidence from exploratory and confirmatory factor analyses. *Journal of Social and Clinical Psychology*. 2007; 26(1):29–63.
- Hankin BL, Oppenheimer C, Jenness J, Barrocas A, Shapero BG. Developmental origins of cognitive vulnerabilities to depression: Review of processes contributing to stability and change across time. *Journal of Clinical Psychology*. 2009; 65(12):1327–1338. [PubMed: 19827008]
- Hankin BL, Snyder HR, Gulley LD. Cognitive risks in developmental psychopathology. In: Cicchetti D, editor *Developmental Psychopathology, Maladaptation and Psychopathology*. 3. Hoboken, N.J: Wiley; 2016. 312–385.
- Hankin BL, Stone L, Wright PA. Corumination, interpersonal stress generation, and internalizing symptoms: Accumulating effects and transactional influences in a multiwave study of adolescents. *Development and Psychopathology*. 2010; 22:217–235. [PubMed: 20102657]
- Hankin BL, Young JF, Smolen A, Jenness JL, Gulley LD, Technow JR, Barrocas Gottlieb A, Cohen JR, Oppenheimer CW. Depression from childhood in late adolescence: Influence of gender, development, genetic susceptibility, and peer stress. *Journal of Abnormal Psychology*. 2015; 124:803–816. [PubMed: 26595469]
- Harter S. *Self-perception profile for children manual*. 1985.
- Horowitz JL, Garber J. The prevention of depressive symptoms in children and adolescents: A meta-analytic review. *Journal of Consulting and Clinical Psychology*. 2006; 74:401. [PubMed: 16822098]
- Huibers MJH, Cohen ZD, Lemmens LHJM, Arntz A, Peeters FPML, Cuijpers P, DeRubeis RJ. Predicting optimal outcomes in cognitive therapy or interpersonal psychotherapy for depressed individuals using the personalized advantage index approach. *PLoS ONE*. 2015; 10(11):1–16. DOI: 10.1371/journal.pone.0140771
- Iniesta R, Stahl D, McGuffin P. Machine learning, statistical learning and the future of biological research in psychiatry. *Psychological Medicine*. 2016; 46(12):2455–2465. [PubMed: 27406289]
- Joiner TE, Metalsky GI. A prospective test of an integrative interpersonal theory of depression: A naturalistic study of college roommates. *Journal of Personality and Social Psychology*. 1995; 69(4):778–788. [PubMed: 7473031]
- Kapur S, Phillips AG, Insel TR. Why has it taken so long for biological psychiatry to develop clinical tests and what to do about it? *Molecular Psychiatry*. 2012; 17:1174–1179. [PubMed: 22869033]
- Kaufman J, Birmaher B, Brent D, Rao UMA, Flynn C, Moreci P, ... Ryan N. Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1997; 36(7):980–988. [PubMed: 9204677]
- Kessler RC, van Loo HM, Wardenaar KJ, Bossarte RM, Brenner LA, Cai T, ... Nierenberg AA. Testing a machine-learning algorithm to predict the persistence and severity of major depressive

- disorder from baseline self-reports. *Molecular Psychiatry*. 2016; 21(10):1366–1371. [PubMed: 26728563]
- Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. Prior juvenile diagnoses in adults with mental disorder. *Archives of General Psychiatry*. 2003; 60:709–717. [PubMed: 12860775]
- Klein DN, Dougherty LR, Olinio TM. Toward guidelines for evidence-based assessment of depression in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*. 2005; 34:412–432. [PubMed: 16026212]
- La Greca AM, Harrison HM. Adolescent peer relations, friendships, and romantic relationships: do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology*. 2005; 34(1):49–61. [PubMed: 15677280]
- Merry SN, Hetrick SE, Cox GR, Brudevold-Iversen T, Bir JJ, McDowell H. Psychological and educational interventions for preventing depression in children and adolescents. *Evidence-Based Child Health: A Cochrane Review Journal*. 2011; 7(5):1409–1685.
- Muñoz RF, Cuijpers P, Smit F, Barrera AZ, Leykin Y. Prevention of major depression. *Annual Review of Clinical Psychology*. 2010; 6:181–212.
- Niles AN, Loerinc AG, Krull JL, Roy-Byrne P, Sullivan G, Sherbourne CD, ... Craske MG. Advancing personalized medicine: Application of a novel statistical method to identify treatment moderators in the coordinated anxiety learning and management study. *Behavior Therapy*. 2017; 48(4):490–500. [PubMed: 28577585]
- Pintea S, Moldovan R. The Receiver-Operating Characteristic (ROC) analysis: Fundamentals and applications in clinical psychology. *Journal of Cognitive and Behavioral Psychotherapies*. 2009; 9(1):49–66.
- Rose AJ. Co-rumination in the friendships of girls and boys. *Child Development*. 2002; 73(6):1830–1843. [PubMed: 12487497]
- Rudolph KD, Lansford JE, Rodkin PC. Interpersonal theories of developmental psychopathology. In: Cicchetti D, editor *Developmental Psychopathology, Maladaptation and Psychopathology*. 3. Hoboken, N.J: Wiley; 2016. 312–385.
- Stice E, Shaw H, Bohon C, Marti CN, Rohde P. A meta-analytic review of depression prevention programs for children and adolescents: factors that predict magnitude of intervention effects. *Journal of Consulting and Clinical Psychology*. 2009; 77:486–503. [PubMed: 19485590]
- World Health Organization. *Mental Health: A Call for Action by World Health Ministers*. World Health Organization; 2001.
- Young JF, Mufron L, Schueler CM. *Preventing Adolescent Depression: Interpersonal Psychotherapy-Adolescent Skills Training*. NY: Oxford University Press; 2016.

Baseline data (N=467)

Cognitive vulnerability	Interpersonal vulnerability	
	Low	High
Low	N = 86 18.8%	N = 105 22.7%
High	N = 108 20.8%	N = 168 37.6%

18-month follow-up data (N=387)

Cognitive vulnerability	Interpersonal vulnerability	
	Low	High
Low	N = 85 22%	N = 98 25.3%
High	N = 84 21.7%	N = 120 31%

Figure 1.

Results of youth comprising the 2×2 matrix of risk group classification for high and low levels of cognitive and interpersonal vulnerabilities (based on tertile cutoffs)

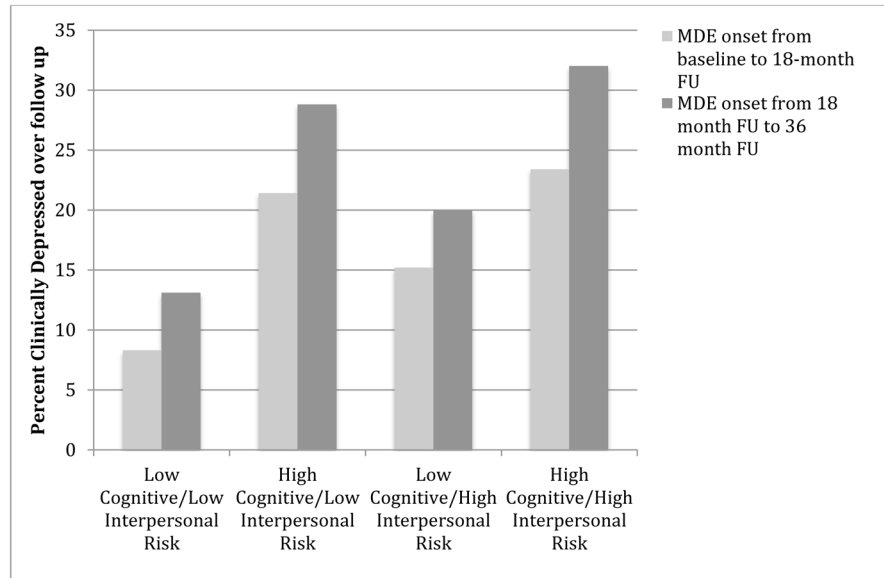


Figure 2. Predicting prospective depressive episodes based on risk group classification of cognitive and interpersonal vulnerabilities.

Table 1
 Descriptive Statistics and Correlations Among Cognitive and Interpersonal Vulnerabilities at Baseline and Later Depression Onset

Variable	Negative Cognitive Style	Dysfunctional Attitudes	Rumination	Social Competence	Excessive Reassurance-Seeking	Co-rumination	Same-Sex Friend Social Support	Same-Sex Friend Conflict	Parent Social Support	Parent Conflict	Depression Onset to 36 Months
Negative Cognitive Style	--										
Dysfunctional Attitudes	.46**	--									
Rumination	.49**	.44**	--								
Social Competence	.23**	.15**	.26**	--							
Excessive Reassurance-Seeking	.15**	.20**	.27**	.15**	--						
Co-rumination	.15**	.12*	.26**	-.14**	.11*	--					
Same-Sex Friend Social Support	-.09	-.07	.01	-.33**	-.04	.38**	--				
Same-Sex Friend Conflict	.08	0.3	.18**	.06	.10*	.10*	-.01	--			
Parent Social Support	-.18**	-.10*	-.10*	-.27**	.01	.15**	.33**	-.25	--		
Parent Conflict	.26**	.21**	.25**	.04	.08	.16**	.11*	.36**	-.25**	--	
Depression Onset to 36 months	.23**	.19**	.27**	.11*	.08	.11*	-.10*	.04	-.11*	.15**	--
<i>Mean</i>	2.97	33.25	26.39	11.43	4.35	25.42	25.24	10.92	25.67	13.63	31%
<i>SD</i>	.88	7.43	7.87	3.90	1.07	8.27	6.15	4.355	.37	4.98	.46

Note: N = 467.

* $p < 0.05$,

** $p < 0.01$.

“Depression onset to 36 months” indicates that an adolescent received a research-based depression diagnosis at some point across the 36 months of prospective follow-ups using the KSADs interview administered repeatedly at regular 6-month intervals. See Method for additional details.

Table 2

Varimax rotated factor loadings of manifest cognitive and interpersonal measures.

Manifest Scale Measure	Factor 1	Factor 2	Factor 3
<i>Baseline data, N=467</i>			
Rumination	.67	.01	.22
Negative Cognitive Style	.62	.06	.08
Dysfunctional Attitudes	.59	.06	.08
Excessive Reassurance Seeking	.32	.001	.02
Same-Sex Friend Social Support	.03	.64	.07
Co-Rumination	.26	.48	.15
Parent Social Support	.09	.46	.21
Social Competence	.28	-.44	.01
Parent Conflict	.19	.002	.59
Same-Sex Friend Conflict	.06	.01	.46
Eigenvalue for factor	2.48	2.18	1.09
% variance explained by factor	24.81%	21.77%	10.98%
<i>18-month follow-up data, N=387</i>			
Rumination	.62	.01	.36
Negative Cognitive Style	.69	.16	.18
Dysfunctional Attitudes	.59	.04	.18
Excessive Reassurance Seeking	.21	.07	.30
Same-Sex Friend Social Support	.15	.65	.05
Co-Rumination	.35	.43	.19
Parent Social Support	.15	.44	.19
Social Competence	.31	-.47	.09
Parent Conflict	.22	.09	.48
Same-Sex Friend Conflict	.09	.06	.48
Eigenvalue for factor	3.02	1.92	1.48
% variance explained by factor	30.27%	19.27%	14.77%

Note: Factor loadings >.40 are in boldface.

Table 3

Varimax rotated factor loadings of manifest cognitive and interpersonal measures for abbreviated set of measures.

Manifest Scale Measure	Factor 1	Factor 2	Factor 3
<i>Baseline data</i>			
Rumination	.68	.01	.18
Negative Cognitive Style	.70	-.12	.11
Dysfunctional Attitudes	.65	-.06	.04
Same-Sex Friend Social Support	-.04	.52	.11
Parent Social Support	-.08	.66	-.12
Parent Conflict	.16	-.05	.82
Same-Sex Friend Conflict	.07	.04	.44
Eigenvalue for factor	2.17	1.33	1.18
% variance explained by factor	31.07%	19.13%	16.97%
<i>18-month follow-up data</i>			
Rumination	.67	.01	.21
Negative Cognitive Style	.84	-.11	.06
Dysfunctional Attitudes	.60	-.11	.05
Same-Sex Friend Social Support	.04	.95	.06
Parent Social Support	-.17	.87	-.04
Parent Conflict	.27	-.05	.87
Same-Sex Friend Conflict	.03	-.05	.82
Eigenvalue for factor	2.63	1.63	1.38
% variance explained by factor	37.45%	23.25%	19.73%

Note: Factor loadings >.40 are in boldface.