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The Structure of Executive Dysfunction in Depression and Anxiety

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Abstract

Background: Although research has demonstrated that depression and anxiety are associated with problematic executive function (EF), results are often inconsistent and underspecified. Delineating specific EF impairments in depression and anxiety has the potential to provide a mechanistic account of symptom presentation and course in these highly co-occurring disorders. The present study evaluated associations between components of EF and symptom dimensions of depression (depressed mood) and anxiety (anxious apprehension, anxious arousal) using factor analyses and structural equation modeling.

Methods: Undergraduates ($N = 1,123$) completed self-report measures of EF in everyday life and of psychopathology. Based on a three-factor model (Miyake et al., 2000), item-level exploratory ($n = 561$) and confirmatory ($n = 562$) factor analyses were conducted on inhibition, shifting, and working memory scales chosen from the EF measure. Structural equation modeling tested the relationship of EF factors to dimensions of psychopathology using the total sample.

Results: A three-factor model of EF best fit the data and was replicated via confirmatory factor analysis. Depressed mood and anxious arousal evidenced broad deficits across all EF domains, whereas anxious apprehension evidenced shifting disruptions.

Limitations: Perceived EF may not index the same constructs as performance-based EF tests. Further, the present study was restricted to college students, warranting replication in other samples.

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Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Conclusions: Findings suggest that depressed mood and anxious arousal are characterized by a general disruption in the ability to maintain task goals, whereas anxious apprehension is characterized by cognitive inflexibility. EF impairments are likely contributory factors in the maintenance of affective disorders.

Keywords

executive function; affect; psychopathology; arousal; factor analysis; latent class analysis

Introduction

Depression and anxiety are the most prevalent and burdensome forms of psychopathology worldwide, costing between \$47 and \$210 billion per year in care and lost time (Greenberg et al., 2015; Kessler et al., 2015). Individuals with co-occurring depression and anxiety are more likely to experience severe and persistent symptoms and greater treatment resistance (Jakubovski & Bloch, 2014; Kessler et al., 2015; Saveanu et al., 2015). Their high comorbidity (up to 70%; Kessler et al., 2005) and recurrence rates suggest that there are specific factors that increase people's risk for repeated episodes.

One probable but poorly understood domain of cognitive vulnerability is executive function (EF) and its relationship to symptom development, presentation, and course in depression and anxiety. Executive function refers to processes that regulate non-executive cognitive processes (e.g., motor responses) to flexibly guide behavior toward a goal, especially in novel situations (Banich, 2009). It is likely that the cognitive difficulties in attention and memory that are associated with depression and anxiety (e.g., difficulty concentrating and indecisiveness; APA, 2013) are driven in part by EF deficits (Snyder et al., 2015b). Supporting this view, research has demonstrated that depression is associated with impaired performance in specific domains of EF such as inhibition or termination of a pre-potent response (Bredemeier, Warren, Berenbaum, Miller, & Heller, 2016; Joormann & Gotlib, 2010) as well as shifting or alternating attention between tasks or mental sets (Austin et al., 2001; Bredemeier et al., 2016). Others have concluded that depression is associated with broad impairments in EF (for reviews, see Rock et al., 2014; Snyder, 2013). Research on anxiety-related EF impairment is less well developed. Evidence suggests deficits in working memory capacity (e.g., Eysenck, Payne, & Derakshan, 2005; Hayes, Hirsch, & Mathews, 2008; Moran, 2016), updating working memory (e.g., Snyder et al., 2015a), and shifting attention (Airaksinen, Larsson, & Forsell, 2005), though these findings are inconsistent (e.g., Castaneda et al., 2010; Snyder et al., 2015b).

Cognitive models of depression posit that deficits in controlling information in working memory maintain mood symptoms (see LeMoult & Gotlib, 2019, for a review). Specifically, difficulties inhibiting and updating negative information in working memory are thought to contribute to cognitive biases and maladaptive emotion regulation strategies (e.g., excessive rumination). A prominent theory in cognitive research proposes that anxiety impairs performance because it reduces attentional control in the presence of salient distracters (Berggren & Derakshan, 2012; Eysenck, Derakshan, Santos, & Calvo, 2007). According to Attentional Control Theory, anxiety is hypothesized to affect inhibition of task-irrelevant

stimuli and shifting attention. Although Attentional Control Theory represented significant progress in that it targets specific EF components (unlike cognitive theories of depression), this theory does not distinguish among dimensions of anxiety. Anxiety is a heterogeneous construct, and it has been proposed that it is composed of anxious apprehension, or a propensity to engage in worry, and anxious arousal, a tendency to experience enduring patterns of hypervigilance and symptoms of intense fear and/or panic in response to relatively mild stressors (Sharp, Miller, & Heller, 2015). As anxious apprehension and anxious arousal are psychologically and physiologically distinct dimensions of anxiety that are associated with distinct neural mechanisms (see Sharp, Miller, & Heller, 2015, for a review), they should be characterized by different executive dysfunction profiles. The present study investigated EFs associated with specific dimensions of depression and anxiety by drawing upon an empirically supported theory of EF and utilizing a statistical framework that fosters systematic examination of executive impairment.

An Executive Function Framework

A significant problem in the study of EF has been conceptual in nature (Stuss & Alexander, 2000). EF is often difficult to define and is frequently framed or operationalized imprecisely (Martin & Failows, 2010). Despite these limitations, neuropsychological research supports distinguishing EFs (Miyake et al., 2000), although the exact decomposition remains a matter of debate. Given the variable definitions of EF, it is not surprising that inconsistent findings of EF integrity/impairment in psychopathology have emerged.

In an influential contribution, Miyake et al. (2000) used latent variable analysis to demonstrate that EF is multi-dimensional, parsing it into three separable but related fundamental domains: 1) shifting or alternating attention between tasks/mental sets, 2) updating of working memory representations, and 3) inhibition of dominant or prepotent responses (the latter reconceptualized as subsumed by a more general ability to maintain task goals; Miyake & Friedman, 2012). Although the component processes of shifting, updating, and inhibition are not intended to be an exhaustive list of executive processes, they are frequently identified in the literature as important EFs and are more circumscribed than some other executive processes (e.g., “planning”).

Shifting, updating, and inhibition are considered to act as control functions for working memory. Working memory is defined variously in the literature, with a common view that it covers both the focus of attention and the active representation and manipulation of context-specific information (Baddeley, 2003). Accordingly, the concept refers both to the contents of a particular type of memory and certain EF operations that work with that content. Working memory is a limited capacity system (Engle, Kane, & Tuholski, 1999) that relies on EF processes to effectively update and manage its contents (Friedman & Miyake, 2004). EFs allow relevant information to enter, block entrance of intrusive irrelevant material, and discard information that is no longer relevant (Engle et al., 1999). Given that the experience of negative mood states and negative life events activates mood-congruent representations in working memory (Siemer, 2005), the ability to control the contents of working memory could be crucial in understanding why some individuals more easily recover from negative affect, whereas others initiate and persist in using maladaptive emotion-regulation strategies

that promote and maintain negative affect. Identifying specific EF impairments in specific dimensions of depression and anxiety has the potential to provide a novel, mechanistic account of such maladaptive patterns of behavior, as well as understanding emotion-regulation proclivities.

The Present Study

The present study drew upon on the Miyake et al. (2000) model to test contrasting hypotheses and findings regarding the nature of EF disruptions in depression and anxiety. We tested whether depression is best characterized by deficits that are specific to inhibition and updating (as suggested by cognitive and neurobiological frameworks; e.g., De Raedt & Koster, 2019; Joormann & Gotlib, 2010; LeMoult & Gotlib, 2010) and/or to shifting (as suggested by Austin et al., 2001), or that reflect a general EF deficit manifested across inhibition, shifting, and updating (Snyder, 2013). Similarly, we evaluated whether anxious apprehension and anxious arousal would result in inhibition and shifting deficits as predicted by Attentional Control Theory (Eysenck et al., 2007) or in updating deficits (e.g., Snyder et al., 2015a). It was hypothesized that depression would be associated with broad EF impairments, as affective neuroscience studies have demonstrated functional abnormalities across several prefrontal cortical regions associated with multiple EFs (e.g., Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Zilverstand, Parvaz, & Goldstein, 2017). Based on neuroimaging evidence supporting distinct patterns of brain activity associated with individual differences in anxious apprehension and anxious arousal during an EF task (Engels et al., 2007; 2010; Warren et al., 2013; for a review, see Sharp et al., 2015), it was hypothesized that anxious apprehension (i.e., worry) would be associated with problems in shifting and that anxious arousal would be associated with broad EF impairments.

A challenge in assessing EF performance in the context of professional and laboratory settings (typically one-at-a-time tasks administered in a quiet environment) is its poor ecological validity, which can overestimate or otherwise misrepresent real-world performance (Chaytor, & Schmitter-Edgecombe, 2003). Some have argued that traditional EF tests are based on hypothetical constructs that have little predictive value for EF in the real world (Burgess et al., 2006; Parsons, Carlew, Magtoto, & Stonecipher, 2017). A psychometric concern is that EF tests frequently employed in clinical and experimental settings that are reliable at the group level may not translate well to individual differences contexts because they minimize between-subject variability. As a result, some EF tests may not consistently distinguish between individuals (Hedge, Powell, & Sumner, 2018). Some work has indicated that EF self-report measures are more predictive of functional impairment and psychopathology symptoms than EF tests of overt performance (e.g., Barkley & Fischer, 2011; Barkley & Murphy, 2010; Knouse, Barkley, & Murphy, 2013), suggesting that EF rating scales index typical performance, whereas EF tests index optimal performance (Toplak, West, & Stanovich, 2013). The present study used the Behavior Rating Inventory of Executive Function – Self-Report (BRIEF-SR; Guy, Isquith, & Gioia, 2004) to improve ecological and predictive validity relative to most standard EF tests.

The items comprising the BRIEF-SR's shifting, updating of working memory, and inhibition scales were used to evaluate the utility of Miyake et al.'s (2000) three-factor model in

differentiating dimensions of depression and anxiety. As the development of the BRIEF-SR was informed by clinical interactions and the broader EF literature (Guy et al., 2004) not specific to Miyake et al.'s (2000) three-factor model, an Exploratory Factor Analysis (EFA) was implemented to evaluate and maximize inter-item correlations between indicators and construct variance. The latent factors that emerged from the EFA were used to define EF constructs. The measurement model resulting from EFA was subsequently tested via confirmatory factor analysis (CFA) in a non-overlapping sample of participants. Pooling the samples, structural equation modeling (SEM) was then used to estimate relationships between the EF latent variables and dimensional measures of psychopathology symptoms, specifically anxious apprehension, anxious arousal, and depressed mood.

Methods

Participants

Participants were undergraduates ($n=1,140$) who provided informed consent prior to completing a series of questionnaires for credit in a psychology course. The questionnaires assessed symptoms associated with anxiety and depression: the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the Anxious Arousal and Anhedonic Depression scales of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). Participants filled out the BRIEF-SR during the same questionnaire session. Data from 17 participants were excluded from analyses if questionnaire data had missing or illegible values or unusual response patterns or did not meet tests of multivariate normality (Mahalanobis, 1936). The final sample consisted of 635 females and 451 males¹ (mean age² = 18.7 years, $SD = 1.1$). The final sample ($N = 1,123$) was divided in half as observations were randomly selected for exploratory ($n=561$) and confirmatory ($n=562$) factor analyses. The total sample ($N = 1,123$) was used for SEM. As the present sample served as a recruitment pool for a neuroimaging study on affective and cognitive risk factors for psychopathology, all participants were right-handed, native speakers of English with self-reported normal hearing and color vision. Using cutoff scores established by receiver-operating characteristic analyses of the PSWQ (Behar, Alcaine, Zuellig & Borkovec, 2003; Fresco, Mennin, Heimberg, & Turk, 2003) and the depressed mood scale of the MASQ (Bredemeier et al., 2010), degree of psychopathology in the present sample was estimated as follows: between 13.5% and 18.9% of the sample met the clinical cutoff for generalized anxiety disorder, between 15.6% and 23.6% of the sample met the clinical cutoff for major depressive disorder, and between 4.1% and 7.3% of the sample met the clinical cutoffs for both disorders. Rates of depression and anxiety in the present sample are consistent with national estimates in college samples (Duffy, Twenge, & Joiner, 2019). Subsets of participants from the present sample with clinically diagnosed anxiety and mood disorders are published elsewhere (e.g., Bredemeier et al., 2016; Madian et al., 2019; Sadeh, Spielberg, Warren, Miller & Heller, 2014; Warren et al., 2013). The study was approved by the University of Illinois at Urbana-Champaign Institutional Review Board.

¹Thirty-seven individuals did not specify their gender

²Forty individuals did not specify their age

Questionnaires and Procedures

Participants completed the BRIEF-SR questionnaire (Guy, Isquith, & Gioia, 2004), involving 80 items assessing EF problems in daily life during the last six months on a three-point scale (1 = *never*; 2 = *sometimes*; 3 = *often*). Research indicates that the BRIEF-SR has good clinical utility (e.g., Niendam, Horwitz, Bearden & Cannon, 2007) and internal consistency in normative and clinical samples (Cronbach's alpha = .82 for shifting, updating of working memory, and inhibition scales; Guy et al., 2004). Only items from the shifting, updating, and inhibition scales ($n=35$) were subjected to EFA as the goal of the present study was to test specific executive dysfunction hypotheses characterizing depression and anxiety dimensions based on Miyake et al.'s (2000) three-factor model.

The 16-item PSWQ was used to assess anxious apprehension (i.e., the tendency to engage in worry). Participants responded to questions such as "My worries overwhelm me," by rating how characteristic (1 = *not at all* to 5 = *very typical*) each statement was of them. The Anxious Arousal scale of the MASQ (MASQAA) consists of 17 items in which participants responded to statements such as "Startled easily." An eight-item subscale of the MASQ Anhedonic Depression (MASQAD8) was used to measure depressed mood (Nitschke et al., 2001), given its utility in predicting current depressive disorders (Bredemeier, Spielberg, Siltan, Berenbaum, Heller, & Miller, 2010). The MASQAD8 scale consists of items such as "Felt like nothing was very enjoyable." For both MASQ scales, participants rated how much they experienced each item during the previous week (1 = *not at all*, 5 = *extremely*). Research has shown that the PSWQ and MASQ have excellent test-retest reliability and good convergent and discriminant validity in undergraduate and clinical samples (Meyer, et al., 1990; Nitschke et al. 2001; Watson et al., 1995). Internal consistencies for the present sample were .93, .84, and .75, respectively. Dimensional measures of anxiety and depression were selected because they have been shown to effectively distinguish these highly co-occurring constructs, which share many overlapping symptoms (Nitschke et al., 2001).

Data Analysis

The distributions of the observed responses to the BRIEF-SR items did not have a multivariate-normal distribution. Research has indicated that using normal-theory estimation (e.g., Pearson product-moment correlations) factor analytic techniques for ordered, categorical responses to Likert-type scales could result in biased model fit statistics, negatively biased parameter estimates, inflated error variances, and extraction of illegitimate factors (Flora, Finkel, & Foshee, 2003). Thus, polychoric correlations were used for EFA and CFA (Olsson, 1979). Additionally, robust maximum likelihood estimation mean- and variance-adjusted weighted least squares (WLSMV; Muthén, du Toit, & Spisic, 1997) were implemented, as this method has been shown to perform well when modeling categorical data (Brown, 2006; Flora & Curran, 2004).

Mplus 8 software (Muthén & Muthén, 2017) was used to conduct factor analyses and SEM. Resulting items from EFA using a first sample were used as indicators in CFA of a second, independent sample. CFA served as an objective test of the statistical fit against the EF factor model established using EFA. Model fit (CFA and SEM) was evaluated using the mean- and variance-adjusted chi-square goodness-of-fit statistic (χ^2 ; Muthén et al., 1997),

the comparative fit index (CFI; Bentler, 1990), the Tucker-Lewis index (TLI; Tucker & Lewis, 1973), and root-mean-square error of approximation (RMSEA; Steiger, 1990). Simulation studies in Yu and Muthén (2001) suggested the following goodness-of-fit values for categorical outcomes: CFI>.95, TLI>.95, and RMSEA<.06, which are consistent with Hu and Bentler's (1999) recommendations. The error variances of two inhibition items were allowed to co-vary to account for similarity in question structure.

Scores on the dimensional measures of anxiety and depression were added as manifest variables. SEM was used to test the relationships between the latent EF variables and the three psychopathology scores, as this method allows for these relationships to be estimated simultaneously and (unlike regression) explicitly accounts for measurement error in predictor variables. Additional structural tests of this model were conducted in order to evaluate potentially distinct relationships between EF latent variables and psychopathology scores. A series of nested models was created in which pairs of standardized psychopathology regression weights leading to one of the latent variables were constrained to be equal and were subsequently compared to a model in which all regression weights were allowed to be freely estimated. All difference tests of the nested models were performed using a chi-square difference procedure described by Asparouhov and Muthén (2006). Model χ^2 values and degrees of freedom are not reported for these nested model tests, as they are not interpretable when using WLSMV (only p-values are interpretable; Muthén, 2008).

Results

Exploratory Factor Analysis

Thirty-five items from the BRIEF-SR shifting ($n=10$), updating of working memory ($n=12$), and inhibition ($n=13$) scales were subjected to EFA. Given theoretical and empirical support for moderate correlations among shifting, updating, and inhibition EF processes (e.g., Miyake et al. 2000), an oblique rotation, the Promax method, was applied. In order to obtain simple factor structure, items were retained if their primary loading was $\geq .45$ and cross-loading was $\leq .2$. Following procedures outlined by Brown (2006), factor retention was determined using multiple methods: examination of a scree plot of the eigenvalues, goodness of model fit statistics (χ^2 , RMSEA), and evaluation of the meaningfulness and interpretability of the factors that emerged. Poorly defined factors (e.g., a one-item loading) were eliminated.

Nineteen items (shifting $n=4$; updating $n=5$; inhibition $n=10$) were retained that met the above outlined criteria. Examination of the scree plot (Figure 1), model fit statistics, and interpretability of factors indicated that a three-factor solution best explained the relationships among the items (RMSEA values for one- and two-factor solutions $\geq .1$; χ^2 p s<.001). The complete three-factor solution and model fit statistics are presented in Table 1. Correlations among the three EF latent factors were small to moderate, ranging from .23 to .46 (p <.001; see Table 1).

Confirmatory Factor Analysis

Although the chi-square goodness-of-fit statistic (χ^2) is typically used to test the fit of CFA models, several fit statistics are reported here, given this statistic's sensitivity to large sample sizes and consequently excessive Type I error rates (Kline, 2010). The three-factor model was successfully estimated and associated with a χ^2_{61} value of 315, $p < .001$ (Figure 2). Fit indices indicated that this three-factor model provided an excellent fit to the data (CFI=.968; TLI=.963; RMSEA=.045, 90% confidence interval = .038 to .052). Measurement weights for each BRIEF item were significant at $p < .001$ (see Table 2 for standardized estimates). Correlations among the three EF latent factors were moderate, ranging from .32 to .44 ($p < .001$; see Figure 2 and Table 2).

Structural Equation Modeling

Descriptive statistics of the psychopathology measures for the total sample are presented in Table 3. Criteria for evaluating model fit were identical to those for the CFA procedure. The model was successfully estimated and associated with a χ^2_{196} value of 578, $p < .001$. Fit indices indicated that this model provided adequate to good fit to the data (CFI=.954; TLI=.946; RMSEA=.042, 90% confidence interval = .038 to .046)³. Measurement weights for each BRIEF item were significant at $p < .001$ and were virtually identical to the measurement weights determined by the CFA procedure. Similarly, the correlations among the EF latent factors were small to moderate, ranging from .21 to .38 ($p < .001$; see Figure 3). The proportion of variance explained by each latent factor was the following: inhibition, 13.6%, shifting 26%, and updating 15.3%.

The psychopathology manifest variables (PSWQ, MASQAA, and MASQAD8) were modeled as exogenous (independent) variables predicting endogenous (dependent) EF latent variables. Increased levels of anxiety and depression were differentially associated with worse EF (see Figure 3). As shown in Table 4, PSWQ positively predicted problems with shifting, whereas MASQAA and MASQAD8 positively predicted problems with all three domains of EF. Additional structural tests between psychopathology domains determined that the magnitude of the γ (path from depression or anxiety independent variables to EF dependent variables) for PSWQ predicting shifting was larger than the γ 's for MASQAA ($p < .001$) and MASQAD8 ($p < .001$). The γ for MASQAD8 predicting shifting was larger than the γ for MASQAA ($p < .04$). For updating, the γ for MASQAA was larger than the γ 's for PSWQ ($p < .001$) and MASQAD8 ($p = .02$); MASQAD8 γ was larger than PSWQ γ ($p < .01$). Finally, the γ for MASQAA predicting inhibition was larger than the γ 's for PSWQ ($p < .001$) and MASQAD8 ($p < .001$). The γ s for MASQAD8 and PSWQ predicting inhibition were not statistically different ($p = .08$).

³As there are known gender differences in the prevalence of mood and anxiety disorders, the present SEM was repeated adding gender as a covariate. Model fit statistics were nearly identical (χ^2_{260} value of 629, $p < .001$; CFI=.953; TLI=.945; RMSEA=.036, 90% confidence interval = .033 to .040). Shifting impairment was greater for females than males ($\gamma = -0.082$, $p < .01$). The effect of depressed mood on shifting was greater for females than males ($\gamma = -0.066$, $p < .05$). Lastly, the effect of anxious apprehension on shifting was greater for males than females ($\gamma = 0.069$, $p < .038$). No other gender effects were observed.

Discussion

Although research has demonstrated that anxiety and depression are associated with disruptions in EF, the specific effects observed have been mixed. The present study tested the statistical effects of dimensions of depressed mood and anxiety (operationalized as anxious apprehension and anxious arousal) on EF. As expected, an EFA applied to an ecologically-sensitive, self-report measure of EF in everyday life yielded a three-factor structure representing shifting, updating, and inhibition domains and was replicated via CFA in an independent sample. Also consistent with hypotheses, SEM indicated that depression and anxious arousal demonstrated broad deficits in EF, whereas anxious apprehension was associated with deficits in shifting. As there are few studies that compare prominent mood and anxiety dimensions in terms of their relationships with specific EF domains, present findings can help explain inconsistencies in previous research and provide novel insights into the nature of EF deficits in depression, apprehension, and arousal.

The fact that depressed mood and anxious arousal evidenced reductions across all EF domains indicates that these dimensions may be better characterized by a general disruption in the ability to maintain task goals, also referred to as “common EF” or what is shared among EF domains (Miyake & Friedman, 2012). Common EF represents stability (via goal maintenance) and demonstrates an opposing relationship with shifting (or mental flexibility; for a review, see Snyder et al., 2015b). Delineating how common and specific EF deficits predict clinically relevant phenomena has the potential to advance understanding of EF contributions to psychopathology development, comorbidity, and heterogeneity in symptom presentation. For example, present results indicate that deficits in common EF represent a transdiagnostic feature of depression and anxiety. Indeed, recent work suggests that disruptions in common EF coupled with elevated repetitive negative thought (an element common to worry and rumination; Hur, Heller, Kern, & Berenbaum, 2017) is an important pathway to the development of psychopathology (Madian, Bredemeier, Heller, Miller, & Warren, 2019). Although depressed mood and anxious arousal share common EF reductions, their behavioral phenotypes demonstrate opposing presentations – depressed mood as a lack of arousal and anxious arousal as a pattern of enduring hypervigilance. Future research on depression and anxiety would benefit from exploring how EF deficits predict and interact with other symptom dimensions (e.g., positive and negative affect) that give rise to pathways of disorder co-occurrence and symptom heterogeneity (e.g., Hur et al., 2015; Madian et al., 2019).

Present findings might also have important theoretical implications. Attentional Control Theory (Eysenck et al., 2007) asserts that (undifferentiated) “anxiety” is related to shifting and inhibition impairments. As evidenced here, the nature of executive dysfunction depended on carefully differentiating anxiety dimensions, as well as measuring multiple components of EF. Anxious apprehension was associated with shifting impairments only, whereas anxious arousal demonstrated impairments across all three EF domains. In the context of Attentional Control Theory, results suggest that alterations in shifting have an important influence on cognition and emotion regulation associated with anxious apprehension or excessive worry, but that common EF disruptions, rather than inhibition per se, play a prominent role in anxious arousal. Results also suggest that anxious arousal, rather

than anxious apprehension or worry, is the dominant component of anxiety's effect on cognition and perhaps processing efficiency, although this remains to be empirically tested.

As hypothesized, anxious apprehension predicted impairment in shifting, suggesting that individuals who experience elevated levels of worry have difficulties making necessary cognitive adjustments for switching task sets, resulting in cognitive inflexibility. Impaired shifting could prevent appropriate selection of working memory contents that are pertinent to the task at hand, and could cause difficulty making transitions, problem-solving inflexibility (e.g., approaching a different problem with the same strategy), and difficulty changing focus from one mindset or topic to another. Individuals with elevated anxious apprehension may have difficulty switching attention from a particular set of thoughts to new thoughts or task-relevant thoughts, consequently maintaining dysfunctional worry. Present results have implications for understanding psychological factors maintaining and perhaps contributing to Generalized Anxiety Disorder (GAD). Although excessive and stable patterns of worry are defining criteria of GAD, approaching the development and maintenance of GAD as an emergent property of component interactions - anxious apprehension (the propensity to engage in worry), shifting impairment (cognitive inflexibility), and other relevant components (e.g., negative affect, avoidance motivation) - may help to identify treatment mechanisms and refine treatment approaches to better target those mechanisms.

Although the present study provides new insights into alterations of EF affected by specific dimensions of psychopathology, there are some limitations. First, the study was restricted to an undergraduate sample, and results may not generalize to more cognitively diverse samples. For example, the degree of EF domain separability may be less pronounced in general community samples (e.g., Legree, Pifer, & Grafton, 1996) and may vary across the lifespan. To the degree that distinct brain regions implement these executive processes, neuroimaging evidence suggests that older adults recruit additional bilateral prefrontal regions (for a review, see Reuter-Lorenz & Lustig, 2005). Thus, generalizability to different samples remains to be established. Nonetheless, present findings could serve as a baseline of executive dysfunction in early development of depression and anxiety.

Second, although executive dysfunctions are often viewed as sequelae of psychopathology, it is possible that specific EF deficits confer vulnerability to the development and maintenance of psychopathology, or that there is a bidirectional relationship. The present study was not designed to elucidate causal direction. Prospective studies are needed to draw firm conclusions about the temporal precedence of EF impairment and the development of psychopathology. Indeed, emerging prospective work suggests that executive dysfunction is not merely a reaction to depression and anxiety but may carry its own consequences for emotion regulation and the development of psychopathology (Kertz, Belden, Tilman, & Luby, 2015; Letkiewicz et al., 2014). Experimental studies that directly test causal associations between EF and affective symptoms are needed to explicate these relationships.

Third, as the present study used self-report measures of EF and psychopathology, it is unclear to what extent self-report measures of cognition accurately index cognition in daily life versus perceptions of cognition seen through the lens of current mood. Self-report

cognition scales and performance-based measures of cognitive processes correlate relatively poorly (e.g., McAuley, Chen, Goos, Schachar, & Crosbie, 2010; Quigley, Wright, Dobson, & Sears, 2017; Toplak et al., 2013), indicating that they are likely not measuring the same construct. Some work suggests that discrepancies between these two classes of assessment are a result of measuring different aspects of cognitive and behavioral functioning that contribute independently to clinical phenomena. Specifically, EF performance-based tests assess cognitive efficiency within an optimal setting (e.g., distraction-free environment, one test given at a time) and measure broadly defined constructs narrowly (e.g., inhibition measured via a Stroop task does not translate to all areas of inhibition in an individual's life). On the other hand, EF rating scales index goal pursuit and behaviors related to achieving those goals in everyday life (Toplak et al., 2010), which may improve ecological validity but may carry interpretation limitations (e.g., personality, mood state, or motivation may influence perception and overt behavior rather than EF per se; Buchanan, 2016). Nonetheless, EF rating scales predict meaningful outcomes such as academic performance, and in at least one study good agreement was found between informant and self-report (for review, see Letkewitz et al., 2014). Further research is needed to test the extent to which performance-based neuropsychological tests replicate present findings, as well as how present findings can augment standardized testing. As both performance-based tests and EF scales provide clinically useful information but are not necessarily interchangeable measures, an integrative framework may advance the development of standardized testing measures that predict everyday behavior.

Despite these limitations, the present research provides novel insights into domain-specific EF impairments that are likely to be important factors in the maintenance, and possibly the development, of distinct dimensions of depression and anxiety. More specifically, present findings implicate EF mechanisms of maladaptive emotion-regulation processes associated with aspects of depression and anxiety. EF deficits may impair an individual's ability to evaluate, initiate, or engage in pleasurable activities or stimuli that promote pleasant emotional states. Importantly, the present study highlights typically occurring self-reported executive dysfunction in everyday living that is associated with depression and anxiety, extending previous EF research obtained in formal (and typically artificial) evaluation settings. Indeed, the cognitive processes that formal tests of EF purport to measure are still not well known, and the range of behaviors and activities in an individual's everyday environment that require these same processes remains to be established (Burgess, Alderman, Volle, Benoit, & Gilbert, 2009). Finally, this is the only within-subjects study to date that explicitly assesses specific EF impairments, at the level of latent variables, among anxious apprehension, anxious arousal, and depressed mood, so replication is needed.

In summary, the present study tested the effects of depressed mood, anxious arousal, and anxious apprehension on multiple dimensions of EF. Depression and anxious arousal demonstrated broad deficits in EF, whereas anxious apprehension was associated with deficits in shifting. As evidenced here, executive dysfunction associated with depression and anxious arousal could not be accurately accounted for by examining one aspect of EF. Furthermore, if the focus is on just one dimension of EF, as has often been the case in the literature, it is possible that what appears to be a primary EF deficit in depression or anxiety is actually the result of another correlated, yet separable EF component (e.g., inhibition vs.

shifting for depression). Comprehensive approaches in the measurement of EF domains will likely yield fruitful avenues for psychopathology research.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights:

- Executive function (EF) disruptions in depression and anxiety are underspecified
- Factor analyses supported and replicated an EF model using a self-report measure
- SEM tested types of EF difficulties related to affective symptoms
- Depressed mood and anxious arousal evidenced broad EF deficits
- Anxious apprehension (worry) evidenced a deficit in cognitive shifting

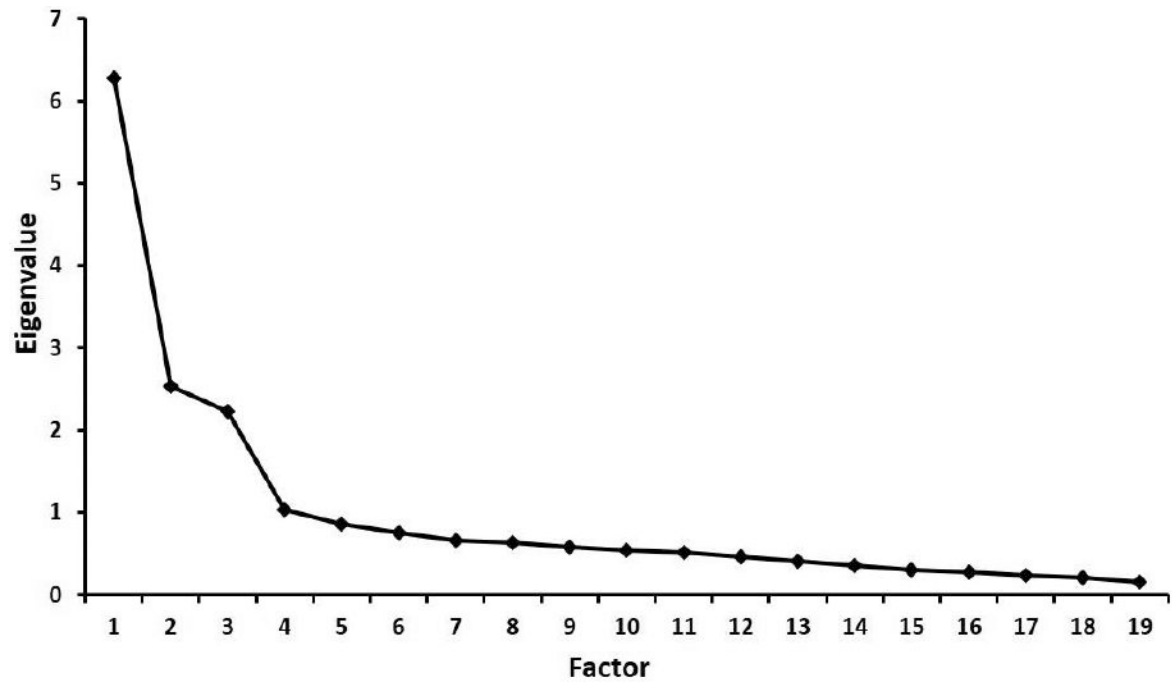


Figure 1:
Scree plot from the Exploratory Factor Analysis model indicating that three factors should be extracted. $N=561$.

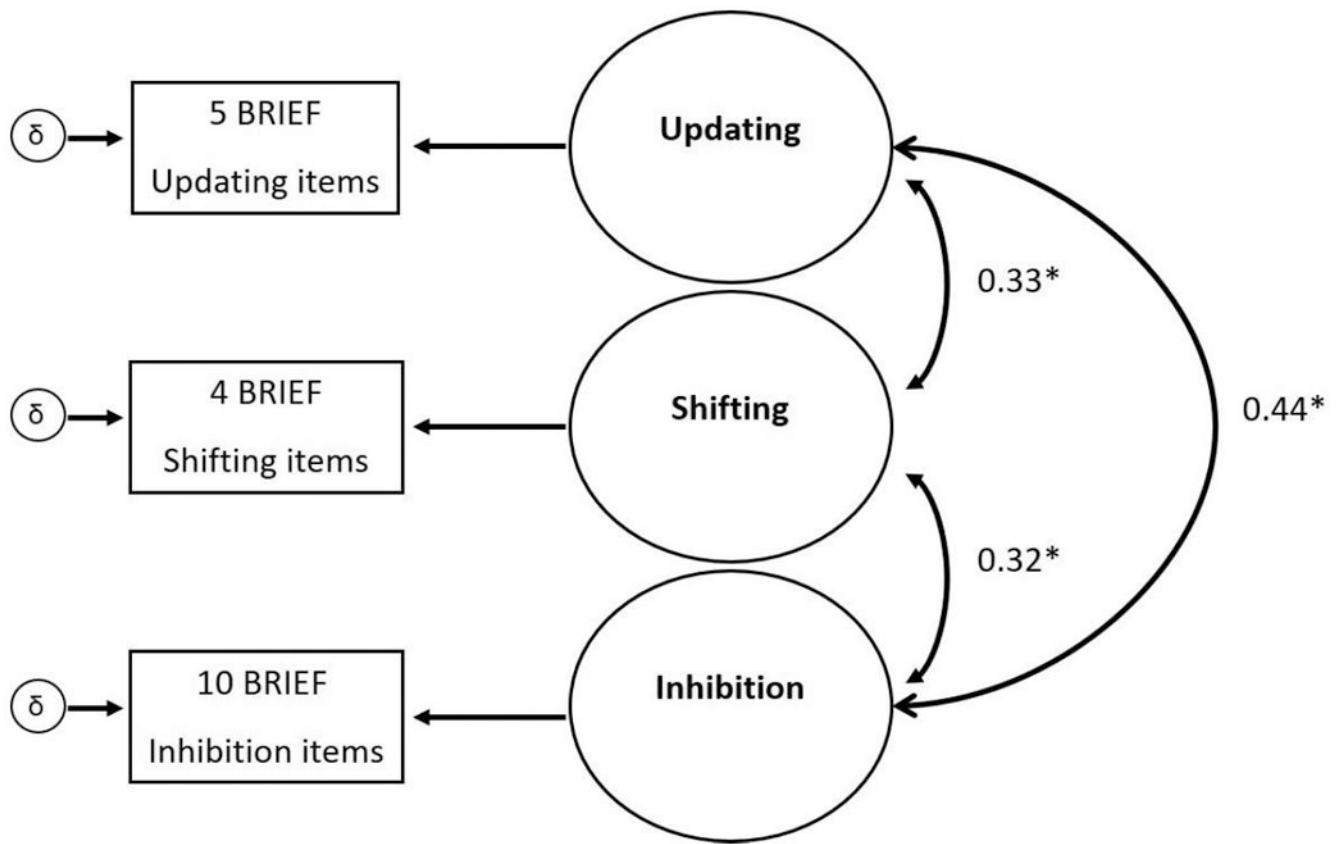
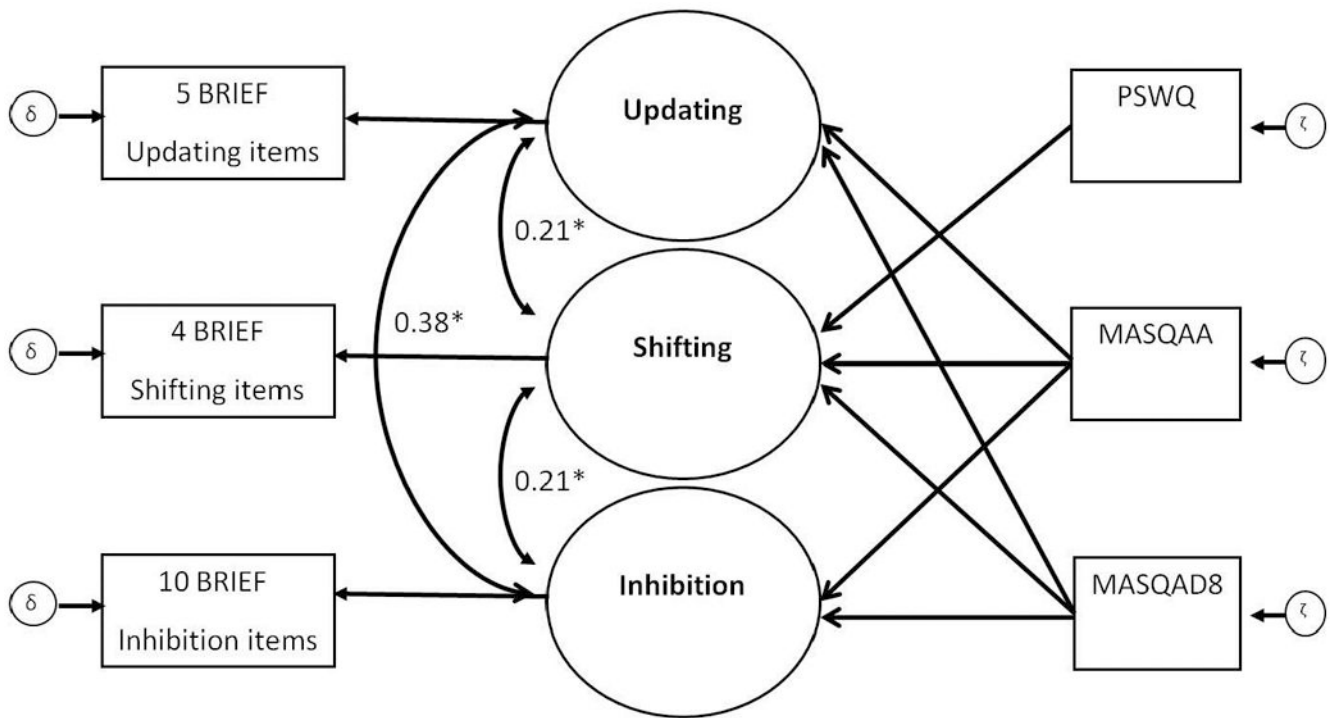


Figure 2: Confirmatory Factor Analysis model for $N=562$. Updating, Shifting, and Inhibition are latent factors. BRIEF = Behavior Rating Inventory of Executive Function. δ is a measurement error term for the observed latent factor indicators. The individual BRIEF items and covariances between error terms are not pictured for conciseness. * $p < .001$

**Figure 3:**

Structural equation model for $N=1,123$. Psychopathology measures predicting latent executive function variables updating, shifting, and inhibition. PSWQ = Penn State Worry Questionnaire. MASQAA = Mood and Anxiety Symptom Questionnaire Anxious Arousal scale. MASQAD8 = Mood and Anxiety Symptom Questionnaire Anhedonic Depression 8-item subscale for depressed mood. BRIEF = Behavior Rating Inventory of Executive Function. δ is a measurement error term for the observed exogenous (dependent) variables. ζ is a structural (regression) error term for the variance in updating, shifting, and inhibition that is unexplained by PSWQ, MASQAA, and MASQAD8. The regression paths from PSWQ, MASQAA, and MASQAD8 to the EF latent factors are referred to as γ (see Table 4 for values). The covariances between error terms, the individual BRIEF items, and the standardized regression coefficients for the psychopathology exogenous variables (see Table 4) are not pictured for conciseness. * $p < .001$

Table 1

Exploratory Factor Analysis: Three-Factor Solution

	Factor		
	1	2	3
Promax-Rotated Pattern Coefficient			
Item			
I71	0.93	-0.02	-0.17
I54	0.80	0.09	-0.10
I79	0.65	-0.04	0.19
I66	0.62	0.00	0.02
I61	0.61	-0.06	0.15
I19	0.57	0.17	-0.04
I10	0.56	0.01	-0.10
I80	0.56	-0.02	0.11
I37	0.53	-0.15	0.08
I28	0.52	0.04	0.13
S27	-0.03	0.87	0.02
S9	0.03	0.86	-0.04
S18	0.01	0.80	0.02
S36	0.03	0.58	0.12
WM73	0.01	0.01	0.83
WM63	-0.07	0.09	0.80
WM48	0.00	-0.05	0.76
WM3	-0.14	-0.01	0.69
WM39	0.19	-0.03	0.57
Interfactor Correlations			
Factor			
2	0.23		
3	0.46	0.23	

Note. $N=561$. $\chi^2_{117}=258$, $p<.001$. RMSEA = 0.046. Interafactor correlations were significant at $p<.001$. Entries in bold are the highest loading per item. I=Inhibition; S= Shifting; WM=Working Memory. The number indicates the item number on the BRIEF-SR.

Table 2

Confirmatory Factor Analysis: Standardized Regression Coefficients

Item	Factor		
	Inhibit	Shift	Update
I79	0.82	-	-
I61	0.72	-	-
I80	0.68	-	-
I28	0.64	-	-
I54	0.62	-	-
I66	0.61	-	-
I71	0.59	-	-
I19	0.59	-	-
I37	0.58	-	-
I10	0.47	-	-
S18	-	0.88	-
S27	-	0.87	-
S9	-	0.77	-
S36	-	0.59	-
WM73	-	-	0.90
WM48	-	-	0.79
WM63	-	-	0.74
WM39	-	-	0.73
WM3	-	-	0.67
Interfactor Correlations			
Factor			
Shift	0.32		
Update	0.44	0.33	

Note. $N=562$. $\chi^2_{61}=315$, $p<.001$. CFI=.968; TLI=.963; RMSEA=.045. All measurement weights and interfactor correlations were significant at $p<.001$. I=Inhibition; S=Shifting; WM=Working Memory. The number indicates the item number on the BRIEF-SR.

Table 3

Self-Report Psychopathology Scores (N=1,123)

Questionnaire	Mean	SD	Min	Max
PSWQ (Anxious Apprehension)	48.69	13.45	16	80
MASQAA (Anxious Arousal)	28.31	8.55	17	80
MASQAD8(Depressed Mood)	17.15	5.19	8	39

Note. PSWQ = Penn State Worry Questionnaire. MASQAA = Mood and Anxiety Symptom Questionnaire Anxious Arousal scale. MASQAD8 = Mood and Anxiety Symptom Questionnaire Anhedonic Depression 8-item subscale for depressed mood.

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Table 4

Structural Equation Modeling: Standardized Regression Coefficients

Variable	λ	P
Exogenous variable: PSWQ		
Updating	<.01	0.98
Shifting	0.45	<.01
Inhibition	<.01	0.91
Exogenous variable: MASQAA		
Updating	0.32	<.01
Shifting	0.08	0.04
Inhibition	0.34	<.01
Exogenous variable: MASQAD8		
Updating	0.17	<.01
Shifting	0.22	<.01
Inhibition	0.11	<.01

Note. $N=1123$. $\chi^2_{196}=578$, $p<.001$. CFI=.954; TLI=.946; RMSEA=.042. PSWQ = Penn State Worry Questionnaire. MASQAA = Mood and Anxiety Symptom Questionnaire Anxious Arousal scale. MASQAD8 = Mood and Anxiety Symptom Questionnaire Anhedonic Depression 8-item subscale for depressed mood. Updating, shifting, and inhibition represent latent variables derived from EFA and CFA.