

The Relationship between Worry, Rumination and Executive Functions in a Mixed Clinical Sample

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This study aimed to investigate the association between executive dysfunction, worry, and rumination in a sample of 51 inpatients diagnosed with depression and anxiety. The results highlighted a positive correlation between executive dysfunction and levels of worry and rumination. Notably, rumination was more strongly associated with executive dysfunction than worry, suggesting implications for the comorbidity of anxiety and depression disorders. The findings indicated that rumination acts as a mediator between executive functions and both depression and anxiety. However, worry did not show a similar mediating effect. While some of the observed executive function deficits may be explained by the presence of psychiatric and medical comorbidities, it is essential for future research to delve deeper into understanding the contribution of these variables to executive dysfunction. Possible limitations and future implications are discussed.

Keywords: executive functions, worry, rumination, depression, anxiety.

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Introduction

Emerging research has consistently highlighted a link between deficits in executive functions (EF) and symptoms of depression, observed across both clinical and non-clinical populations. Despite a wealth of studies, predominantly focused on student samples, the unique contribution and prospective predictive value of EF deficits in relation to depression symptoms remain areas of ongoing inquiry.

EF is an umbrella construct, a set of self-regulatory processes crucial for goal achievement, especially in social situations. According to Barkley (2014) EF is a broad construct comprising various interrelated mental modules, each contributing to its effectiveness. Dysfunctions in any of these modules can significantly affect overall EF performance. Barkley (2014) identifies the main components of EF as shifting, inhibition, verbal and nonverbal working memory, planning, and problem-solving, illustrating the integrated role of EF in cognitive and behavioural regulation.

In the realm of mental health, the comorbidity of depressive and anxiety disorders presents as a prevalent yet complex phenomenon, warranting deeper exploration. Ter Meulen et al. (2021) emphasize the nature of this comorbidity, which is reflected in a meta-analysis by Steel et al. (2014), noting that approximately one in five adults experience a mood, anxiety, or substance use disorder at some point in their lives. Adding to this complexity, Kalin (2020) observes that anxiety and depressive disorders not only frequently co-occur but also share symptomatology, a

point underscored by Coplan et al. (2015) and Almeida et al. (2012) who found significant overlap in lifetime prevalence rates of these disorders.

This prevalence and interrelation of anxiety and depression underscore the necessity to understand the underpinning factors that drive their development, severity, and progression. Such insights are essential for developing effective prevention and treatment strategies. Central to this understanding are the transdiagnostic mechanisms, particularly emotional regulation (ER) and Repetitive Negative Thinking (RNT), which appear to play significant roles in both anxiety and depression.

RNT, a behaviourally measurable cognitive process, is represented by thoughts oriented towards the future (worry) and the past (rumination). It represents the process of thinking rather than its temporal orientation or content. Worry, a hallmark of Generalized Anxiety Disorder (GAD; American Psychiatric Association, 2013), and rumination, a significant contributor to Major Depressive Disorder (MDD; American Psychiatric Association, 2013), are main forms of RNT. These negative thinking patterns are linked to various emotional and behavioural issues (Calmes & Roberts, 2007) and are associated with cognitive decline and an increased risk of Alzheimer's disease (Marchant et al., 2020).

Rumination is defined as a mode of responding to distress that involves a repetitive, passive focus on the symptoms of one's suffering and their potential causes and consequences (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Conceptualized as a maladaptive form of ER,

rumination is extensively implicated in the development and persistence of various psychopathological conditions (Aldao et al., 2010). Worry, characterized as a chain of uncontrollable, negatively charged thoughts and images, predominantly focused on potential negative outcomes (Borkovec, Robinson, Pruzinsky, & DePree, 1983), is similarly associated with anxious and depressive symptoms in adults (Fresco et al., 2002).

Recent studies have begun to clarify the role of rumination as a risk factor not only in mood disorders like bipolar affective disorder (Gruber et al., 2011) and eating disorders (Holm-Denoma & Hankin, 2010; Nolen-Hoeksema et al., 2007) but also in predicting anxiety symptoms (Calmes & Roberts, 2007; Nolen-Hoeksema, 2000). The interplay of rumination and worry, as common mechanisms in both depression and anxiety, is particularly intriguing, especially in light of their high comorbidity (Brown et al., 2001; Ehring & Watkins, 2008). Hankin (2008) further illuminates this connection, demonstrating the interaction between initial rumination and anxious arousal in predicting depressive symptoms. However, much of this research has been limited to younger, non-clinical populations.

Similarly, the relationship between trait anxiety, EF, and their influence on inhibitory control processes and task shifting has been explored in adult populations (Pacheco-Unguetti et al., 2010; Visu-Petra et al., 2013). Yet, questions linger about the extent to which these findings translate to clinical settings, particularly regarding the interaction between state anxiety and inhibitory control.

The concept of individual differences in EF, as detailed by Banich (2009), provides a broader perspective on human behaviour and health. These differences are not only essential in day-to-day functioning but are also closely linked to various forms of psychopathology, especially affective disorders (Snyder, Miyake, and Hankin, 2015). The exploration of EF as a transdiagnostic risk factor thus becomes central in understanding the nuances of mental health disorders.

Building upon these foundations, it is important to understand mechanisms underlying the comorbidity of depressive and anxiety disorders, with a particular focus on ER, RNT, and their subcomponents: rumination and worry. Recent empirical findings, such as those by Kim & Newman (2023) and Jamil & Llera (2021), explore the Contrast Avoidance Model (CAM) and its implications for understanding the emotional dynamics in GAD and MDD. CAM, as proposed by Newman & Llera (2011), posits that the escalation and maintenance of anxiety through worry serve as a mechanism to avert abrupt negative emotional shifts. This model implies that the act of worrying effectively acts as a buffer against sudden and intense negative emotional experiences. As a result, the reinforcement of worry occurs due to its role in staving off these sharp emotional contrasts, often referred to as Negative Emotional Contrasts (NEC) in the model by Newman & Llera (2011).

There is evidence suggesting that NEC also applies to rumination. A recent study indicated that rumination and worry amplified negative emotions and diminished emotional contrast in response to negative feedback on an intelligence test. Furthermore, the study found that groups with GAD (compared to a non-clinical group) and MDD (compared to a non-clinical group) reported using worry and rumination, respectively, as coping mechanisms for negative feedback (Jamil & Llera, 2021).

In summary, this research is situated at the confluence of key psychological concepts — EF, RNT and their specific forms, worry and rumination. It explores these constructs within the extensive framework of anxiety and depressive disorders, particularly focusing on their comorbid nature.

Objective

This research aims to deepen our understanding of the relationship between RNT, depression, anxiety, and EF deficits within a clinical population. The objectives are to assess the individual relationships between EF, worry, rumination, and symptoms of anxiety and depression, to explore how EF, worry, and rumination together influence the manifestation of symptoms of depression and anxiety, to analyse the extent to which interactions between EF and RNT components (worry and rumination) explain variations in anxiety and depression symptoms, and to investigate the mediating roles of these variables and their impact on psychopathology. The main goal is to clarify the mechanisms by which RNT, particularly in the forms of worry and rumination, interacts with cognitive functioning and contributes to the symptomatology of depression and anxiety disorders.

Research hypotheses

H1. Deficits in EF have a significant direct effect on the level of depression and anxiety, but this effect is mediated by rumination and worry.

H2. Rumination and worry mediate the effect of executive dysfunctions on the level of depression and anxiety, and they are important control variables in the relationship between executive functions and the level of depression and anxiety, respectively.

H3. The indirect effect of executive dysfunctions on the level of depression through rumination will be significant, implying that executive dysfunctions will influence the level of rumination, which in turn will influence the level of depression.

H4. The indirect effect of executive dysfunctions on the level of anxiety through worry will be significant, implying that executive dysfunctions will influence the level of worry, which in turn will influence the level of anxiety.

H5. The interaction between rumination and worry would explain additional variance in symptoms of depression and anxiety.

Method

Participants

For this research, a group of 51 psychiatric inpatients was methodically selected from the Psychiatry Clinic at Cluj-Napoca Municipal Hospital. The required sample size was determined via GPower analysis, indicating that 53 participants were needed to detect a medium effect size with 80% power and an alpha level set at .05, according to the guidelines by Faul et al. (2007). Although the GPower analysis recommended a slightly higher number, the study proceeded with 51 participants, a number that closely approaches the recommended sample size and is expected to maintain a high level of statistical validity. This minor deviation was due to practical limitations in the recruitment process. Participants included in the study were selected based on diagnoses of depressive episode (35.2%), recurrent depressive disorder (27.5%), dysthymia (5.9%), and generalized anxiety disorder (25.5%), panic disorder with agoraphobia (5.9%) at the time of their admission. Out of the 51 participants included in the analysis, aged between 22

and 78 years old ($M=53.2$, $SD=12.6$), 40 were women (78.4%) and 11 were men (21.6%). The demographic data of the sample are presented in Table 1.

Table 1. Demographic data of participants.

	Clinical group (n=51)	
	n	%
Gender		
women	40	78,4
men	11	21,6
Level of education		
Graduate of general school	8	15,7
Graduate of vocational school	13	25,5
High school graduate	24	47,1
Post-high school graduate	1	2,0
Higher education graduate	5	9,8
Ethnicity		
Romanian	43	84,3
Hungarian	5	9,8
Roma	2	3,9
Ukrainian	1	2,0
Environment		
rural	25	49,0
urban	26	51,0
Marital status		
married	28	54,9
single	8	15,7
divorced	5	9,8
widower	10	19,6
Occupational status		
employed	13	25,5
unemployed	5	9,8
housekeeper	11	21,6
retired	22	43,1
Diagnosis		
Depression	35	68,6
Anxiety	16	31,4
Psychiatric medication, hospitalization	51	100

Procedure

Each participant, after providing informed consent, was engaged in the research protocol, which included the administration of a set of five questionnaires, the protocol being carefully standardized to ensure uniformity of the procedure. Participants were ensured privacy and a conducive environment within the hospital premises to minimize distractions and facilitate focus during the completion of the questionnaires. Prior to the initiation of data collection, participants were fully informed of their right to withdraw from the study at any time, with the understanding that their decision would have no detrimental consequences. In addition, the study implemented stringent procedures to protect the confidentiality of all participant

information, in strict adherence to the ethical standards. Together with the psychological assessments, demographic and clinical data for each participant was recorded (such as gender, age, marital status, educational background, living environment - rural or urban, clinical diagnosis, and current pharmacological treatments). This information was gathered to enable comprehensive analysis and to control for variables that could influence the study's outcomes.

Instruments

Penn State Worry Questionnaire (PSWQ) (Meyer, Miller, Metzger, & Borkovec, 1990). This is a robust self-reporting tool from a psychometric standpoint, with good validity for assessing worry as a trait. Internal consistency is high (Cronbach's $\alpha = 0.91$), and test-retest reliability is quite good (the test-retest stability coefficient for an interval of 8-10 weeks was reported to be .92). The scale is composed of 16 items evaluated on a 5-point Likert scale, ranging from 1 (not at all typical for me) to 5 (very typical for me). The PSWQ showed a Cronbach's alpha of .791 in this study, indicating satisfactory internal consistency

Ruminative Response Scale (RRS), which is a subscale of the ruminative style questionnaire RSQ (Nolen-Hoeksema & Morrow, 1991). Most studies on ruminative tendencies have used RRS as an evaluation method (Roberts et al., 1998). This is a self-assessment tool composed of 22 items, measured on a 4-point Likert scale. Participants were asked to indicate what they think or do when they feel sad, unmotivated, or depressed. The ruminative style measured by RRS is a relatively stable interindividual trait over time (Just & Alloy, 1997). Previous studies have established that RRS, as a subscale of the Ruminative Responses Questionnaire (RSQ), has good internal consistency (α Cronbach = .89, Nolen-Hoeksema & Morrow, 1991), test-retest reliability over a period of 5 months ($r = .80$, Spasojević & Alloy, 2001), and predictive validity for depression (Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991). For the RRS, the Cronbach's alpha was .942, reflecting high internal consistency within our sample.

The Barkley Deficits in Executive Functioning Scale - long form (BDEFS-LF), developed by Russell A. Barkley and published by Guilford in 2011, is a self-assessment tool that can be used to test adults aged between 18 and 81 years old, and it evaluates the cognitive and behavioural manifestations of executive dysfunction. BDEFS-LF assesses those neuropsychological abilities that support and contribute to self-regulation over time, oriented towards the future: self-management to time, self-organization/problem-solving, self-restraint (inhibition), self-motivation, and self-regulation of emotions. BDEFS is a self-assessment tool organized on several sub-domains or factors (Barkley et al., 2022). The BDEFS scale has been translated and adapted into Romanian by Cognitrom, and has been standardized for the Romanian population. The BDEFS scale has good psychometric properties (reliability, validity), and is useful in evaluating the dimensions of EF in daily activities. Barkley (2014) reported a Cronbach's alpha coefficient of internal consistency >0.91 , based on a more representative sample from the USA. The BDEFS manual adapted for the Romanian population reports the internal consistency of each BDEFS-LF subscale (Cronbach's alpha coefficient), which proved to be satisfactory: Self-management to time, α Cronbach=0.949; Self-organization/Problem-solving, α Cronbach =0.958; Self-restraint, α Cronbach = .930; Self-motivation, α Cronbach = .914; and Emotion self-regulation, α Cronbach = .946.

In the current study, the BDEFS-LF demonstrated excellent internal consistency. The Total EF Score (89 items) had a Cronbach's alpha of .982. For its subscales, the alpha values were as follows: Self-management to Time (21 items) α Cronbach = .941; Self-Organization/Problem Solving (24 items) α Cronbach = .965; Self-Restraint (19 items) α Cronbach = .924; Self-Motivation (12 items) α Cronbach = .866; and Self-Regulation of Emotion (13 items) α Cronbach = .929. For the ADHD-FE Index (11 items), the internal consistency in our study was α Cronbach = .845.

Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1993) is a self-report questionnaire that measures the severity of anxiety in psychiatric and non-clinical populations. It is composed of 21 items that identify anxiety symptoms and quantify their intensity. Respondents are asked to evaluate how much they have been bothered by each item in the last week, including today, on a four-point scale, ranging from 0 ("not at all") to 3 ("severe"). BAI proved highly internally consistent (α Cronbach = .94) (Fydrich, Dowdall & Chambless, 1992).

Beck Depression Inventory-II (BDI-II; A. T. Beck, Steer, & Brown, 1996) measures the presence and severity of depression in patients diagnosed psychiatrically as well as in the non-clinical population. The BDI-II is a questionnaire consisting of 21 items that assess the severity of depressive symptoms. Each item is evaluated on a scale from 0 to 3, with summary scores ranging from 0 to 63. Numerous studies have shown that the BDI-II has high internal consistency (Cronbach's alpha = .93, Beck et al., 1996). High validity has been demonstrated, and diagnostic discrimination has been established (Dozois, Dobson, & Ahnberg, 1998). The BDI inventory has good psychometric properties and has been adapted for the Romanian population. A meta-analysis that included studies over a 25-year period estimated the internal consistency of the BDI and determined an average alpha coefficient of 0.86 for psychiatric patients and 0.81 for non-psychiatric subjects.

Design

In this study, we employed a correlational research design to explore the associations between rumination, worry, EF, depression, and anxiety. These associations were evaluated using standardized assessment tools, with the aim of understanding how rumination and worry might mediate the impact of executive dysfunctions on anxiety and depression levels. Our analysis was operationalized through the construction and testing of a structural equation model. Within this model, rumination and worry were posited as mediating variables, potentially explicating the link between executive dysfunctions and the severity of depression and anxiety symptoms. We also recorded participants' demographic characteristics—such as age, sex, and level of education—as these factors may act as potential control variables. The inclusion of these demographic characteristics allowed for a more nuanced interpretation of our results and added a layer of control for potential confounding influences.

Analysis Method

Descriptive statistical methods were used for the analysis of demographic data: means, frequencies. Correlation analysis was done using SPSS, bivariate correlation analysis, the association between EF, depressive and anxious state and repetitive negative thinking (worry and rumination) was examined by verifying the assumptions of the Spearman test.

In order to perform the mediation analysis, the assumption of linearity and homoscedasticity (homogeneity of variances) was verified (Hayes & Cai, 2007). The mediation analysis was done with the help of the R program version 4.2.2, using SEM (structural equations model), because the mediation process was extended to several mediators and dependent variables, and the MLR estimator (maximum likelihood robust) as a method of estimating standard errors. Worry and rumination variables were tested as mediators in the relationship between the predictor variable - executive functioning and the criterion variables depression and anxiety, respectively. Significant effects were reported at the traditional level of significance ($p < .05$) (Field, 2009).

Results

Descriptive statistics analysis and the Pearson correlation for the study's measures are presented in Table 2. It displays the mean scores and the Pearson correlations between BDI, BAI, and total and subscale scores of EF (BDEFS-LF).

As can be seen in Table 2, all measured variables correlate positively and very strongly. Also, there is a statistically significant correlation between BDI II and BAI, showing that in most cases scores on these two scales tend to appear together.

Table 2. Averages, standard deviations, and correlations with confidence intervals.

	<i>M</i>	<i>SD</i>	1	2	3	4
1. BDEFS total	198.14	63.73				
2. BAI	26.00	15.12	.63**			
			[.43, .77]			
3. BDI II	25.76	13.18	.78**	.69**		
			[.65, .87]	[.51, .81]		
4. RRS total	50.29	10.60	.52**	.57**	.53**	
			[.29, .70]	[.34, .73]	[.30, .71]	
5. PSWQ total	59.39	14.37	.75**	.75**	.81**	.72**
			[.60, .85]	[.60, .85]	[.69, .89]	[.56, .83]

Notes. BDEFS total = Barkley Deficits in Executive Functioning Scale – Total Score, BAI = Beck Anxiety Inventory, BDI II = Beck Depression Inventory II, RRS total = Ruminative Responses Scale – Total Score, PSWQ total = Penn State Worry Questionnaire – Total Score. *M* and *SD* are used to represent the mean and standard deviation, respectively. The values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. $p < .05$. *** $p < .001$.

As can be seen in Table 3, the skewness values of the distributions fall within the range (-0.5; 0.5), which means that the distribution is approximately symmetrical, without outliers. Kurtosis values are negative and less than 3, indicating a platykurtic distribution (Hair et al., 2021).

The results of the mediation analysis are presented in Table 4, that assessed the effect of the predictor variable on the criterion, with the role of the mediating variable, indicated here as a label, explained in the following.

Figure 1 illustrates the mediation model derived from the structural equations analysis. Executive dysfunctions, considered as independent variables, influence depression levels, referred to here as dependent variable, but not anxiety. This influence is both direct for depression but not for anxiety, and mediated by rumination and worry. The arrows illustrate the trajectories of the relationships between

these variables, and the coefficients associated with these trajectories reflect the magnitude and direction of these relationships, where positive values indicate a directly proportional relationship. The model thus suggests that executive dysfunctions have both a direct and an indirect effect on depression levels, through rumination and worry. The direct effect is the path from BDEFS total to BDI II, respectively BAI, controlling at the same time the mediators RRS and PSWQ. The indirect effect describes the path from BDEFS total to BDI II, respectively BAI through mediators. Finally, the total effect is the sum of the direct and indirect effects of BDEFS total on BDI II, respectively BAI (Gunzler, Chen, Wu, & Zhang, 2013).

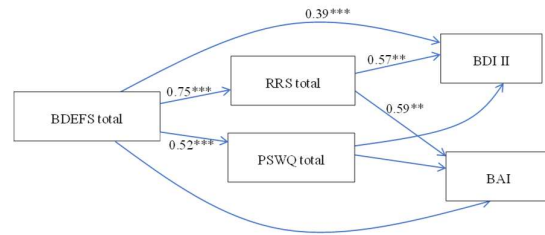


Figure 1. Graphic representation of the mediation model

Table 3. Data about the score distributions.

Variables	N	median	min	max	range	Skewness	kurtosis
BDEFS total score	51	187.00	93.00	327.00	234.00	0.55	-0.74
BAI	51	25.00	0.00	55.00	55.00	0.21	-1.10
BDI_II	51	24.00	4.00	58.00	54.00	0.32	-0.52
PSWQ_total	51	50.00	24.00	76.00	52.00	0.02	-0.35
RRS_total	51	57.00	31.00	85.00	54.00	-0.07	-1.20

Notes. BDEFS total = Barkley Deficits in Executive Functioning Scale – Total Score, BAI = Beck Anxiety Inventory, BDI II = Beck Depression Inventory II, RRS total = Ruminative Responses Scale – Total Score, PSWQ total = Penn State Worry Questionnaire – Total Score.

Tabel 4. Mediation Analysis

Criteria	Predictor	label	β	p	CI	
					LL	UL
PSWQ total	BDEFS total	a1	0.09	0.00	0.05	0.13
RRS total	BDEFS total	a2	0.17	0.00	0.14	0.20
BDI II	BDEFS total	c1	0.08	0.00	0.04	0.13
BAI	BDEFS total	c2	0.04	0.12	-0.01	0.09
BDI II	PSWQ total	b1	-0.11	0.38	-0.37	0.14
BDI II	RRS total	b3	0.53	0.00	0.28	0.78
BAI	PSWQ total	b2	0.08	0.68	-0.30	0.46
BAI	RRS total	b4	0.61	0.00	0.30	0.93
ind1		a1*b1	-0.01	0.42	-0.03	0.01
ind2		a1*b2	0.01	0.68	-0.03	0.04
ind3		a2*b3	0.09	0.00	0.04	0.14
ind4		a2*b4	0.10	0.00	0.05	0.16

Notes. PSWQ total = Penn State Worry Questionnaire – Total Score, RRS total = Ruminative Responses Scale – Total Score, BDI II = Beck Depression Inventory II, BAI = Beck Anxiety Inventory, BDEFS total = Barkley Deficits in Executive Functioning Scale – Total Score; 'a1', 'a2', 'b1', 'b2', 'b3', 'b4', 'c1', 'c2' = different pathways in the analysis, and 'ind1', 'ind2', 'ind3', 'ind4' = indirect effects; CI = Confidence Interval, LL = Lower Limit, UL = Upper Limit; β = regression coefficient, p = significance threshold (p < .05).

The effect of the total BDEFS score on BDI II was mediated by RRS. As Table 35 illustrates, the direct effect between BDEFS total and BDI II ($\beta=0.08$, 95% CI (0.04;

0.13), p < .05), respectively BAI ($\beta=0.04$, 95% CI (-0.01; 0.09), p < .05), and the mediator RRS on the predictor BDI II ($\beta=0.53$, 95% CI (0.28; 0.78), p < .001), respectively BAI was significant ($\beta=0.61$, 95% CI (0.30; 0.93), p < .001).

Indirect effects were found when RRS mediated the relationship between BDEFS total and BDI II ($\beta=0.09$, 95% CI (0.04; 0.14), $p < .05$), respectively the relationship between BDEFS total and BAI ($\beta=0.10$, 95% CI (0.05; 0.16), $p < .001$). The significance of this indirect effect was tested using bootstrapping procedures. The unstandardized indirect effects were calculated for each of the 1,000 bootstrapped samples, and the 95% confidence interval was calculated by determining the indirect effects at the 2.5 and 97.5 percentiles. The PSWQ variable did not mediate the relationship between BDEFS total and BDI II and BAI.

There is a direct statistical relationship (direct effect) between BDEFS total and BDI (coefficient = .39, $p < .001$), but there is no effect between BDEFS and BAI, but RRS mediates the relationship between BDEFS and BAI. This means that the RRS mediator explains the relationship between BDEFS and BAI, but partially explains the relationship between BDEFS and BDI II, as there are other mechanisms that explain the relationship, mechanisms that we have not included in mediation.

Discussion and Conclusions

This research aims to explore the relationships between EF deficits, RNT and depressive and anxious symptoms, in a clinical sample. The focus is on understanding how EF, along with worry and rumination, individually and jointly influence depression and anxiety symptoms, and on investigating the mediating effects of rumination and worry. The study hypothesizes that EF deficits directly impact levels of depression and anxiety, a relationship mediated by rumination and worry. Both rumination and worry are posited as key mediators in how executive dysfunctions influence these mental health conditions. Additionally, the study suggests that EF deficits indirectly affect depression through rumination and anxiety through worry. Moreover, it's hypothesized that the interaction between rumination and worry further explains variances in depression and anxiety symptoms.

Our findings partially support the hypotheses 1 and 2 of the study, which posited that deficits in EF would significantly affect depression and anxiety levels, mediated by rumination and worry. Specifically, the study reveals that rumination, rather than worry, serves as a significant mediator between EF and both depression and anxiety. This aligns with Alexopoulos's depressive-executive dysfunction hypothesis (2003) and the work of Philippot and Agrigoroaiei (2017), suggesting that a lack of executive resources may exacerbate repetitive negative thinking, thereby worsening mood. The partial mediation observed indicates that while EF deficits contribute to depression and anxiety through rumination, there are other contributing factors at play.

The study confirms hypotheses 3 and 4, with the data revealing a significant indirect effect of executive dysfunctions on depression and anxiety through rumination and worry, respectively. This suggests that executive dysfunctions exacerbate levels of rumination and worry, which in turn aggravate depressive and anxiety symptoms. This finding is consistent with the notion that impaired EF may hinder an individual's ability to disengage from negative, repetitive thoughts. The study delves into the cognitive mechanisms underlying rumination and executive dysfunction. Consistent with Linville (1996) and Joormann and colleagues (2005, 2014), our findings indicate that deficits in EF contribute significantly to rumination, which in turn impacts mood disorders. This provides a detailed

perspective on the cognitive processes involved in rumination and supports our hypothesis, highlighting the role of executive dysfunctions in exacerbating depressive symptoms through rumination.

Contrary to hypothesis 5, our results did not find significant evidence to suggest that the interaction between rumination and worry explains additional variance in symptoms of depression and anxiety. This may be due to the dominant influence of rumination over worry in this specific clinical population, as indicated by our results.

Our results are similar with findings from Geronimi et al. (2016) and Yang et al. (2017), where increased worry and rumination were associated with decreased EF. Particularly, our findings regarding rumination's stronger association with executive dysfunction offer new insights into the comorbidity of anxiety and depression disorders.

The stronger link between rumination and executive dysfunction compared to worry may be attributed to the measurement tools used. The BAI scale's emphasis on psycho-physiological symptoms of anxiety might have contributed to the underrepresentation of cognitive components like worry in our findings. This observation is important in understanding the nuances of how anxiety and depression manifest and are measured, aligning with Cox et al. (1996), who criticized BAI for its emphasis on panic-related symptoms.

Limits and further directions

While our findings offer valuable insights, they are not without limitations. The reliance on self-report scales introduces the possibility of common method bias and negative cognitive bias, as participants might lean towards more negative self-assessments. This represents a critical limitation of our study and suggests the need for future research incorporating performance tests to gain a more accurate picture of the relationships between executive functions and mood disorders. Also, the absence of a non-clinical comparative sample limits our ability to generalize the EF differences observed. Comorbidities, both psychiatric and somatic, in our sample might have influenced EF assessments, necessitating further research to untangle these complex relationships. The predominance of female participants in our study reflects the demographic patterns of these disorders but limits the generalizability of findings to a male population. The cross-sectional nature of the study constrains our understanding of the causal dynamics of these relationships. Longitudinal studies would provide a clearer picture of how these interactions unfold over time.

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