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ORIGINAL ARTICLE

Transdiagnostic Mechanisms of Psychopathology in Youth: Executive Functions, Dependent Stress, and Rumination

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Abstract

Executive function (EF) deficits have been proposed as transdiagnostic risk factors for psychopathology, and recent research suggests EF impairments are associated with what is shared across forms of psychopathology (p factor). However, most research has not employed methods that differentiate between EF components, and little is known about the mediating mechanisms linking EF and psychopathology dimensions. The current study tested associations between the latent unity/diversity model of EF and latent dimensions of psychopathology and investigated mediating mechanisms in a community sample of 292 youth age 13–22. The results confirmed the finding that poor EF is associated with internalizing psychopathology in older youth via higher dependent stress and rumination, and showed that this pathway was transdiagnostic, predicting the p factor rather than internalizing specifically. Links with psychopathology were specific to the common EF factor, rather than updating- or shifting-specific EF.

Keywords Executive dysfunction · P factor · Internalizing · Externalizing · Stress · Rumination

Executive function (EF) processes enable us to respond flexibly to the environment and regulate our thoughts and behaviors, enabling self-directed behavior towards goals (e.g., Miyake and Friedman 2012). Meta-analytic evidence indicates that EF deficits are pervasive across prevalent psychopathologies and EF tasks (Snyder et al. 2015), leading to proposals that they may be transdiagnostic risk factors for psychopathology (e.g., Beauchaine and Zisner 2017; Goschke 2014; McTeague et al. 2016; Snyder et al. 2015). Four key limitations in the majority of prior research impede progress in rigorously testing this hypothesis.

First, most studies have used standard neuropsychological test approaches that confound different aspects of EF (e.g., Friedman and Miyake 2017), potentially leading to the seemingly undifferentiated nature of EF impairments across disorders. Second, the vast majority of research has investigated individual disorders or symptom dimensions without taking the pattern across disorders into account. However, the extensive comorbidity between psychopathologies suggests that the search for specific neuropsychological signatures associated with a singular psychiatric diagnosis may be misplaced (e.g., Kapur et al. 2012). An alternative approach that emphasizes broader latent dimensional psychopathology classification may thus yield a clearer and more parsimonious pattern. Third, most research has focused on adults, missing the key adolescent and emerging adult window for EF development and psychopathology risk. Finally, few studies have examined how (mediation) and for whom (moderation) EF and psychopathology are related. The current study seeks to address these limitations by linkages between latent dimensions of EF and latent psychopathology dimensions in a community sample of adolescents and emerging adults.

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Unity/Diversity Model of EF

EF is best characterized as separable but related cognitive processes, with both unique and shared individual differences, genetic influences, and neural substrates—a view shared across multiple models of EF (e.g., Baddeley and Repovs 2006; Diamond 2013; Shallice 2002). Here we focus on one such model, the unity/diversity model (Friedman and Miyake 2017; Friedman et al. 2008; Miyake et al. 2000). This model focuses on three aspects of EF. *Shifting* is defined as switching between task sets or response rules (e.g., classifying by shape or color). *Inhibition* is defined as suppressing or resisting prepotent (automatic) responses in order to make less automatic but task-relevant responses (e.g., naming the ink color rather than reading the word in the Stroop task). *Updating* is defined as monitoring for and adding task relevant information to working memory, and deleting no longer relevant information. These abilities correlate, suggesting that there is a *common EF* ability involved in all three aspects of EF; common EF is posited to be the ability to monitor for and maintain goal and context information and use that information to bias ongoing processing (Friedman and Miyake 2017).

Using latent variable bifactor modeling, each EF ability (e.g., updating) can be decomposed into what is common across all three EFs, or unity (common EF), and what is unique to that particular ability, or diversity. Two large, independent youth and adult samples have supported a model with a common EF factor and updating- and shifting-specific factors; common EF fully accounts for individual differences in inhibition (i.e., there is no inhibition-specific factor; Friedman et al. 2008, 2016; Ito et al. 2015). Most importantly for clinical research, the different components of EF identified in this model differentially predict individual differences in clinically relevant behaviors, with recent evidence finding that the common EF factor is the primary predictor, relating to behavioral disinhibition (Miyake and Friedman 2012), attention problems (Herd et al. 2014), and transdiagnostic psychopathology (Hatoum et al. 2017). Meta-analytic evidence suggests that effect sizes are generally similar across these core EF domains (Snyder et al. 2015), consistent with the theory that individuals with multiple forms of psychopathology have impairments in the unitary component of EF (i.e., common EF), although other explanations are also possible (e.g., multiple specific aspects of EF could be independently impaired).

Bifactor Models of Psychopathology and Links to EF

Importantly, psychopathology has also been shown to consist of both common and specific factors. In contrast to historical conceptualizations of psychological disorders as distinct, categorical conditions, it is now widely understood that co- and multi-morbidities between disorders are extensive, and that psychopathology is best conceptualized and modeled as continuous symptom dimensions. As a result, there has been a call to better understand the dimensional liabilities that are common to and influence the development of multiple psychopathologies (e.g., Kotov et al. 2017; Lahey et al. 2017).

There is a long history of modeling internalizing and externalizing dimensions of psychopathology liability, which has more recently been expanded to include a common factor (i.e., p factor; e.g., Caspi et al. 2014). This p factor model has replicated in multiple youth and adult samples (for review see Lahey et al. 2017). Beyond model fit, the critical question to ask of such models is whether they are useful, that is, whether they are related to theoretically and practically important risk factors and outcomes and thus hold promise to advance clinical science (Snyder and Hankin 2017). In the last few years, significant evidence has accumulated validating the p factor in relation to a wide variety of psychopathology risks (genetic, neural, psychosocial) and outcomes in youth and adults (for review see Carver et al. 2017; Lahey et al. 2017) and developmental continuity (Murray et al. 2016; Olino et al. 2018; Snyder et al. 2017c).

Such models thus hold promise for clarifying the many-to-many (multifinality and equifinality) patterns between risk factors, including executive dysfunction, and categorically defined disorders. Recent conceptual models have proposed that executive dysfunction is a risk factor for common psychopathology (p factor; e.g., Beauchaine and Zisner 2017; Hankin et al. 2016b; Snyder et al. 2015). Supporting these models, several recent studies in community samples have found that the p factor is associated with poorer performance on EF tasks, including working memory and a single EF composite in children (Huang-Pollock et al. 2016; Martel et al. 2017), working memory, flexibility, response inhibition, and updating tasks in adolescents (Bloemen et al. 2018; Castellanos-Ryan et al. 2016; Shanmugan et al. 2016; White et al. 2017), and working memory and shifting tasks in adults (Caspi et al. 2014). Some studies have found that specific internalizing (Bloemen et al. 2018; White et al. 2017) and externalizing dimensions (Huang-Pollock et al. 2016; Shanmugan et al. 2016; White et al. 2017) were also associated with poorer EF in youth. The p factor has also been linked to structure and function of prefrontal areas involved in EF in youth (Shanmugan et al. 2016; Snyder et al. 2017a).

Of note, these studies either used manifest EF variables or single, unitary EF factors[1], and thus did not directly test the

[1] Bloemen et al. (2018) also used a bifactor model, putatively of EF, but it included multiple components that fall outside the unity/diversity model, several of which are not generally considered components of EF (processing speed, pattern search, sustained attention), making the results difficult to interpret purely in terms of EF.

hypothesis that common psychopathology liability is linked to poorer common EF. Only one study to date has tested links between the bifactor model of psychopathology and the unity/diversity bifactor model of EF (Hatoum et al. 2017). In a large longitudinal twin sample, the common EF factor assessed at age 17 was predicted by the p factor assessed across childhood and adolescence, but only for male participants and only based on teacher, not parent, ratings. When internalizing and externalizing were modeled separately, internalizing related to poorer common EF across genders and raters, whereas externalizing related to *better* shifting-specific EF in some analyses, consistent with prior evidence that behavior problems are sometimes associated with better shifting-specific abilities (Hatoum et al. 2017; Herd et al. 2014). Thus, associations between the p factor and poorer EF are fairly consistently found across studies conducted to date, but results have varied somewhat, potentially due to differences in ages, reporters, and the assessment and modeling of psychopathology and EF across studies.

Mechanisms Linking EF and Psychopathology

Critically, there has been a lack of research investigating *why* EF is associated with psychopathology dimensions, a critical next step. Hankin et al. (2016b) posited that stress could mediate the risk between EF and expression of common and internalizing psychopathology. Consistent with this proposal, a previous study in a community sample of youth showed that poorer EF contributed to elevations in internalizing symptoms via higher levels of dependent stressful life events (i.e., stress generation) and subsequent rumination (Snyder and Hankin 2016). Dependent stressors are negative life events that are at least in part influenced by an individual's behavior (e.g., failing an exam, arguments with a friend). The stress generation model, which has received strong empirical support, posits that individual difference vulnerabilities related to psychopathology impair functioning and thus increase the risk for such dependent stressful life events (e.g., Conway et al. 2012; Hamilton and Alloy 2017; Hammen 1991; Liu 2013; Liu and Alloy 2010; Meyer and Curry 2017). It has been proposed that poor EF is one factor that can contribute to stress generation (Williams et al. 2009; Snyder and Hankin 2016). Specifically, poor EF may contribute to functional impairments that result in dependent stressful life events (e.g., failing an exam because of failure to plan or stay focused; interpersonal conflicts due to poor ability to stop counterproductive social behaviors). Importantly, we found that the link between EF task performance and internalizing symptoms increased with age from early adolescence to emerging adulthood, due to an increased association between EF and dependent stressful life events (Snyder and Hankin 2016). We speculated this effect may occur because adult caretakers compensate for younger adolescents' poor EF (e.g., reminders to complete homework), preventing poor EF from being translated into behaviors that lead to stressful life events (e.g., bad grades).

Dependent stressful life events in turn strongly predict rumination (e.g., Smith and Alloy 2009), a pattern of repetitive thought in response to an emotional state. Although originally proposed as a risk factor for depression, rumination has been found to demonstrate multifinality, predicting both internalizing and externalizing psychopathology symptoms and disorders (for meta-analysis see Aldao et al. 2010). Thus, it has been proposed that rumination acts as a transdiagnostic risk factor by amplifying current mood states and impairing problem solving and instrumental behavior (Nolen-Hoeksema and Watkins 2011). This predicts that rumination should be associated with the p factor, but this hypothesis has not previously been directly tested.

Mediation by stress generation and rumination could thus potentially explain why EF impairments are so broadly associated with psychopathology. In a previous study, dependent stressful life events predicted internalizing symptoms both directly and via increased rumination (Snyder and Hankin 2016). Both factors are transdiagnostic risks (e.g., Grant et al. 2014; Hankin et al. 2016a; Johnson et al. 2016), and stress predicts the p factor (Schaefer et al. 2017; Snyder et al. 2017b). Thus, these pathways may reflect associations between common EF and the p factor; however, as only links to internalizing symptoms were assessed, the extent to which these pathways are specific to internalizing vs. broadly transdiagnostic is unknown. In addition, since EF was assessed with a single composite variable (z-score across inhibition, updating, shifting and working memory tasks), the prior study did not test the specificity of EF-psychopathology links to common or specific components of EF.

Current Study

In sum, recent research using latent dimensional models of psychopathology has begun to disentangle the sources of the broad EF impairments observed across disorders. This research suggests that these associations may be mainly driven by shared psychopathology liability (p factor), consistent with the hypothesis that impaired common EF is a transdiagnostic risk factor for psychopathology. Most previous research on EF in relation to p factor psychopathology models has not employed EF models that can differentiate between EF components (but see Hatoum et al. 2017 for an exception). Last, the mediating mechanisms by which poorer EF might confer risk for common psychopathology liability remain speculative and have not been directly tested.

The current study therefore tests associations between the latent unity/diversity model of EF and latent dimensions of psychopathology liability in a community sample of youth during the key adolescent to emerging adult period of enhanced psychopathology risk (e.g., Merikangas et al. 2010) and continuing EF development (e.g., Friedman et al. 2016). We evaluate our hypotheses in a community sample, consistent with most previous studies of p factor models in youth (for a review, see Lahey et al. 2017); studying such community samples is valuable because there is strong evidence that psychopathology symptoms are best conceptualized and modeled as continuous in the population rather than by using arbitrary diagnostic thresholds and that even symptoms which do not meet diagnostic cut-offs cause significant distress and impairment (e.g., Clark et al. 2017).

The current study is cross-sectional, but enables preliminary tests of potential mediating mechanisms, which future longitudinal studies can then investigate. In doing so, we aim to clarify potential risk pathways between EF impairments and particular forms of psychopathology liability, and accelerate progress in understanding how EF impairments may contribute to co-occurrence across psychopathologies. Specifically, this study extends previous research demonstrating that poor EF predicted anxiety and depression symptoms via stress generation and subsequent rumination, and that these effects were stronger in older youth (Snyder and Hankin 2016). First, we extend the previous study by directly testing the hypothesis that common EF (rather than shifting or updating-specific components) drives associations with a latent internalizing liability factor. Second, we extend this model to test associations with the latent dimensions from the bifactor model of psychopathology to determine if this pathway is specific to internalizing liability, or instead is a broad pathway predicting the p factor.

We hypothesized that the seemingly broad deficits on EF tasks associated with multiple forms of psychopathology are best explained by poorer common EF associated with common psychopathology liability (p factor; Hatoum et al. 2017). Given mixed and limited prior evidence, we did not make a priori hypotheses regarding the externalizing- or internalizing- specific liability factors or the shifting and/or updating-specific EF factors, but conduct exploratory analyses with these factors. If, as predicted, the common EF factor was associated with the p factor, we hypothesized that this association would be at least partially mediated by the indirect path through dependent stressful life events and rumination, and that the EF-stress path would be stronger for older youth, as in Snyder & Hankin (2016). These hypotheses were pre-registered[2].

[2] https://osf.io/hfcra/?view_only=e3afaa7ba6ae44cdb9f05c0549f6a11f.

Method

Participants

Participants were 292 13–22-year-old youth (mean age = 16.20 years, SD = 2.35; 56% female) recruited from the greater Denver metro area through direct mail to ZIP codes selected to maximize racial and economic diversity and from an ongoing longitudinal study. Interested families contacted the lab and were screened for age range eligibility and English fluency (including consenting parent of minors). Sample size was determined by a priori power analyses in the grant proposal. Participants identified as 70% white, 11% more than one race, 9% African American, 4% American Indian/Native Alaskan, 2% Asian, and 4% other or declined to answer; 19% identified as Hispanic/Latino.

Procedure

As part of a larger research protocol, youth participated in one 5-h or two 2.5-h laboratory visits, with breaks to reduce fatigue. Participants gave written informed consent (18–22) or assent with parental consent (13–17). Youth completed three EF tasks each assessing updating, shifting, and inhibition. Participants were asked to complete self-report questionnaires online from home before their visit; if they had not finished the questionnaires, they completed them at the end of their first lab visit.[3] All study procedures were approved by the university Institutional Review Board.

Measures

EF Tasks

EF tasks were from Friedman et al. (2016), with the exception of the stop signal task, which was from Chatham et al. (2012). Tasks are briefly described here, and additional details of task methods and data processing are provided in Supplemental Materials. The tasks from Friedman et al. (2016), and a similar stop signal task, have been found to have good internal and test–retest reliability, and convergent validity including significant factor loadings on both common EF and, for updating and shifting tasks, the updating-specific and shifting-specific factors (Friedman et al. 2016). The stop signal task has been validated in reference to mathematical models and neural indices of response inhibition (Chatham et al. 2012).

[3] There were no significant differences on any study measure between those that completed questionnaires prior to or at the end of their visit.

Updating For all updating tasks, the performance measure is the proportion of correct responses across all trials.

Keep Track On each trial, participants are shown the names of 2–5 target categories (e.g., animals, colors), which remain on the bottom of the screen throughout the trial, in which 15–25 words are serially presented. Participants are instructed to recall the last exemplar seen in the target categories at the end of each trial. Since multiple exemplars from each category are presented in each trial, this requires updating which exemplars to remember.

Letter Memory Letters are presented serially in the center of the screen, with 9–13 letters in each trial, and participants say out loud the last three letters, adding the most recent letter and dropping the fourth letter back. Each letter triad was scored as correct if participants reported all three letters correctly in order.

Spatial 2-Back Twelve squares scattered across the screen become dark one at a time, and participants press a button to indicate if the dark square is the same as the one two trials earlier; 30% of "no" trials are "lures": flashes that match the square from three flashes back.

Shifting For all tasks, participants first practiced a block of each sub-task separately, followed by mixed-task blocks. For all tasks, the cue-to-stimulus and response-to-stimulus intervals were 350 ms, and 50% of trials require a task switch. The performance measure for all shifting tasks is the switch cost: the difference in mean RT between correct task switch trials and task repeat trials in the mixed blocks. Responses were recorded using a ms accurate button box, using the same two buttons for both subtasks within each task. Participants were instructed to respond as quickly as possible without making mistakes, which were indicated by an error beep.

Number–Letter A number–letter pair (e.g. 7G) is presented on each trial in the top or bottom squares of a four-square grid. Before the pair appears, the border of one square turns dark, cueing the task. When it is at the top, participants indicate whether the number is odd or even. When it is at the bottom, they indicate if the letter is a vowel or consonant.

Color–Shape On each trial a cue (C for color or S for shape) is presented above a colored rectangle (green or red) with a shape in it (circle or triangle), and participants indicate the color or shape.

Category Switch On each trial a cue symbol above a word indicates whether the word (from a list of 16 pre-familiarized words), should be categorized as living vs. non-living or as smaller vs. larger than a soccer ball.

Inhibition *Antisaccade* On each trial, a cue flashes on one side of the screen (200–250 ms), followed by a target (a box containing a number) on the other side of the screen that is masked after 150 ms. Thus, to identify the number, participants must inhibit the automatic tendency to saccade to the cue and instead immediately look in the opposite direction. The performance measure is accuracy.

Stop Signal Participants press a button to indicate if an arrow is pointing left or right as quickly as possible. A square signaling participants to not respond is presented on 25% of trials after a stop signal delay (100–300 ms). The performance measure is the stop-signal RT (SSRT, the average time needed to stop a response), calculated using the integration method (Logan and Cowan 1984).

Stroop Participants name the color of each stimulus for blocks of neutral trials (asterisks in color ink) and incongruent trials (color words in a different color ink), with RT measured by ms-accurate voice-onset microphone. The performance measure is interference (mean incongruent RT–mean neutral RT).

Questionnaires

Children's Depression Inventory (CDI; Kovacs 1985)

The CDI assesses depressive symptoms in children and adolescents, and has been shown to have good reliability and convergent validity (Klein et al. 2005; Smucker et al. 1986). Internal consistency in the current sample was high ($\alpha = 0.895$).

Penn State Worry Questionnaire for Children (PSWQ-C; Chorpita et al. 1997)

The PSWQ-C is adapted from the adult version of PSWQ for use with children and adolescents and has been shown to have good reliability and convergent and divergent validity (Chorpita et al. 1997). Internal consistency in the current sample was high ($\alpha = 0.937$).

Manifest Anxiety Scale for Children (MASC; March et al. 1997)

The MASC assesses anxious symptoms in children and adolescents, with subscales for Physical Symptoms, Social Anxiety, and Separation Anxiety. (In addition, there is a Harm Avoidance subscale, which was not used in the current study given evidence that it does not assess anxiety but rather risk aversion; Snyder et al. 2015). The MASC has been found to have good reliability and convergent and discriminant validity (March et al. 1997; Muris et al. 2002). Internal consistencies in the current study were high for Physical Symptoms ($\alpha = 0.897$) and Social Anxiety ($\alpha = 0.894$), and moderate for Separation Anxiety ($\alpha = 0.694$).

Child Behavior Checklist Youth Self-Report (YSR, Achenbach and Rescorla 2001)

The current study used the Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) *DSM*-oriented scales, which have been found to have good reliability and validity in adolescents (Achenbach and Rescorla 2001). Internal consistencies in the current sample were moderate (ODD $\alpha = 0.658$, CD $\alpha = 0.599$).

Multisite Multimodal Treatment Study of Children with ADHD (MTA) SNAP-IV (SNAP; Swanson et al. 2001)

The SNAP covers *DSM* criteria for ADHD, with inattention and hyperactivity subscales, and has shown good reliability and validity (Bussing et al. 2008). For the current study, the items were re-worded from parent report to self-report. Internal consistencies were high (Inattention $\alpha = 0.884$, Hyperactivity $\alpha = 0.849$).

Strengths and Difficulties Questionnaire (SDQ; Goodman 2001)

The SDQ is a frequently used brief measure of youth psychopathology, which has been shown to have good reliability and validity (Goodman 2001). The prosocial behavior (reverse coded) subscale was included as an additional indicator of externalizing psychopathology. Internal consistency in the current sample was moderate ($\alpha = 0.644$). Other subscales from the SDQ were not included here because their content was more extensively covered by the other measures in the study.

Adolescent Life Events Questionnaire Revised (ALEQ-R, Fassett-Carman et al. 2018)

The ALEQ (Hankin and Abramson 2002) self- report instrument assesses a broad range of negative life events typically experienced by youth, occurring in the past 6 months. The ALEQ was revised for the current study to include broader coverage of stressful life events. For all items, participants rated how often such events occurred from 1 (*never*) to 5 (*always*). Since stress generation was tested, the current study used only dependent stressful life events items (e.g., getting bad grades, arguments or problems with friends; see Fassett-Carman et al. 2018 for coding methods and reliability).

Children's Response Styles Questionnaire (CRSQ; Abela et al. 2000)

CRSQ rumination subscale assesses rumination in response to sad mood in youth. It has been shown to have good reliability and validity (e.g., Abela and Hankin 2011; Abela et al. 2012). Internal consistency in the current sample was high ($\alpha = 0.930$).

Analysis Approach

Analyses use confirmatory factors analysis (CFA), and structural equation modeling (SEM), implemented using Mplus Version 8 using full information maximum likelihood (FIML) to handle missing data (Muthén and Muthén 2017). For all models, because the χ^2 is sensitive to sample size, fit criteria were as follows: root mean square error of approximation (RMSEA) < 0.06 good, < 0.08 acceptable; comparative fit index (CFI) > 0.95 good, > 0.90 acceptable; and standardized root mean square residual (SRMR) < 0.08 good, acceptable 0.10 (Hu and Bentler 1999). Significance of parameters was assessed with *z*-tests (i.e., the parameter divided by its standard error). Additional details on data cleaning and calculation of measures are described in Supplemental Materials.

EF Measurement Model

The EF model is based on previous research (e.g., Friedman et al. 2016), in which there is a common EF latent variable on which all 9 tasks load as well as updating-specific and shifting-specific latent variables on which the updating and shifting tasks, respectively, also load. Research has found that common EF variance is isomorphic with the inhibition latent variable, so that there is no inhibiting-specific variance (for discussion see Friedman and Miyake 2017). Because the common EF factor captures the variance common to all three EFs, the updating-specific and shifting-specific factors capture the variance that is unique to updating and shifting, respectively. Hence, they are constrained to be uncorrelated with the common EF factor and with each other.

Psychopathology Measurement Models

Two psychopathology liability models were tested. The first model, designed to extend earlier tests of this mediation model with anxiety and depression symptoms (Snyder and Hankin 2016), was a one-factor internalizing model, with the CDI, PSWQ and MASC subscales (physical symptoms, social anxiety and separation anxiety) as indicators. The second, to test whether the mediation model is transdiagnostic beyond internalizing, used a bifactor (aka p factor) model, based on extensive previous research as described in in the introduction. All psychopathology measures were loaded onto a common factor (p factor) as well as their specific factor that represents the unique variance associated with internalizing (CDI depression, PSWQ worry, and MASC physical symptoms, social anxiety and separation anxiety) and

externalizing (YSR CD and ODD, SNAP hyperactivity and inattention, SDQ low prosocial behavior) psychopathology not accounted for by the p factor. As for the EF model, factors were constrained not to correlate because what is shared between factors is already captured by the common factor.

SEMs

Dependent stressful life events (ALEQ-R) and rumination (CRSQ) were modeled as manifest variables. Because the ALEQ assesses the frequency of different stressful life events, rather than multiple indicators of a particular construct, it would not be appropriate to model it as a latent variable (which assumes indicators are *caused* by the latent construct). Although the CRSQ could be modeled as a latent factor, this factor correlated $r = .99$ with the manifest CRSQ total score, as might be expected given the very high internal consistency of this measure ($\alpha = 0.93$); given that the manifest score thus provides nearly identical information to the factor, we use the manifest score to minimize model complexity.

Because age moderation was hypothesized based on previous research (Snyder and Hankin 2016), all models included EF x age interaction terms estimated with TYPE = RANDOM and numerical integration, which provided FIML estimates of the interactions (Muthén and Muthén 2017). Loop plots with bootstrapped (10,000 iterations) 95% confidence intervals were generated to examine the significance of effects across the age range. First, total effects models were tested with EF factors predicting each psychopathology factor. All three EF factors (common EF, updating-specific and shifting-specific) were initially included, but as only the common EF × age interaction was a significant predictor in any model, paths from updating-specific and shifting-specific were dropped in further models (see Results). Second, path models with dependent stressful life events and rumination as sequential mediators and age as a moderator of the EF-stress link were tested consistent with previous research (Snyder and Hankin 2016). Direct links from EF to rumination were also initially tested; replicating the previous study (Snyder and Hankin 2016), they were not significant and thus eliminated.

Results

Descriptive statistics are provided in Table 1.

Measurement Models

EF task model

The model had good fit (CFI = 0.99, RMSEA = 0.019, SRMR = .036; χ^2 (24) = 23.24, $p = .33$). All tasks loaded significantly on their factors, with the exception of the Category Switch task, which loaded strongly on the shifting-specific factor but only marginally significantly on the common EF factor (Supplemental Materials Table S1). Replicating previous research with this unity/diversity model, an alternative model including an inhibition-specific factor found no significant variance for the specific factor, indicating that covariance among the inhibition tasks is fully accounted for by the common EF factor; therefore, no inhibition-specific factor was included.

Internalizing Model

The model had good fit (CFI = 0.99, RMSEA = 0.054, SRMR = .017; χ^2 (4) = 7.41, $p = .17$), and all indicators loaded significantly (Supplemental Materials Table S2). Based on the modification index, a negative residual correlation was included between CDI and MASC separation anxiety ($r = -.33$), most likely due to opposite age trends for depression and separation anxiety; without this residual, model fit was significantly worse ($\Delta\chi^2$ (1) = 16.93, $p < .001$) and model fit was not acceptable by RMSEA (0.115), so the modification was retained for all structural models. However, all results remained nearly identical when this residual was omitted (Supplemental Materials Table S3).

Bifactor (p factor) Model

The model had good fit (CFI = 0.97, RMSEA = 0.075, SRMR = .042; χ^2 (23) = 61.00, $p < .001$), and all indicators loaded significantly on their factors with the exception of SNAP inattention, which loaded marginally on the externalizing-specific factor but strongly on the p factor (Supplemental Materials Table S2). Because SNAP hyperactivity and inattention measure two symptom clusters of the same disorder (ADHD), a residual correlation was included between them ($r = .44$); without this residual correlation, the model did not converge. Based on modification indices, a residual correlation was included between SNAP hyperactivity and MASC social anxiety ($r = -.28$), which may reflect opposite behavioral patterns (over-exuberant vs. withdrawn); without this correlation, model fit was significantly worse ($\Delta\chi^2$ (1) = 19.03, $p < .001$) and model fit was not acceptable by RMSEA (0.090), so the modification was retained for all structural models. However, all results remained nearly identical when this residual was omitted (Supplemental Materials Table S4).

Table 1 Descriptive statistics

Measure	Mean	SD	*n*	Skewness	Kurtosis
PSWQ-C	30.43	9.52	292	0.58	− 0.44
MASC physical symptoms	10.42	7.25	293	0.64	− 0.23
MASC social anxiety	11.60	6.64	293	0.19	− 0.68
MASC separation anxiety	5.90	4.08	293	0.81	0.50
CDI	8.01	7.53	293	1.47	2.39
CBCL-YSR ODD	2.56	1.97	289	0.79	0.23
CBCL-YSR CD	3.30	2.43	289	0.79	0.22
SDQ prosocial (rev.)	2.08	1.77	291	0.77	0.29
SNAP ADHD inattention	7.08	5.56	289	0.82	0.26
SNAP ADHD hyperactivity	5.33	4.98	289	1.59	3.42
ALEQ dependent stress frequency	13.28	10.41	293	0.43	− 0.26
CRSQ rumination	25.72	9.41	293	0.73	− 0.087
Stroop blocked interference (ms)	155.29	85.48	288	− 0.60	0.40
Antisaccade proportion correct	0.68	0.16	292	− 0.07	− 0.34
Stop signal SSRT (ms)	272.33	136.81	281	− 0.45	− 0.54
Category switch cost (ms)	272.25	159.16	268	0.04	0.37
Color–shape switch cost (ms)	242.33	158.16	287	− 0.36	0.05
Number–letter switch cost (ms)	391.12	212.26	290	− 0.58	0.55
Keep track proportion correct	0.70	0.11	292	− 0.08	0.00
Letter memory proportion correct	0.86	0.13	292	− 0.25	0.13
Spatial 2-back proportion correct	0.79	0.10	291	0.13	− 0.25

In the full mediation model, MASC separation anxiety loaded only on the internalizing-specific factor (the p factor loading was estimated as negative and eliminated); a measurement model version with this modification and the same residual correlations had good to acceptable fit (CFI = 0.96, RMSEA = 0.080, SRMR = .063; χ^2 (24) = 68.72, $p < .001$). Results from a model retaining the negative MASC separation anxiety loading on the p factor had similar results (Supplemental Materials Table S5).

One-Factor Internalizing Model Analyses

Age (centered) and gender (coded − 1 female, 1 male) were included as covariates in all path regressions predicting each mediator and latent psychopathology factor.

Total Effects Model

In the initial model with all three EF factors, only the common EF factor significantly interacted with age to predict the internalizing factor (updating-specific and shifting-specific main effects and age interactions *p*s > 0.5); thus, only paths involving common EF were retained in subsequent models. There was a significant common EF x age interaction, such that common EF was more strongly negatively associated with internalizing psychopathology in older participants (β = − 0.244, $p < .001$; Table 2; Fig. 1A). A loop plot of the interaction demonstrated a significant negative relation between common EF and internalizing in older youth ages 18+ (Fig. 1a).

Moderated Mediation Models

The moderated mediation models included the common EF factor as the predictor, ALEQ dependent stressful life events and rumination as the mediators, and the internalizing factor as the outcome, with age moderating all effects of EF (Table 2; Fig. 1c). There was a significant age × common EF interaction predicting dependent stress, such that older participants with poorer EF reported experiencing more dependent stressful life events (β = − 0.199, $p < .001$). A loop plot of the interaction demonstrated a significant negative relation between common EF and dependent stressful life events in older youth ages 18+ (Fig. 1d). Participants reporting higher levels of dependent stressful life events reported higher levels of rumination (β = 0.548, $p < .001$). Both higher dependent stressful life events (β = 0.338, $p < .001$) and rumination (β = 0.519, $p < .001$) were associated with the internalizing factor. There were no significant age interactions for any of these effects, indicating that these associations were equally strong across younger and older youth (*p*s > 0.09; Supplemental Materials Table S6). The direct effect of common EF x age on internalizing remained marginally significant (β = − 0.085, $p = .053$), suggesting partial mediation. Loop plots of the conditional indirect effects

Table 2 Parameter estimates for one-factor internalizing model analyses with age moderation

Outcome variable	Predictor	Unstandardized estimates				Standardized estimates			
		b	SE	*z*	*p*	*β*	SE	*z*	*p*
Total effects model									
Internalizing factor	Common EF × age	− 0.156	0.042	− 3.69	< .001**	− 0.244	0.067	− 3.82	< .001**
	Common EF	− 0.055	0.120	− 0.46	0.644	− 0.037	0.080	− 0.46	0.643
	Age	− 0.050	0.043	− 1.16	0.245	− 0.078	0.067	− 1.17	0.244
	Gender	− 0.469	0.093	− 5.05	< .001**	− 0.314	0.052	− 3.82	< .001**
Moderated mediation model									
ALEQ dep. stressors	Common EF × age	− 0.113	0.031	− 3.64	< .001**	− 0.199	0.054	− 3.70	< .001**
	Common EF	− 0.007	0.101	− 0.07	0.948	− 0.005	0.076	− 0.062	0.950
	Age	− 0.039	0.034	− 1.12	0.263	− 0.068	0.060	− 1.13	0.261
	Gender	− 0.216	0.077	− 2.82	0.005*	− 0.162	0.056	− 3.70	< .001**
CRSQ rumination	ALEQ dep. stressors	0.298	0.028	10.46	< .001**	0.548	0.045	12.29	< .001**
	Age	0.009	0.014	0.68	0.496	0.030	0.045	0.68	0.497
	Gender	− 0.072	0.036	− 2.01	0.044*	− 0.099	0.049	− 2.03	.043*
Internalizing factor	ALEQ dep. stressors	0.388	0.061	6.32	< .001**	0.339	0.048	7.08	< .001**
	CSRQ rumination	1.093	0.131	8.37	< .001**	0.519	0.045	11.50	< .001**
	Common EF × Age	− 0.055	0.028	− 1.97	0.049*	− 0.085	0.044	− 1.94	0.053^
	Common EF	− 0.118	0.081	− 1.46	0.143	− 0.078	0.053	− 1.47	0.141
	age	− 0.012	0.028	− 0.42	0.675	− 0.018	0.043	− 0.42	0.677
	Gender	− 0.250	0.063	− 3.98	< .001**	− 0.163	0.041	− 4.01	< .001**

Negative values for gender indicate lower values for male than female participants

**$p < .001$, *$p < .05$

demonstrated significant indirect effects of common EF on the internalizing factor for older youth ages 18 + via stressful life events and rumination and stressful life events alone (Fig. 1e and f).

Bifactor Model Analyses

Age (centered) and gender (coded − 1 female, 1 male) were included as covariates in all path regressions for all analyses.

Total Effects Model

An initial model did not show any significant updating-specific and shifting-specific main effects or age interactions (*p*s > 0.3); thus, only paths involving common EF were retained in subsequent models. There was a significant common EF × age interaction, such that common EF was more strongly negatively associated with higher common psychopathology (p factor) in older participants ($\beta = -0.241$, $p < .001$), with a trend in the same direction for the internalizing-specific factor ($\beta = -0.139$, $p = .074$; Table 3; Fig. 2a). Loop plots of the interactions demonstrated significant negative relations between common EF and the p factor and internalizing-specific factor for youth age 19+ (Fig. 2b and c).

Moderated Mediation Models

The moderated mediation models included the common EF factor as the predictor, ALEQ dependent stressful life events and rumination as the mediators, and the p, internalizing-specific and externalizing-specific factors as the outcomes, with age moderating all effects of EF (Table 3; Fig. 2d).[4] As in the one-factor internalizing model, there was a significant age x common EF interaction, such that older participants with poorer EF reported experiencing more dependent stressful life events ($\beta = -0.202$, $p = .003$). Participants reporting higher levels of dependent stressful life events reported higher levels

[4] It has also been suggested that EF and stress may interact, such that individuals with poorer EF engage in a more maladaptive patterns of coping with stress, increasing psychopathology risk (e.g., Compas et al. 2009). We thus tested alternative moderation models, with dependent stressful life events, common EF, and their interaction predicting (1) the internalizing factor in the internalizing only model, and (2) all three psychopathology factors in the bifactor model. We did not find any significant interactions between dependent stressful life events and common EF in association with any of the psychopathology factors (*p*s > 0.4), and there were no significant interactions for either younger or older youth. This does not preclude the possibility that EF may affect specific stress coping mechanisms (e.g., reappraisal, e.g., Cohen and Mor 2017), which is an important area of continuing research.

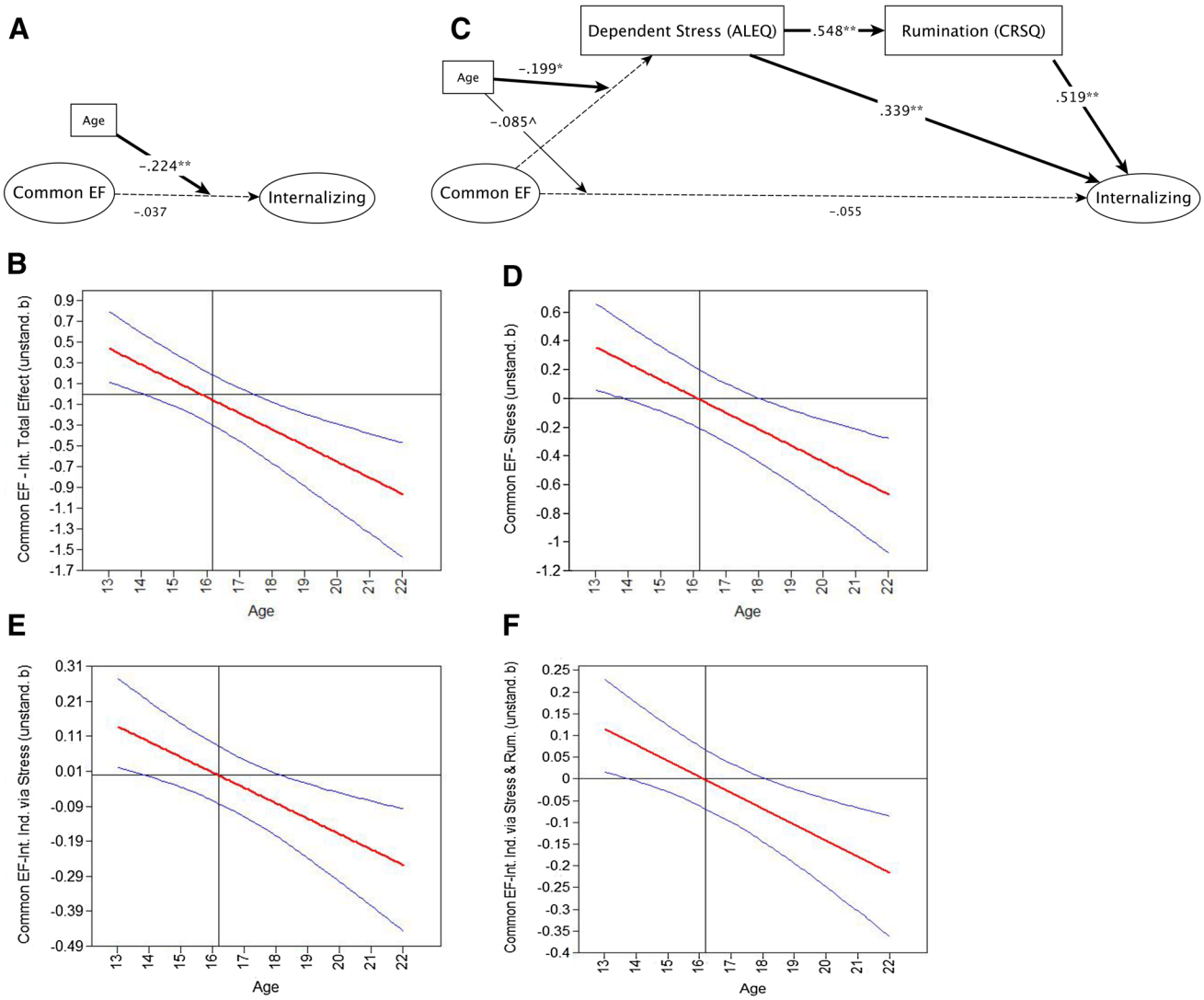

Fig. 1 Total effects and mediation path models predicting internalizing liability, moderated by age. Path model diagrams (**a, c**) show standardized path coefficients. Graphs (**b, d–f**) show unstandardized regression coefficients (inner line) with bootstrapped bias-corrected confidence intervals (outer lines) showing the age range over which the effect was significant. (Vertical axis as at the mean age.) In the total effect model, there was a significant age x common executive function (EF) interaction predicting latent internalizing liability (**a**), with poorer common EF significantly associated with higher internalizing for youth 18+ (**b**). In the mediation model, there was a significant age x common EF interaction predicting dependent stress (**c**) with poorer common EF significantly associated was higher stress for youth 18+ (**d**). For all youth, dependent stress was associated with higher rumination, and both stress and rumination were associated with higher internalizing (**c**). Conditional indirect effects of common EF on the internalizing factor via stress alone (**e**) and stress then rumination (**f**) where significant for youth ages 18+. *Ind*. indirect effect, *Unstand*. unstandardized, *Int*. internalizing factor, *Rum*. rumination $^{**}p < .001$, $^{*}p < .05$, $^{\wedge}p < .10$

of rumination (β = 0.547, $p < .001$). Dependent stressful life events were associated with all three psychopathology factors (p factor: β = 0.323, $p < .001$; internalizing-specific factor: β = 0.232, $p = .004$; externalizing-specific factor: β = 0.425, $p = .002$) and rumination was associated with higher levels of the p factor (β = 0.614, $p < .001$), and internalizing-specific factor (β = 0.190, $p = .013$), but lower levels of the externalizing-specific factor (β = − 0.471, $p = .049$). There were no significant age interactions for any of these effects, indicating that these associations were equally strong across younger and older youth ($ps > 0.10$; Supplemental Materials Table S6). Although all paths were significant for older youth, meeting the traditional criteria for mediation (Baron and Kenny 1986), loop plots of the conditional indirect effects approached but did not reach significance at any age for the p factor or internalizing specific factor (Supplemental Materials Figure S1 A–D).

Table 3 Parameter estimates for bifactor model analyses with age moderation

Outcome variable	Predictor	Unstandardized estimates				Standardized estimates			
		b	SE	*z*	*p*	*β*	SE	*z*	*p*
Total effects model									
p factor	Common EF × age	− 0.093	0.033	− 2.83	0.005*	− 0.241	0.079	− 3.03	0.002*
	Common EF	0.003	0.080	0.04	0.966	0.004	0.089	0.04	0.966
	Age	0.025	0.030	0.83	0.408	0.065	0.078	0.83	0.407
	Gender	− 0.133	0.066	− 2.02	0.043*	− 0.147	0.071	− 2.07	0.038*
Internalizing factor	Common EF × age	− 0.058	0.033	− 1.76	0.079^	− 0.139	0.078	− 1.79	0.074^
	Common EF	− 0.071	0.083	− 0.86	0.393	− 0.072	0.085	− 0.86	0.393
	Age	− 0.076	0.030	− 2.51	0.012*	− 0.183	0.071	− 2.58	0.010*
	Gender	− 0.310	0.069	− 4.51	< .001**	− 0.317	0.061	− 5.19	< .001**
Externalizing factor	Common EF × Age	0.090	0.068	1.33	0.183	0.140	0.105	1.33	0.183
	Common EF	0.062	0.160	0.39	0.699	0.041	0.105	0.39	0.697
	Age	− 0.117	0.058	− 2.02	0.043*	− 0.181	0.090	− 2.02	0.043*
	Gender	0.077	0.136	0.56	0.573	0.051	0.089	0.57	0.570
Moderated mediation model									
ALEQ dep. stressors	Common EF × age	− 0.115	0.038	− 2.99	0.003*	− 0.202	0.066	− 3.05	0.002*
	Common EF	− 0.008	0.100	− 0.075	0.940	− 0.006	0.075	− 0.08	0.940
	Age	− 0.039	0.037	− 1.06	0.291	− 0.068	0.064	− 1.06	0.290
	Gender	− 0.217	0.077	− 2.84	0.005*	− 0.163	0.056	− 2.91	0.004*
CRSQ rumination	ALEQ dep. stressors	0.298	0.027	11.18	< .001**	0.547	0.041	13.23	< .001**
	Age	0.009	0.015	0.61	0.541	0.030	0.048	0.61	0.541
	Gender	− 0.072	0.036	− 2.03	0.042*	− 0.100	0.049	− 2.04	0.041*
p factor	ALEQ dep. stressors	0.334	0.072	4.65	< .001**	0.323	0.057	5.62	< .001**
	CSRQ rumination	1.17	0.119	9.78	< .001**	0.614	0.059	10.48	< .001**
	Common EF × age	− 0.037	0.034	− 1.09	0.275	− 0.063	0.057	− 1.11	0.266
	Common EF	− 0.067	0.085	− 0.79	0.432	− 0.048	0.060	− 0.79	0.425
	Age	0.060	0.033	1.81	0.070^	0.102	0.077	1.32	0.138
	Gender	− 0.084	0.074	− 1.14	0.255	− 0.061	0.042	− 1.17	0.241
Internalizing-specific	ALEQ dep. stressors	0.148	0.069	2.15	0.031*	0.232	0.081	2.84	0.004*
	CSRQ rumination	0.223	0.108	2.06	0.040*	0.190	0.077	2.47	0.013*
	Common EF × age	− 0.024	0.029	− 0.84	0.402	− 0.067	0.077	− 0.87	0.386
	Common EF	− 0.086	0.070	− 1.23	0.218	− 0.102	0.081	− 1.25	0.211
	Age	− 0.063	0.028	− 2.25	0.024*	− 0.173	0.062	− 2.56	0.010*
	Gender	− 0.196	0.067	− 2.93	0.003*	− 0.231	0.062	− 3.74	< .001**
Externalizing-specific	ALEQ dep. stressors	0.516	0.174	2.97	0.003*	0.425	0.136	3.12	0.002*
	CSRQ rumination	− 1.05	0.536	− 1.99	0.046*	− 0.471	0.239	− 1.97	0.049*
	Common EF × age	0.115	0.067	1.72	0.086^	0.166	0.097	1.72	0.085^
	Common EF	0.141	0.160	0.88	0.377	0.087	0.097	0.90	0.367
	Age	− 0.105	0.072	− 1.47	0.141	− 0.153	0.106	− 1.44	0.149
	Gender	0.201	0.139	1.45	0.148	0.124	0.082	1.52	0.129

Negative values for gender indicate lower values for male than female participants

**$p < .001$, *$p < .05$, ^$p < .10$

Discussion

The current study sought to better understand possible risk pathways between poorer EF and internalizing and common psychopathology (p factor) liability. For older youth, poorer common EF, but not the updating or shifting-specific factors, was associated with higher internalizing liability in a one-factor internalizing model, and higher common psychopathology (p factor) in the bifactor model (with a trend in the same direction for the internalizing-specific factor). Consistent with and extending a process

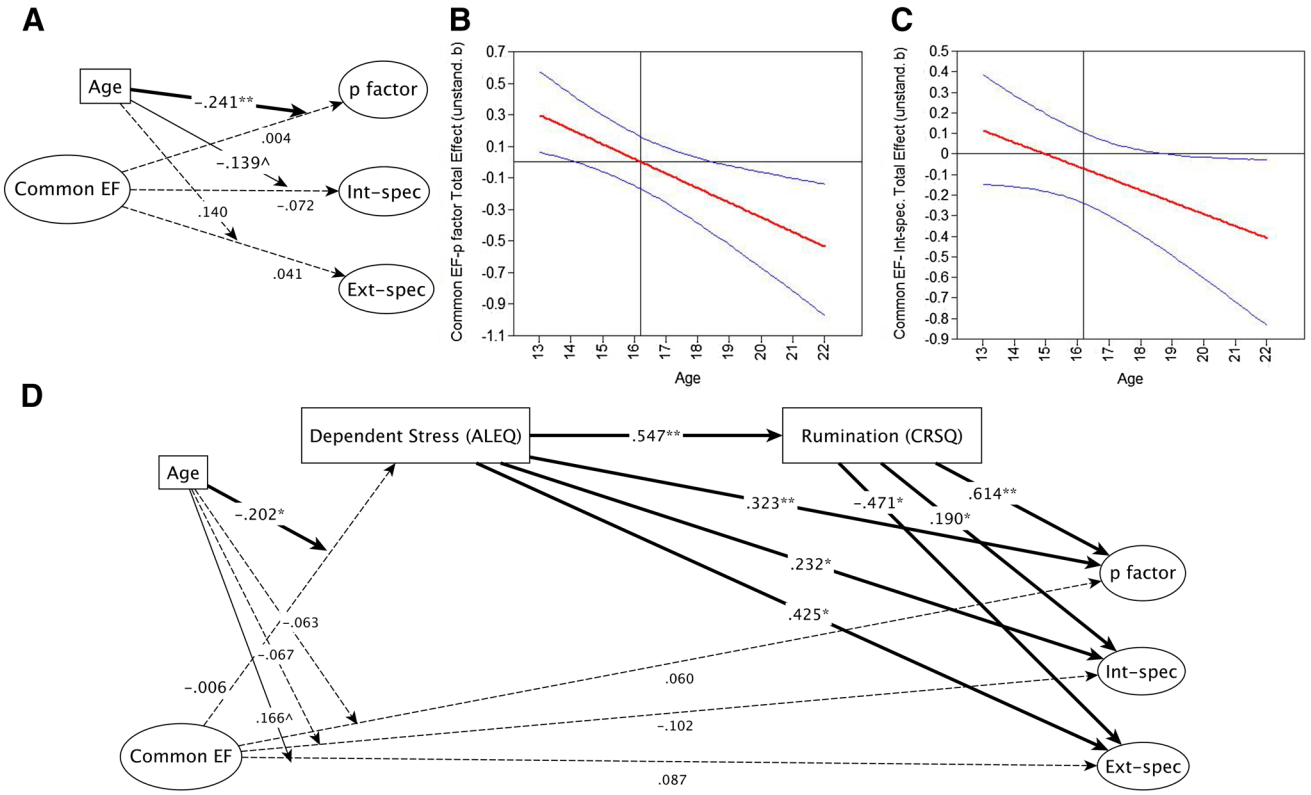

Fig. 2 Total effects and mediation path models predicting the bifactor psychopathology model, moderated by age. Path model diagrams (**a, d**) show standardized path coefficients. Graphs (**b, c**) show unstandardized regression coefficients (inner line) with bootstrapped bias-corrected confidence intervals (outer lines) showing the age range over which the effect was significant. (Vertical axis as at the mean age). In the total effect model, there was a significant age × common executive function (EF) interaction predicting the p factor, and a marginal interaction predicting the internalizing-specific factor (**a**), with poorer common EF significantly associated was higher levels of the p factor and internalizing-specific factor for youth 19+ (**b, c**). In the moderated mediation model (**d**), there was a significant age x common EF interaction predicting dependent stress, with poorer common EF significantly associated was higher stress for youth 18+ (see Fig. 1d). For all youth, dependent stress was associated with higher rumination. Stress was associated with higher levels of all three psychopathology liability factors, whereas rumination was associated with higher levels of p factor and internalizing-specific factor but lower levels of the externalizing-specific factor. Conditional indirect effects of common EF on the p factor and internalizing-specific factors via stress alone and stress then rumination approached but did not reach significance in older youth (Supplemental Materials Fig. S1). *Ind*. indirect effect, *Unstand*. unstandardized, *Int. Spec*. internalizing-specific factor, *Ext. Spec*. Externalizing-specific factor. $^{**}p < .001$, $^{*}p < .05$, $^{\wedge}p < .10$

model previously supported for internalizing symptoms, in which EF-internalizing links were mediated by dependent stressful life events and rumination in older youth (Snyder and Hankin 2016), poorer common EF was also associated with more dependent stressful life events in older youth, which in turn were associated with higher rumination for all youth. For all youth, rumination and dependent stressful life events were associated with higher internalizing liability in a one-factor internalizing model, and with higher levels of both the p factor and internalizing-specific factor in the bifactor model. It should be noted, however, that although all paths were significant for older youth, the conditional indirect effects of common EF on the p factor and internalizing-specific factor did not reach significance. Overall, the results suggest that the many-to-many relations between different forms of psychopathology and measures of different aspects of EF may be more parsimoniously explained by a link between common EF and both common psychopathology liability (p factor) and internalizing-specific liability in older youth, and suggest that stress and rumination may serve as mediators of these relations.

Specificity to EF Dimensions

Links with psychopathology liability were specific to the common EF factor, rather than updating- or shifting-specific EF. This is consistent with the one previous study to test associations between the p factor and the unity/diversity EF model (Hatoum et al. 2017), and most previous studies with more specific symptom dimensions (e.g., Gustavson et al. 2017; Herd et al. 2014), as well as the broad impairments

across different EF domains found in meta-analyses for most disorders (Snyder et al. 2015). However, one study found that depression symptoms were associated with lower common EF cross-sectionally, but prospectively predicted lower updating-specific EF, suggesting that concurrent and longitudinal associations may differ in some cases (Friedman et al. 2018).

Why might the common EF factor be particularly linked to psychopathology liability? It is thought to capture the ability to actively maintain and manage goals and use them to control ongoing processing, a demand shared by all EF tasks (Friedman and Miyake 2017). Such goal-directed behavior is arguably also essential for all non-automatic behavior, including planning and prioritizing tasks, resisting distraction and temptation, and making decisions. On the other hand, updating- and shifting-specific demands are likely to occur only intermittently in daily life (e.g., when there is a need to switch between activities or stop perseverating on a particular thought or behavior). Thus, poor common EF may be particularly likely to impair functioning, including in ways that increase stress. This relation is likely to be transactional, with stress also leading to impairments in EF (e.g., Shields et al. 2016). Future longitudinal research is needed to investigate these possibilities, although as we discuss further below, the relatively stable individual differences in both EF and psychopathology dimensions will make determining temporal precedence challenging.

Age Moderation

As in the prior study with a unitary EF composite score (Snyder and Hankin 2016), common EF in the current study significantly interacted with age, such that poorer common EF was only associated with dependent stressful life events in older adolescents/emerging adults. We speculate that this may be due to the role adults (e.g., parents and teachers) play in compensating for younger adolescents' poor EF, thus buffering against stress generation. This may be particularly true for common EF, given that adults may frequently help scaffold adolescents' goal management (e.g., providing structure and reminders to keep them on task). Future research could test this possibility by investigating whether caregiver scaffolding behaviors moderate the common EF-dependent stress association.

Although we predicted age moderation based on past findings (Snyder and Hankin 2016), other studies have found associations between EF and the p factor in younger individuals (Castellanos-Ryan et al. 2016; Hatoum et al. 2017; Huang-Pollock et al. 2016; Martel et al. 2017; Shanmugan et al. 2016). The closest parallel to the current study (Hatoum et al. 2017) used the same EF model as here. However, in contrast to the current study, which assessed youth self-report of recent symptoms, Hatoum et al. (2017) assessed psychopathology with teacher report from age 7–16. Since this captures only those aspects of psychopathology that are stable across time and observable in the classroom, it may represent more severe and/or less well-regulated expressions of symptoms, potentially accounting for the difference. In the same sample, self-reported depression symptoms (Friedman et al. 2018) and substance use behaviors (Gustavson et al. 2017), also tended to be more related to common EF in late adolescence than in early adulthood, in contrast to the current results. However, these trends were based on longitudinal assessments across two waves as opposed to continuous interaction within a cross-sectional assessment; this design difference and differences in reporters and psychopathology measures across studies make the source of the discrepancy difficult to determine.

Stress and Rumination Effects

While the primary focus of the current study was on better understanding EF-psychopathology liability links, it also provided new insights into how stressful life events and rumination relate to the bifactor model of psychopathology liability. First, dependent stressful life events were associated with all three psychopathology liability dimensions. The association with the p factor is consistent with the model of stress as a transdiagnostic risk factor (e.g., Grant et al. 2014), but the associations with the internalizing- and externalizing-specific factors also suggests that stress further confers specific risk for internalizing and externalizing dimensions, potentially through different mechanisms (Lahey et al. 2017). Previous studies found that adolescent chronic stress (Snyder et al. 2017b) and victimization (Schaefer et al. 2017) are associated with the p factor and externalizing-specific factor, and childhood maltreatment is associated with the p factor in adults (Caspi et al. 2014), but none found associations with the internalizing-specific factor. Dependent stressful life events are a particularly strong risk factor for depression and anxiety (e.g., Liu and Alloy 2010), and thus may be more strongly associated with the internalizing-specific factor than other types of stress.

Second, rumination has not previously been examined in relation to the bifactor model of psychopathology liability. In the current study, rumination was strongly associated with the p factor. Rumination was additionally associated with higher internalizing-specific liability, but this association was much weaker, supporting the view that rumination is a broad transdiagnostic risk factor rather than specific to depression or internalizing psychopathology (e.g., Aldao et al. 2010; Johnson et al. 2016; Hankin et al. 2016a; Nolen-Hoeksema and Watkins 2011). Although rumination was

predominantly associated with the p factor, the additional association with internalizing-specific psychopathology suggests it may confer risk for anxiety and depression via both common and specific mechanisms. For example, both the genetic and non-shared environmental influences (which may include stressful life events) on rumination overlap with those on multiple forms of psychopathology; however, the extent of genetic and environmental overlap with rumination varies across disorders, suggesting multiple etiological pathways of risk between rumination and psychopathology (Johnson et al. 2016).

Once variance shared with internalizing is accounted for via the p factor, youth who engage in higher rumination had lower externalizing-specific liability. Potentially, dwelling on the negative consequences of past behaviors can help prevent youth from engaging in further problematic behaviors, at least if they experience guilt when doing so, as guilt is negatively associated with externalizing in youth (e.g., Muris et al. 2016). In the current study, we used a measure of rumination specifically during sad mood; other forms of rumination (e.g., anger rumination) may be positively associated with externalizing (e.g., du Pont et al. 2017).

Limitations and Future Directions

The current study has several limitations, which provide important areas for future research. First, the study was cross-sectional, precluding conclusions about the temporal ordering of variables in the models. The current study tested a hypothesized process model based on previous research and theory, identifying possible mediating mechanisms as a promising starting point for future longitudinal studies. There has been little research thus far on longitudinal links between bifactor models of psychopathology and EF. One study found associations between working memory and psychopathology 2 years later, but did not test prediction of *change* in psychopathology (i.e., controlling for psychopathology at the first time-point; Castellanos-Ryan et al. 2016). Two other studies used factors that captured stability of psychopathology across multiple time points, and found that a p factor across age 7–16 predicted EF at age 17 (Hatoum et al. 2017), and a p factor across age 18–38 predicted EF at age 38 (Caspi et al. 2014). No studies to date have tested whether earlier EF predicts later common psychopathology liability, consistent with the theory that EF is a transdiagnostic risk factor (e.g., Beauchaine and Zisner 2017; Goschke 2014; McTeague et al. 2016; Snyder et al. 2015).

Thus, future longitudinal research with EF and psychopathology assessed at multiple time points is needed to determine if EF may be a risk factor or consequence of common psychopathology, or both (transactional relations). However, given evidence that at the latent level, there is relatively strong stability of individual differences in both EF (Friedman et al. 2016) and psychopathology (e.g., Murray et al. 2016; Olino et al. 2018; Snyder et al. 2017c), it may be difficult to establish temporal precedence even with such longitudinal studies. Rather, EF impairments and psychopathology might best be conceptualized as transactionally affecting one another to maintain high levels of both.

Second, the current study focused on psychopathology and EF in adolescence and emerging adulthood in a non-selected community sample. Future research is needed to determine if the model generalizes to other age groups, and to high-risk or clinical populations. Our age moderation findings suggest the pathway between common EF and stressful life events tested here may be operative in older but not younger youth; studies across the lifespan are needed to replicate this and test whether EF-stress generation links are specific to the late adolescence/emerging adulthood developmental period.

Translational Implications

As noted above, these findings will need to be confirmed in high-risk and clinical samples, and with longitudinal studies, prior to attempts to translate them to practice. Nonetheless, better understanding the mechanisms by which EF may serve as a transdiagnostic risk factor for psychopathology has the potential to inform new targets for intervention. There has been a great deal of interest in EF training as a potential prevention or treatment strategy, but thus far there is limited evidence that training transfers to real-world functioning (Diamond and Ling 2016). Interventions aimed at disrupting the link between poor EF and stress generation might be a more promising approach. That is, rather than attempting to train EF, it may be more beneficial to reduce the need for EF to prevent stressful consequences, through assistance from others, structuring the environment to reduce demands, or ideally by training individuals on compensatory strategies that work around EF deficits. The current study found that the link between EF and dependent stressful life events was specific to common EF, suggesting that training in compensatory strategies for goal-management (goal setting and monitoring, time management, organization and planning) could potentially mitigate the effects of poor common EF to reduce stress and thus potentially reduce psychopathology risk. Such skills are often taught as part of college success curricula for students at risk of academic problems when entering college (e.g., Kennedy 2017). The current study suggests these programs may be promising for youth at risk for psychopathology as well.

Conclusions

Broad patterns of poorer performance on EF tasks associated with multiple forms of psychopathology symptoms may be best explained by associations between common EF and common psychopathology liability, although there are also some internalizing-specific associations. Poorer common EF was associated with internalizing and common (p factor) psychopathology liability via dependent stressful life events and rumination in older youth. This developmental period of increasing demands for independence and reduced adult support may be a critical window for risk associated with poor EF. Interventions aimed at disrupting the link between poor goal-management and stress have potential for reducing dimensional psychopathology, especially among emerging adults.

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Compliance with Ethical Standards

Conflict of Interest Hannah R. Snyder, Naomi P. Friedman and Benjamin L. Hankin declares that they have no conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study. Participants gave written informed consent (ages 18–22) or written informed assent with parental written informed consent (ages 13–17).

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