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Ph.D. THESIS SUMMARY

THE ROLE OF EXECUTIVE FUNCTIONS IN DEPRESSION, ANXIETY AND ALCOHOL DEPENDENCE

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*Notes.*_____

(1) This is to certify by Dana-Andreea Iclozan that:

(a) The thesis includes the original research work of Dana-Andreea Iclozan (author) towards the Ph.D.; the research was scientifically supervised by University Professor Dr. Mircea Miclea;

(b) Parts of the thesis have been submitted for publication.

(c) The thesis was written according to the academic writing standards. All the text of the thesis and its summary was written by Dana-Andreea Iclozan who assumes the all responsibility for the academic writing;

The results of the present thesis are in process of publication as follows:

Articles that are under review:

- **Iclozan, D.-A.** & Pop, I.-F. (under review). Executive Functions in Clinical and Non-Clinical Populations. A Comparative Analysis.
- **Iclozan, D.-A**. & Florean, I. S. (under review). The Relationship Between Worry, Rumination, And Executive Functions in Clinical Population.

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1. THEORETICAL BACKGROUND

Relevance and Impact of the Research Topic

Deficits in executive functioning (EF) are present in many psychiatric disorders, and together with repetitive negative thinking, they represent core features and correlates of psychopathology. This paper aims to analyze EF in the clinical population and compare them with EF in individuals without psychopathology. It also focuses on the associations between cognitive vulnerabilities, such as worry, rumination, and executive functioning in individuals with psychopathology.

From a clinical perspective, it has been observed that many psychiatric disorders exhibit EF deficits. Understanding these deficits and how they affect the individual, particularly the patient in a clinical context, can help develop more effective and specific intervention strategies for each disorder.

Repetitive negative thinking is a dysfunctional thought pattern often studied in individuals with psychopathology, being significant mechanisms that maintain or prolong the impairment of individuals with a clinical diagnosis. Understanding how repetitive negative thinking influences the emotional state and behavior of patients assists in addressing these thought patterns in clinical intervention. Understanding the relationship between EF and repetitive negative thinking can provide important insights into the cognitive mechanisms involved in the development and maintenance of disorders with high prevalence in the clinical context, such as anxiety, depression, or alcohol dependence.

Working as a clinical psychologist in a clinical setting, I have often faced the complicated task of discerning whether the observed deficits in patients' EF are epiphenomena of their mood states or, conversely, reflect a broader cognitive disorder. It is also essential to consider the clinical context, as well as other factors that may influence cognitive performance, such as medication or other somatic comorbidities.

Anxiety, depression, and alcohol dependency are the most frequently diagnosed disorders at the psychiatric clinic where I work, an open clinic. This means that patients are treated in a less restricted environment than in a closed psychiatric ward, they have some awareness of their illness, and they are willing to seek help to recover. They can make decisions about their

treatment and can be involved in its planning, which can enhance their motivation and commitment to the recovery process. EF is closely linked to a person's ability to make decisions, studies showing that EF impairment can have a deeper effect on a person's autonomy than memory impairment (Schillerstrom, Horton, & Royall, 2005).

Therefore, the high prevalence of these mental disorders in psychiatric hospitals makes their study important to better understand the causes and treatment of these conditions with an impact on the disease's course. The proper treatment of these mental disorders can also have a positive impact on associated medical comorbidities.

In conclusion:

1. EF deficits are a significant factor in the etiology and course of mental disorders. They, along with repetitive negative thinking, are involved in many psychiatric disorders, and understanding them can help develop specific intervention strategies for each disorder, more effective and personalized therapeutic interventions.
2. The study of anxiety, depression, and alcohol dependence is essential as they are among the most frequently diagnosed disorders in psychiatric hospitals, and improving the assessment and treatment of EF deficits can have a significant impact.
3. The appropriate assessment and treatment of EF deficits are important for improving the patient's overall functioning and autonomy, disease course, and compliance with treatment.
4. There is a knowledge deficit about EF deficits in hospitalized patients due to a lack of standardized assessment methods, and about how EF can be affected by medical treatments and what the course is after discharge. Routine screening could be a useful method for monitoring this deficit, and overall functionality for this category of individuals.

EF refers to a set of self-regulatory processes that help us achieve our goals, often in a social context (Barkley, 2012). The concept of EF is a meta-construct involving several modules or mental capacities that interact and contribute together to EF. Dysfunctions in one module can affect EF in different ways than dysfunctions in other modules, underscoring the complexity and multifactorial nature of these cognitive functions.

According to Barkley (2014), the components of EF include self-directed attention (shifting), inhibition, working memory (nonverbal - especially visual images and verbal working memory), planning, and problem-solving. It can be noted that the most common constructs

attributed to the term EF are all forms of self-directed actions, meant to modify subsequent behavior and thus effect a change in the likely future (delayed consequences) (Cowan, 1999).

Several researchers (Miyake et al., 2000; Diamond, 2013) highlight the three fundamental components of EF, namely updating (working memory), inhibition, and shifting (attentional switching). Differences between taxonomies can be subtle and often refer to the emphasis placed on different aspects of these components or the way they are described. However, essentially, all definitions acknowledge the importance of these three components within the EF construct and their role in regulating behavior and cognitive processes to allow adaptation and performance in various situations. Generally, these definitions are compatible and convergent.

According to these taxonomies, there are three basic components included in the EF construct (Miyake et al., 2000; Diamond, 2013): working memory, inhibition or inhibitory control (behavioral, emotional, and cognitive inhibition), and cognitive flexibility. From these higher-order EFs derive second-order EFs, which include reasoning, problem-solving, and planning.

Generally, the literature supports that there are several factors that can negatively impact EF, including specific psychological disorders, neurochemical mechanisms, genetic causes, brain injuries, and substance use, among others. These EF deficits can lead to a wide variety of negative consequences. However, the neuropsychological study of the dysexecutive syndrome and appropriate rehabilitation faces inherent difficulties. One of them is the accurate and valid assessment of EF (Chan, Shum, Toulopoulou, & Chen, 2008).

The most common executive dysfunction issues reported in a neurological group with mixed etiology were: planning problems, distractibility, lack of insight, decision-making difficulties, social disinterest, euphoria, restlessness, apathy, disregard for others' feelings, perseverance, aggression, problems with temporal sequencing, social disinhibition, superficial affect, impulsivity, difficulties in inhibiting the response, difficulties in abstract thinking, dissociation between knowing and doing, confabulations (Burgess & Robertson, 2002).

EFs are vulnerable to a wide range of neurological, psychiatric, and medical processes, including many reversible or treatable conditions. The treatment of executive dysfunction needs to be adapted to the individual patient, often requiring a combination of disease-specific pharmacological treatments, occupational therapy, and cognitive rehabilitation (Rabinovici et al., 2015).

On the other hand, deficits in executive functioning that seem fairly mild in a testing environment can nonetheless have a more severe effect on functioning in everyday life. Moreover, they can interfere with the progress of medical treatment. For this reason, assessing and treating executive deficits should be a priority for neurorehabilitation (Burgess & Alderman, 2004).

In conclusion, there is a wide range of factors that can negatively impact EF, including specific psychological disorders, neurochemical mechanisms, genetic factors, brain injuries, and substance use. Deficits in EF can lead to a series of negative consequences on the quality of life, including the ability to work and attend school, to function independently at home, or to develop and maintain appropriate social relationships. In general, assessing and treating executive deficits and dysexecutive syndromes requires an understanding of the components of EF, the neuroanatomy underlying them, and differential diagnosis.

Studies have shown that EFs are particularly affected in patients with depression. The existence and nature of the EF deficit associated with depression remain heavily debated. While many studies have found significant deficits associated with depression regarding neuropsychological measures of EF, others could not demonstrate these deficits due to low statistical power, task impurity, and various patient samples (Snyder, 2013).

Moreover, the effectiveness of cognitive-behavioral therapy, to some extent, depends on the patient's EF abilities (Mohlman & Gorman, 2005). The EF deficit is a transdiagnostic risk factor for psychopathology (Snyder, 2013).

Executive dysfunction is also associated with anxiety. Snyder (2013) suggests that executive dysfunction may be a risk factor for the development of anxiety, although the relationship between these two variables may be bidirectional and complex.

Executive dysfunction is also the most common cognitive impairment among patients with alcohol addiction (Ihara, Berrios, & London, 2000). It is well documented that there is significant brain damage in these patients (Harrison et al., 2017). Macrostructural changes have been observed at the level of the pons, cerebellum, and midbrain (Sullivan, 2003; Chanraud et al., 2007), which were linked to executive deficits, along with frontal lobe impairment.

Studies have shown that the deficit in EF could be a risk factor for the occurrence of alcohol use disorders (Finn, Justus, Mazas, and Steinmetz, 1999), individuals with such a deficit

encounter problems due to consumption (Nigg et al., 2006) and have limited benefit from treatment (Bates, 2000).

Deficits in EF and rumination, as a model of repetitive negative thinking, have been associated transdiagnostically in psychopathology (du Pont, Rhee, Corley, Hewitt, and Friedman, 2019). Some researchers argue that rumination disrupts EF processes, leading to deficits in recalling autobiographical memory events and impaired cognitive processes (Ramponi, Barnard, & Nimmo-Smith, 2004), while others argue that the executive deficit underlies rumination, leading to the inability to inhibit and deactivate negative, self-centered information (Koster, De Lissnyder, Derakshan, & De Raedt, 2011; van Vugt et al., 2018).

2. OBJECTIVES AND GENERAL METHODOLOGY

The motivation of this thesis is closely linked to the need to more deeply understand the specific mechanisms that contribute to EF deficits in psychopathology. It is clear that understanding these mechanisms is important for directing our knowledge towards translational applications that can create effective therapeutic interventions. Also, a key element that led us to this research is the fact that seemingly similar deficits in EF might have different mechanisms at their base.

From the clinical experience gained during the activity carried out in the psychiatry hospital, we directly observed the significant impact that EF deficits, emotions, and dysfunctional thinking modes have on patients with depression, anxiety, and excessive alcohol consumption. These deficits seem to contribute significantly to the difficulties encountered by these patients, difficulties in planning and executing daily tasks, in emotional and behavioral regulation, in achieving goals. These difficulties can contribute to maintaining a vicious cycle of dysfunction. These clinical findings, together with the need for a deeper understanding of the mechanisms associated with EF deficits, form the main motivation of this thesis.

The objectives of this doctoral thesis focus on an integrative approach, meant to explore and better clarify the relationship between EF, emotions, and repetitive negative thinking in the context of the most prevalent psychiatric disorders in the clinical context.

A central and distinctive aspect of this doctoral thesis is that all research is carried out in an ecological context, directly with patients admitted to the psychiatric hospital. This means that all observations, collected data, and proposed interventions are based on the real experience of patients living and dealing with these disorders in hospitalization conditions, an ecological approach.

We aimed to explore and evaluate the concordance between the specific clinical difficulties of patients with psychiatric disorders and what is reported in the specialized literature. Specifically, we analyzed to what extent the academic literature reflects the real, most common experiences and problems faced by these patients, that is, EF deficits, maladaptive emotions such as shame and guilt, and the tendency towards rumination of people with depression, anxiety. The second objective is to understand the mechanisms underlying EF deficits and the relationship between them. Finally, we proposed an intervention plan adapted to

the specific context of ecology. This means that, based on the obtained results, we propose an intervention plan that is designed in such a way as to be practical, applicable, and efficient in the real context of hospital life, considering the peculiarities, constraints, and resources of this environment. This plan is designed to be implemented and to be as efficient as possible. Currently, in Romania, there is a lack of specific interventions. Therefore, this research can provide a replicable model of good practice by developing effective intervention strategies well adapted to their specific patient needs.

In the first study (Figure 1), we comparatively analyzed EF in clinical and non-clinical populations, using computerized tests and performance scales. We extended this analysis by examining EF more deeply among patients with depression and alcohol addiction, to try to better understand these processes in hospitalization contexts.

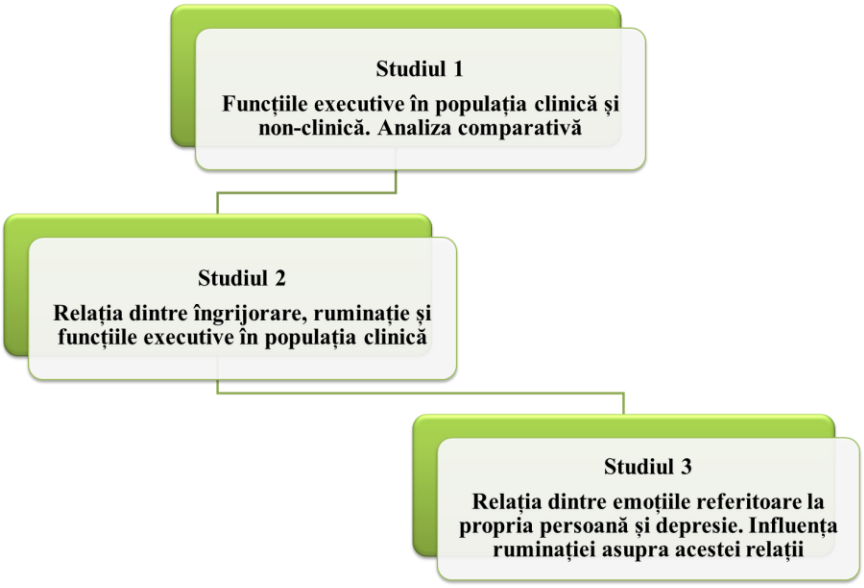

Figure 1. The Schematic Structure of the Thesis

In the second study, we turned our attention to the relationship between EF and repetitive thinking, exploring the connection between EF and worry, respectively rumination in patients with anxiety and depression. The aim was to better understand the relationship between these variables and how they can influence symptoms of anxiety and depression.

In the third study, we focused on the link between self-referential emotions (shame, guilt) and depression. Here we aimed to understand the role that rumination plays in this relationship, investigating whether it moderates the connection between shame, guilt, and depression.

Study participants were recruited from patients with a formal diagnosis, hospitalized and under medication. They were informed about the nature and purpose of the research, including the fact that their participation is voluntary and that they can withdraw at any time without suffering any consequences. All necessary measures were taken to ensure the confidentiality and protection of the participants' personal data. Also, we considered the cognitive capacity of the participants at the time of recruitment, ensured that they were not subjected to unnecessary stress during the assessments, and that they received the necessary support during their participation. All these aspects were considered in order to respect the ethical principles of research in the field of mental health.

3. ORIGINAL RESEARCH

3.1 Executive Functions in Clinical and Non-Clinical Populations. A Comparative Analysis. (*Study 1*)

3.1.1 Introduction

Operationalizing and measuring EF is a key issue that directly impacts the inferences we can make about these skills. Procedures used in studies to operationalize EF in clinical settings use performance-based or evaluation measurements.

Conventional EF measurement has relied on cognitive performance tests (Pennington & Ozonoff, 1996), administered under highly standardized conditions, and based on response accuracy, response time, and/or quick response under time constraint (Toplak, West & Stanovich, 2013).

Self-report scales provide a valid ecological indicator of EF in complex, everyday problem-solving situations (Roth, Isquith & Gioia, 2005). The assumption behind the use of these self-report scales is that they measure behaviors related to processes that are evaluated through EF performance tests (Toplak, West & Stanovich, 2013).

If computerized tests and self-reporting tests evaluate the same general construct, then these measures should show a strong and positive correlation. Toplak and colleagues (2013) conducted a literature analysis on the correlation between the two types of evaluations and concluded that they are only minimally correlated. The authors argue that the two types of measurements evaluate different aspects of cognitive and behavioral functioning, which independently contribute to clinical problems.

Numerous other studies have shown that the relationship between self-reported questionnaires and objective, performance tests of EF is weak or nonexistent (Bogod, Mateer & MacDonald, 2003; Burgess, Alderman, Evans, Emslie & Wilson, 1998; Nęcka, Lech, Sobczyk & Śmieja, 2012; Wingo, Kalkut, Tuminello, AsConape, & Han, 2013). This has been demonstrated for both healthy adults (Wingo et al., 2013; Nęcka et al., 2012) and clinical populations (Bogod et al., 2003; Burgess et al., 1998). Factors that may contribute to this lack of association include

differences in abilities measured by these methods, self-reports influenced by personality (Goldberg, 2017).

Difficulties in EF have been implicated in several neurological and psychiatric conditions, such as traumatic brain injuries (Clark, Manes, Antoun, Sahakian & Robbins, 2003; Labudda et al., 2009); schizophrenia (Cavallaro et al., 2003; Kester et al., 2006; Nakamura et al., 2008); substance use (Barry & Petry, 2008; Ernst et al., 2003); obsessive-compulsive disorder (Lawrence et al., 2006); psychopathy (Mahmut, Homewood & Stevenson, 2008); ADHD (Toplak, Jain & Tannock, 2005); pathological gambling (Toplak, Liu, MacPherson, Toneatto & Stanovich, 2007).

Pimontel et al. (2016) showed that decreased planning and organization were associated with a lower response to antidepressant treatment. Clinicians might anticipate the likely outcomes of treatment and decide on subsequent therapeutic models (McLennan & Mathias, 2010).

Considering the results of previous studies on clinical and non-clinical populations, the main objective of the study is to evaluate EF through performance tests and self-evaluation scale in patients with psychopathology, in an ecological environment, and compare these results with the non-clinical population. We also aimed to evaluate and compare EF in groups of people with depression and alcohol dependency (the main two pathologies in the clinical group) using both a performance test and a self-assessment scale, to determine if there is a significant difference between the two groups and to comparatively evaluate and analyze the manifestation of the executive deficit between the two groups.

3.1.2 Method

Participants

The participants in the clinical group (n=65) were recruited from the Psychiatry Clinic of the Municipal Clinical Hospital Cluj-Napoca, being hospitalized. The clinical group - N1=65 (32 women (49.2%) and 33 men (50.8%) aged between 23 and 73 years, m=51.25 years, diagnosed with recurrent depressive disorder, alcohol dependence, generalized anxiety disorder, bipolar affective disorder, somatization disorder, organic delusional disorder. The non-clinical group -

N2=65 (32 women (49.2%) and 33 men (50.8%) aged between 24 and 75 years, m= 50.55 years, without psychiatric history and without a current diagnosis.

Instruments

Demographic data such as age, educational level, marital status, level of schooling, occupation, rural/urban environment, socio-economic status. Medical data were also collected: treatment for various somatic diseases, neurological or neurocognitive diagnosis, psychiatric diagnosis, medication, substance use.

EF by self-evaluation scale

The "*Barkley Deficits in Executive Functioning Scale*" (BDEFS-LF), developed by Russell A. Barkley and published by Guilford in 2011, a well-founded theoretical and empirical instrument, organized on several sub-domains or factors (Barkley et al., 2022): self-management in relation to time, self-organization/problem-solving, self-control, self-motivation, self-regulation of emotion, ADHD index, and symptom number. The BDEFS scale has good psychometric properties, Barkley (2011) reporting a Cronbach's alpha coefficient >0.91.

EF by performance tests

The *Corsi* Test (Traditional Corsi Block-Tapping Test, t-Corsi), developed by Philip Michael Corsi (1972), measures visuospatial working memory (Kessels et al., 2010). The task used in the study was developed by Cognitrom company based on validated specifications from the specialized literature (Berch, Krikorian & Huha, 1998).

STROOP Test (TIC-S)

The classic test designed by Stroop (1935) is a widely used neuropsychological test that assesses the ability to inhibit cognitive interference that occurs when processing a feature of a stimulus affects the simultaneous processing of another attribute of the same stimulus, i.e., the ability to inhibit competing responses in the presence of obvious contradictory information (Scarpina & Tagini, 2017). The task used in the study was developed by the Cognitrom company.

Choice RT - RTC Test

RT (reaction time) was defined as the time in milliseconds from stimulus presentation to a recorded response. The task used in the study checked how quickly the tested person responds in a choice task between two alternatives..

3.1.3 Results

Table 1 presents the means and standard deviations, minimum and maximum values on the Corsi sub-scales and for the total EF score (for BDEFS-SF) within the non-clinical group.

Table 1. Means and standard deviations for Corsi and BDEFS in the non-clinical group.

	n_1	Min.	Max.	Mean	SD
Age	65	24.00	75.00	50.7538	9.88059
Corsi scor final	65	2.50	7.50	5.4846	1.05315
Corsi fwd. span	65	2.00	9.00	5.6769	1.27607
Corsi bkwd. span	65	3.00	8.00	5.2923	1.05657
BDEFS SF total	65	20.00	47.00	28.7077	7.57406
n_1	65				

Table 2 presents the means and standard deviations, minimum and maximum values on the Corsi subscales and for the total EF score (for BDEFS-SF) within the clinical group.

Table 2. *M*eans and standard deviations for Corsi and BDEFS in the clinical group

	n_2	Min.	Max.	Mean	SD
Age	65	23.00	73.00	51.2462	9.82190
Corsi scor final	65	.00	6.00	3.8385	1.44727
Corsi fwd. span	65	.00	7.00	4.0308	1.35749
Corsi bkwd. span	65	.00	6.00	3.6462	1.89102
BDEFS SF total	65	20.00	66.00	40.4110	12.16588
n_2	65				

The descriptive analysis of the specific data for the two groups (clinical vs non-clinical) is presented in Table 3.

Tabel 3. The descriptive analysis of EF tests on the two groups: clinical vs non-clinical.

		N	Mean	SD	SE	95% CI		Min.	Max.
						LL	UL		
Corsi final score	non-clinical	65	5.48	1.05	0.131	5.2237	5.7456	2.50	7.50
	clinical	65	3.84	1.45	0.180	3.4798	4.1971	0.00	6.00
Corsi fwd span	non-clinical	65	5.68	1.28	0.158	5.3607	5.9931	2.00	9.00
	clinical	65	4.03	1.36	0.168	3.6944	4.3671	0.00	7.00
Corsi bkwd span	non-clinical	65	5.29	1.06	0.131	5.0305	5.5541	3.00	8.00
	clinical	65	3.65	1.89	0.235	3.1776	4.1147	0.00	6.00

BDEFS total	non-clinical	65	28.71	7.57	0.939	26.83	30.58	20	47
	clinical	65	40.41	12.17	1.509	37.40	43.43	20	66
BDEFS symp count	non-clinical	65	1.35	2.27	0.281	0.79	1.92	0	9
	clinical	65	5.58	5.40	0.670	4.25	6.92	0	18

Note. BDEFS total, Total EF score; BDEFS sympt count, symptom count scale; Corsi fwd span, Corsi forward subscale, Corsi bkwd span, Corsi backward subscale; Corsi final score, total Corsi score; SD=standard deviation, SE=standard error, CI=confidence interval, LL=lower limit, UL=upper limit.

There was a significant effect of psychopathology on: the final Corsi score at p<.05 level [F(1, 128) = 54.98, p <0.001]; the Corsi forward score [F(1, 128) = 50.74, p <0.001]; the Corsi backward score [F(1, 128) = 37.54, p <0.001]; the total BDEFS score [F(1, 128) = 43.35, p <0.001]; and on the symptom count scale [F(1, 128) = 33.90, p <0.001] (Table 4).

Table 4. ANOVA analysis.

		df	Mean Square	F	*p*.
Corsi final score	**Between Groups**	**1**	**88,069**	**54,98**	**<0,001**
	Within Groups	128	1,602		
	Total	129			
corsi fwd span	**Between Groups**	**1**	**88,069**	**50,74**	**<0,001**
	Within Groups	128	1,736		
	Total	129			
corsi bkwd span	**Between Groups**	**1**	**88,069**	**37,54**	**<0,001**
	Within Groups	128	2,346		
	Total	129			
BDEFS total	**Between Groups**	**1**	**4451,406**	**43,35**	**<0,001**
	Within Groups	128	102,687		
	Total	129			
BDEFS symp count	**Between Groups**	**1**	**581,731**	**33,90**	**<0,001**
	Within Groups	128	17,161		
	Total	129			

Moving forward, we excluded from the analysis those participants diagnosed with the following conditions: generalized anxiety disorder, bipolar affective disorder - manic episode, somatoform disorder, and organic delusional disorder. This was due to the small number of participants falling into these diagnostic categories.

Descriptive statistics related to the executive functioning tests (BDEFS and CORSI) performance across the three groups are displayed in Table 5. The minimum and maximum scores observed range between 2 and 9 for the Corsi test, and 20 to 66 for the BDEFS.

Table 5. Descriptive statistics associated with performance on EF tests.

		Mean	SD	SE	95% CI		Min.	Max.
					LL	UL		
Corsi final score	non-clinical	5,48	1,05	0,131	5,2237	5,7456	2,50	7,50
	alcohol	3,53	1,72	0,314	2,8904	4,1762	0,00	6,00
	depression	4,35	0,87	0,170	3,9951	4,6972	2,00	6,00
Corsi fwd span	non-clinical	5,68	1,28	0,158	5,3607	5,9931	2,00	9,00
	alcohol	3,77	1,70	0,310	3,1336	4,3998	0,00	7,00
	depression	4,35	0,75	0,146	4,0451	4,6472	3,00	6,00
Corsi bkwd span	non-clinical	5,29	1,06	0,131	5,0305	5,5541	3,00	8,00
	alcohol	3,30	2,04	0,372	2,5395	4,0605	0,00	6,00
	depression	4,35	1,29	0,254	3,8233	4,8690	0,00	6,00
BDEFS total	non-clinical	28,71	7,57	0,939	26,83	30,58	20	47
	alcohol	36,36	11,71	2,138	31,99	40,73	20	61
	depression	43,96	11,42	2,239	39,35	48,58	21	66
BDEFS symp count	non-clinical	1,35	2,27	0,281	0,79	1,92	0	9
	alcohol	3,70	4,46	0,814	2,04	5,36	0	14
	depression	7,12	5,54	1,087	4,88	9,36	0	18

Note. BDEFS total, total EF score; BDEFS sympt count, symptom count scale; Corsi fwd span, Corsi forward subscale, Corsi bkwd span, Corsi backward subscale; Corsi final score, total Corsi score; SD=standard deviation, SE=standard error, CI=confidence interval, LL=lower limit, UL=upper limit.

To assess the nature of the observed differences among the three groups at the two types of EF evaluations, the ANOVA analysis was followed by post-hoc comparisons using the Games-Howell test (Table 6).

Table 6. Post-hoc analysis, the Games-Howell test for multiple comparisons.

Dependent variable	a	b	Mean difference (a-b)	SE	*p.*	95% CI	
						LL	UL
corsi final score	non-clinical	alcohol	1.951*	0.340	**<0,001**	1.1222	2.7803
		depression	1.138*	0.215	**<0,001**	0.6213	1.6556
	alcohol	non-clinical	-1.951*	0.340	**<0,001**	-2.7803	-1.1222
		depression	-0.813	0.358	.070	-1.6800	0.0544
	depression	non-clinical	-1.138*	0.215	**<0,001**	-1.6556	-0.6213
		alcohol	0.813	0.358	.070	-0.0544	1.6800
corsi fwd span	non-clinical	alcohol	1.910*	0.348	**<0,001**	1.0675	2.7530

		depresie	1.331*	0.215	**<0,001**	0.8159	1.8457
	alcohol	non-clinical	-1.910*	0.348	**<0,001**	-2.7530	-1.0675
		depression	-0.579	0.342	.220	-1.4119	0.2529
	depression	non-clinical	-1.331*	0.215	**<0,001**	-1.8457	-0.8159
		alcohol	0.579	0.342	.220	-0.2529	1.4119
corsi bkwd span	non-clinical	alcohol	1.992*	0.394	**<0,001**	1.0290	2.9556
		depression	0.946*	0.286	.006	0.2502	1.6421
	alcohol	non-clinical	-1.992*	0.394	**<0,001**	-2.9556	-1.0290
		depression	-1.046	0.450	.062	-2.1338	0.0415
	depression	non-clinical	-.946*	0.286	**.006**	-1.6421	-0.2502
		alcohol	1.046	0.450	.062	-0.0415	2.1338
BDEFS total	non-clinical	alcohol	-7.654*	2.335	**.006**	-13.33	-1.97
		depresie	-15.256*	2.428	**<0,001**	-21.21	-9.31
	alcohol	non-clinical	7.654*	2.335	**.006**	1.97	13.33
		depression	-7.603*	3.096	.045	-15.07	-0.14
	depression	non-clinic	15.256*	2.428	**<0,001**	9.31	21.21
		alcohol	7.603*	3.096	.045	0.14	15.07
BDEFS symp count	non-clinical	alcohol	-2.346*	0.861	.026	-4.45	-0.24
		depresie	-5.762*	1.123	**<0,001**	-8.54	-2.98
	alcohol	non-clinical	2.346*	0.861	**.026**	0.24	4.45
		depression	-3.415*	1.358	**.040**	-6.70	-0.13
	depression	non-clinical	5.762*	1.123	**<0,001**	2.98	8.54
		alcohol	3.415*	1.358	**.040**	0.13	6.70

Note: Corsi fwd span, Corsi forward subscale; Corsi bkwd span, Corsi backward subscale; Corsi final score, total Corsi score; BDEFS total, total EF score; BDEFS symp count, symptom count scale; * mean difference is significant at the 0.05 level.

Pairwise comparisons of the means using the Games-Howell post-hoc test indicated significant comparisons.

To evaluate EF more granularly, we decided to include only participants with alcohol dependence disorders and depression in the study. Descriptive statistics for the Corsi, Stroop, and RT choice tests are presented in Table 7.

Table 7. Descriptive analysis of the Corsi, Stroop, and RT choice tests for the clinical population.

		N	Mean	SD	SE	95% CI		Min.	Max.
						LL	UL		
Corsi final score	alcohol	30	3.5	1.7	0.314	2.890	4.176	0.0	6.0
	depression	26	4.3	0.9	0.170	3.995	4.697	2.0	6.0
	Total	56	3.9	1.4	0.192	3.525	4.296	0.0	6.0
Stroop final score	alcohol	30	15.1	6.5	1.187	12.683	17.540	2.884	30.034
	depression	25	16.8	8.1	1.615	13.450	20.115	5.049	37.267
	Total	55	15.9	7.2	0.976	13.914	17.828	2.884	37.267
Rt choice final score	alcohol	30	695.1	157.5	28.749	636.316	753.913	499.991	1101.857
	depression	26	705.8	170.8	33.504	636.798	774.802	381.889	1089.803
	Total	56	700.1	162.4	21.700	656.589	743.562	381.889	1101.857

Note. Corsi final score, total Corsi score; Stroop final score, total Stroop score; RT choice final score, total RT choice task score; SD=standard deviation, SE=standard error, CI=confidence interval, LL=lower limit, UL=upper limit.

The analysis of variance shows a statistically significant effect only for the total Corsi score [F(1, 56) = 4.74, p =.035]. No statistically significant effect was found for the total Stroop score and RT Choice (Table 8).

Tabel 8. Analiza de varianță ANOVA la testele Corsi, Stroop și RT choice pentru populația clinică

		df	Mean Square	F	Sig.
Corsi final score	Between Groups	1	9.202	**4.74**	**0.034**
	Within Groups	54	1.942		
	Total	55			
Stroop final score	Between Groups	1	38.070	0.72	0.399
	Within Groups	54	52.657		
	Total	55			
Rtchoice final score	Between Groups	1	1590.194	0.06	0.809
	Within Groups	54	26827.597		
	Total	55			

Descriptive statistics of the BDEFS test subscales for the two categories of patients are presented in Table 9. By analyzing the mean values, we observe that on all measured dimensions, patients with depression have obtained higher average scores than patients with alcohol dependence.

Table 9. The results of the BDEFS test subscales for the two categories of patients in the clinical population.

		N	Mean	SD	SE	95% CI		Min.	Max.
						LL	UL		
Self-organisation	alcohol	30	37.4	13.1	2.398	32.487	42.295	21.00	71.00
	depression	26	45.7	15.0	2.943	39.589	51.714	21.00	79.00
	total	56	41.2	14.5	1.939	37.340	45.113	21.00	79.00
Time mgmt.	alcohol	30	45.4	16.9	3.087	39.092	51.721	24.00	82.00
	depression	26	57.3	16.3	3.202	50.706	63.894	25.00	91.00
	total	56	50.9	17.5	2.344	46.230	55.626	24.00	91.00
Self-mgmt.	alcohol	30	36.8	11.6	2.116	32.450	41.103	19.00	60.00
	depression	26	40.0	9.4	1.838	36.215	43.786	21.00	60.00
	total	56	38.3	10.6	1.423	35.422	41.125	19.00	60.00
Self-motivation	alcohol	30	19.4	6.3	1.154	17.072	21.794	12.00	33.00
	depression	26	21.7	8.1	1.582	18.427	24.943	12.00	38.00
	total	56	20.5	7.2	0.963	18.549	22.409	12.00	38.00
Emotion regulation	alcohol	30	26.5	8.5	1.545	23.329	29.647	13.00	41.34
	depression	26	31.7	8.6	1.690	28.182	35.142	14.00	46.00
	total	56	28.9	8.8	1.182	26.521	31.259	13.00	46.00
FE total score	alcohol	30	165.5	52.1	9.510	146.046	184.945	89.00	279.00
	depression	26	196.3	47.4	9.302	177.140	215.457	93.00	296.00
	total	56	179.8	51.9	6.935	165.898	193.696	89.00	296.00
ADHD Index	alcohol	30	19.0	6.2	1.130	16.729	21.350	11.00	34.00
	depression	26	22.5	6.0	1.180	20.064	24.926	12.00	36.00
	total	56	20.6	6.3	0.842	18.957	22.331	11.00	36.00
FE symptoms	alcohol	30	18.5	20.7	3.784	10.727	26.207	0.00	71.00
	depression	26	31.4	22.1	4.325	22.516	40.330	0.00	76.00
	total	56	24.5	22.1	2.958	18.553	30.411	0.00	76.00

Note. Self-organization, Self-organization/Problem-solving subscale; Time Mgmt., Self-management in relation to time subscale; Emotion Regulation, Self-regulation of emotion subscale; Total EF, Total EF score; EF symptoms, Symptom count scale, ADHD Index, ADHD symptom scale.

We conducted an analysis of variance (ANOVA) to evaluate the impact of the diagnosis on the different BDEFS subscales, according to Table 10.

Table 10. Analiza de varianță ANOVA a subscalelor testului BDEFS pentru populația clinică.

		df	Mean Square	F	Sig.
Self-organisation	Between Groups	1	950.318	**4.83**	**0.032**
	Within Groups	54	196.912		
	Total	55			

Time mgmt.	Between Groups	1	1970.282	**7.11**	**0.010**
	Within Groups	54	276.981		
	Total	55			
Self-control	Between Groups	1	144.773	1.28	0.262
	Within Groups	54	112.784		
	Total	55			
Self-motivation	Between Groups	1	70.616	**1.37**	**0.247**
	Within Groups	54	51.597		
	Total	55			
Emotion regulation	Between Groups	1	372.894	**5.12**	**0.028**
	Within Groups	54	72.802		
	Total	55			
FE total score	Between Groups	1	13216.039	**5.29**	**0.025**
	Within Groups	55	2498.624		
	Total	55			
ADHD Index	Between Groups	1	166.294	**4.46**	**0.039**
	Within Groups	54	37.325		
	Total	55			
FE symptoms	Between Groups	1	2338.169	**5.13**	**0.028**
	Within Groups	54	455.885		
	Total	55			

We evaluated the Spearman correlation coefficient to analyze the relationships between different variables within our total sample of mixed psychopathology, according to Table 11.

Table 11. Spearman bivariate correlation coefficients between EF tests in the clinical population.

	1	2	3
1. BDEFS total			
2. Corsi final score	-.117		
3. Stroop final score	**-.363****	**.338****	
4. Rt choice final score	**.294***	**-.262***	**-.442****

Note: **. The correlation is significant at the $p < 0.001$ level
*. The correlation is significant at the $p < 0.05$ level

We further compared the scores of the two gender groups within the clinical group on the BDEFS and CORSI subscales through a one-way analysis of variance (ANOVA); the results are presented in Table 12 below.

Table 12. Comparisons between women and men on the BDEFS and CORSI subscales.

	Women		Men			
Scor	Mean	*SD*	Mean	*SD*	*F*	*p*
Self-organization	46,9	14,5	36,6	12,5	9,27	**0,003**
Time Mgmt	60,4	17,4	44,2	15,9	15,14	**<0,001**
Self-control	40,9	10,0	35,7	10,6	4,07	**0,048**
Self-motivation	21,8	7,6	18,6	5,9	3,56	0,064
Emotion Regulation	33,5	9,0	25,6	7,7	14,09	**<0,001**
FE symptoms	34,6	22,3	16,6	19,0	12,13	**0,001**
FE total	203,5	48,2	160,8	48,2	12,58	**0,001**
Corsi fwd span	4,0	1,2	4,0	1,5	0,00	NS
Corsi bkwd span	3,5	1,8	3,8	2,0	0,33	NS
Corsi scor final	3,8	1,3	3,9	1,6	0,14	NS

Note. Self-organization, Self-organization/Problem-solving subscale; Time Mgmt., Self-management in relation to time subscale; Emotion Regulation, Self-regulation of emotion subscale; Total EF, Total EF score; EF symptoms, Symptom count scale; Corsi fwd span, Corsi forward subscale, Corsi bkwd span, Corsi backward subscale; Corsi final score, total Corsi score; F, results for F tests from variance analysis; p, probability value for the F test if ≤ .05; NS, not significant. The two gender categories were as follows: women (n = 32); men (n = 33).

3.1.4 Discussions and Conclusions

Assessing EF in clinical samples, under conditions of hospitalization and medication, is quite a challenging task as it can involve resistance from patients, task evasion, insufficient involvement, low self-efficacy due to lack of previous computer interaction, all of which are variables that can influence actual performance. To our knowledge, there are no studies comparing EF in people with depression and alcohol dependence.

The conventional way of measuring EF has relied on performance-based tests, which are administered under extremely standardized conditions. Self-report EF scales have been developed to provide a valid ecological indicator of competence in complex, everyday problem-solving situations. The results of studies so far have revealed a surprising lack of association between performance-based EF assessments and self-report ones (Toplak, West, & Stanovich, 2013). In line with previous studies, we found a similar partial discrepancy within the study, partially confirming a lack of association between performance-based EF evaluations and self-report ones (between Corsi and BDEFS). Other computerized tests (Stroop and RT Choice) correlated with BDEFS, showing a significant correlation between the computerized tasks and the self-reported one. Although both types of measurements should correlate because they measure the same construct, a basic principle of convergent validity, this did not happen for all

performance-based measurements. Therefore, these two classes of assessments should not be interpreted as equivalent.

The results obtained confirmed the hypotheses, namely the fact that people with depression have significantly poorer performances compared to control groups. Evidence from meta-analyses shows that people with depression are significantly affected, on all EF components - shifting, inhibition, updating (working memory) (Snyder et al., 2015). As expected, patients in the clinical group performed lower than those in the non-clinical group, both in performance tests and in the self-assessment EF scale. However, there is a big difference between the two groups in terms of education level. 87.8% of the non-clinical group have higher education, compared to only 13.8% in the clinical group. A higher level of education has been repeatedly associated with better performances in cognitive tests, including those measuring executive functions.

A 2021 meta-analysis (Nuño, Gómez-Benito, Carmona, & Pino, 2021) shows a clear performance deficit among people with depression regarding tasks measuring EF. Godard, Grondin, Baruch, and Lafleur (2011) found heterogeneity regarding the nature and extent of cognitive deficits in people with depression. Stordal et al. (2005) found that within the depressive group, a total of 56% had unaffected EF, more than half of patients with recurrent major depression had normal EF levels. These results raise questions about other variables that could explain these differences in this target population.

Currently, the literature presents a series of studies that show EF impairment both in depression and in alcohol dependence. Although these provide valuable insight into how each of these disorders can affect cognitive functions, they are separate and do not compare them.

Cognitive deficits in depression are not just an artifact of medication side effects, as several studies have found significant deterioration in EF in individuals with depression who have not taken medication (Hinkelmann et al., 2009; Taylor Tavares et al., 2007) and teenagers who have never taken psychiatric medication (Cataldo, Nobile, Lorusso, Battaglia & Molteni, 2005; Matthews, Coghill & Rhodes, 2008). Thus, they are not simply the result of using drugs to treat depression, but are directly related to the disease itself. However, some evidence suggests that long-term or repeated use of certain antidepressants can affect cognitive functions (McClintock, Husain & Cullum, 2010). Tricyclic and tetracyclic antidepressants can produce greater impairments than SSRI medication or monoamine oxidase inhibitors (Porter, Bourke &

Gallagher, 2007), although there is some evidence of the negative cognitive effects of SSRIs with anticholinergic or antihistamine actions (Lane & O'Hanlon, 1999).

Therefore, it is known to what extent these variables (such as the subtype of depression, age, number of lifetime depression episodes, and current medication) affect the performance of people with depression in EF tasks, making it difficult to compare results and confidently opt for one explanatory hypothesis or another. This diversity of results indicates a more complex feature observed in people with depression, namely the considerable heterogeneity of their neuropsychological profile (Nuño et al., 2021).

The results of the study reveal that the group of patients with depression tends to self-evaluate negatively, i.e., they believe they have lower performance than their objective performance, measured by computer tasks. Here appears a distortion towards the negative. It is known that people with depression tend to focus their attention on negative information, interpret ambiguous information negatively, and have pessimistic cognitions (Rude, Krantz, and Rosenhan, 1988). Moreover, negative self-evaluation is a complex collection of processes that occur in depression (Beck, 1963).

As for patients with alcohol dependence, most studies have shown that they present clear cognitive deficits, and these deficits persist even after prolonged abstinence and have a major impact on daily life, and the risk of relapse (Brion et al., 2017). The present study shows that, unlike patients with depression, the group of people with alcohol dependence reported higher performance on the self-evaluation questionnaire, but performance on computer tasks was weaker (Corsi test) or without significant differences (Stroop test or TR Choice). Denial is a specific characteristic in alcohol dependence (Rinn, Desai, Rosenblatt & Gastfriend, 2002).

In the literature, a series of interesting parallels have been made between alcoholic denial and the specific denial in anosognosia (unawareness of deficits) (Heilman, 1991). More broadly, it has been characterized as an inability to discover a defect, i.e., the failure to systematically collect, integrate and retain relevant information or the failure to use information to draw an obvious conclusion (Levine, Calvanio & Rinn, 1991).

All facets included in the self-assessment scale of EF were affected, both in the case of patients with depression and in the case of patients with alcohol dependence: self-organization, self-management in relation to time and planning, emotional self-regulation, self-motivation, and self-control. No significant differences were found between the two groups for the self-

motivation and self-control subscales. The biggest differences were seen on the subscale number of symptoms (items rated with 3-often or 4-very often), meaning that patients with depression tend to self-evaluate as having these difficulties most of the time.

For the computer tasks that evaluated interference control and reaction speed, there were no significant differences between the two groups. One explanation could be the small sample size.

If we analyzed the total performance of the mixed psychopathology group, as we expected, positive correlations were obtained between the EF self-assessment scale and performance tests, except in the case of the Corsi test.

The questions addressed going forward relate to the mechanisms underlying these observed deficits and what concrete actions can be taken for these patients. It is important to better understand the specific mechanisms and causal processes that contribute to EF deficits in psychopathology and to direct this knowledge towards translational applications. While EF deficits seem to be a transdiagnostic feature of psychopathology at the level of performance on neuropsychological tasks, the underlying mechanisms require more levels of analysis.

3.2 The relationship between worry, rumination, and executive functions in the clinical population (*Study 2*)

3.2.1 Introduction

Repetitive negative thinking (RNT) refers to the tendency to frequently experience uncontrollable thoughts about some negative topics (Ehring & Watkins, 2008; Gustavson, du Pont, et al., 2018), is a persistent, negatively valenced, and difficult cognitive activity to control (Ehring and Watkins, 2008), and includes worry and rumination. RNT is present in almost all mental disorders such as anxiety, depression, substance use, psychoses (Ehring & Watkins, 2008). Rumination is one of the most examined emotion regulation strategies, and is strongly implicated in the development and maintenance of various forms of psychopathology across the lifespan (Aldao et al., 2010). Worry is known to be associated with anxious and depressive symptoms in adults (Fresco, Frankel, Mennin, Turk, & Heimberg, 2002). However, the precise impact of worry on broad clinical psychopathology is not well understood (Aldao et al., 2010). Worry is a defining feature of generalized anxiety disorder, while rumination is one of the major contributors to major depressive disorder (American Psychiatric Association, 2013).

Executive dysfunction may lead to decreased ability to inhibit ruminative thinking (Von Hippel et al., 2008). The effect of rumination on cognitive functioning is independent of the level of affective mood and may be a mechanism by which depressive symptoms set in (Whitmer & Gotlib, 2013). Rumination and deficit in EF have been transdiagnostically associated in psychopathology (du Pont, Rhee, Corley, Hewitt, & Friedman, 2019). EF deficit may lead to difficulty disengaging from rumination, thereby exacerbating depression (Yu, Tseng, & Lin, 2020).

Comorbidity between depressive and anxiety disorders is common and remains incompletely understood (Ter Meulen et al., 2021). Anxiety and depressive disorders have a high prevalence but tend to co-occur and share similar symptoms (Kalin, 2020). For example, one study found that 62% of people with GAD have a depressive episode in their lifetime (Coplan, Aaronson, Panthangi, & Kim, 2015). Other researchers have shown that 57% of people with depression had comorbid anxiety symptoms (Almeida et al., 2012). One way to increase understanding of the comorbidity between these disorders is to focus on underlying vulnerabilities/mechanisms. The present study focused on the associations between three underlying cognitive vulnerabilities for anxiety and depression disorders, namely, worry, rumination, and EF.

Previous studies suggest that trait anxiety affects inhibitory control processes (Pacheco-Unguetti, Acosta, Callejas, & Lupiáñez, 2010) and performance in shifting tasks (Visu-Petra, Miclea, & Visu-Petra, 2013), but that there is no no relationship between state anxiety and inhibitory control as measured by the traditional Stroop interference control task (Visu-Petra et al., 2013).

Despite their different origins, a considerable number of studies have shown a convergence between the two types of RNT (Kim and Newman, 2023). Worry and rumination have occurred interchangeably in individuals with anxiety and depression (Blagden & Craske, 1996; McLaughlin, Borkovec, & Sibrava, 2007; Szkodny & Newman, 2019). Additionally, a high correlation between worry and rumination has been reported in both non-clinical and clinical samples (Segerstrom, Tsao, Alden, & Craske, 2000; Szkodny & Newman, 2019).

The high prevalence and high impact of comorbidity of depressive and anxiety disorders have resulted in the key rationale for studying depressive and anxiety disorders. Comorbidity is

the rule rather than the exception in over three quarters of patients with depressive and/or anxiety disorders together (Ter Meulen et al., 2021).

Another reason for investigating these disorders concurrently is to further advance the understanding of risk factors and possible etiological pathways to the comorbidity of depressive and anxiety disorders. Little is known about the unique contribution of risk indicators to comorbidity, as most etiological studies have not distinguished between unique and comorbid depressive and/or anxiety disorders.

Previous studies launched Alexopoulos's (2003) depressive-executive dysfunction hypothesis which suggests that the established association between executive functioning and depression is influenced by repetitive thinking. Philippot and Agrigoroaiei (2017), for the first time, brought empirical support for the depressive-executive dysfunction hypothesis according to which the lack of executive resources would favor a repetitive way of thinking, which in turn would worsen the mood. The authors suggest that clinical intervention targeting depression should consider repetitive thought patterns and the executive resources required to disengage from the ruminative response (Philippot and Agrigoroaiei, 2017).

Building on these data, the present study examined the associations between depression and anxiety symptoms, self-reported EF deficits in a clinical sample, and how negative repetitive thinking influences this relationship. The study focused on two specific forms of ER (rumination, worry), considering their influence on clinical symptomatology. Despite the clinical significance of RNT, its underlying mechanisms remain poorly understood. The study examines the association between EF and worry, respectively rumination, and symptoms of anxiety and depression in a clinical population. The first objective of the study was to evaluate the bivariate association between these constructs. The second aim of the study was to assess the multivariate associations between EF, worry, and rumination, on the one hand, and depression and anxiety, on the other hand. Examining the total variance in depression and anxiety explained by the interaction between EF and worry and rumination, respectively. The final aim of the study was to examine statistical mediation and how these variables influence psychopathology.

3.2.2 Method

Participants

A number of 51 participants with psychiatric pathology, N=51 (ages between 22 and 78 years, *m*= 53,2, *SD*=12,6, 78,4% female) (G*Power: 53 participants, medium effect size; Faul et

al., 2007). The formal psychiatric diagnosis of the study participants was: recurrent depressive disorder, major depressive episode, dysthymia, generalized anxiety disorder, panic disorder, and agoraphobia.

Instruments

Sociodemographic characteristics

Demographic data were collected regarding: gender, age, marital status, education level, rural/urban environment, diagnosis and current medication.

Worry - PSWQ - Penn State Worry Questionnaire (PSWQ) (Meyer et al., 1990). This is a self-report instrument with good psychometric properties (internal consistency is high - α Cronbach = 0.91).

Rumination - RRR - the RRS ruminative response scale, which is a subscale of the RSQ ruminative style questionnaire (Nolen-Hoeksema & Morrow, 1991).

Executive functions - "*The Barkley Deficits in Executive Functioning Scale*" (BDEFS-LF), developed by Russell A. Barkley (2011). The questionnaire measures the general dysfunction index and five other sub-domains.

Anxiety - BAI - Beck Anxiety Inventory (BAI; Beck et al., 1988) is a self-report questionnaire that measures anxiety severity in psychiatric and nonclinical populations.

Depression - BDI II - Beck Depression Inventory-II (BDI-II; A. T. Beck, R. A. Steer, & G. K. Brown, 1996) measures the presence and severity of depression in psychiatrically diagnosed patients as well as in the non-clinical population, has high internal consistency (α Cronbach = .93, Beck et al., 1996).

3.2.3 Results

Descriptive statistics analysis and Spearman correlation for the study measures are shown in Table 13. It displays the mean scores and Pearson correlations between the BDI, BAI, and total and subscale scores of EF (BDEFS-LF). All measured variables correlate positively and very strongly. There is also a statistically significant correlation between the BDI II and the BAI, showing that in most cases scores on these two scales tend to co-occur.

Tabel 13. Means, standard deviations, and correlations with confidence intervals.

Variable	*M*	*SD*	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]

Note. M and SD are used to represent the mean and standard deviation, respectively. 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 < .001.

We examined the association between EF and depression and anxiety, respectively, and the mediating role of rumination and worry using a structural equation model.

Table shows the results of the mediation analysis carried out to evaluate the effect of the predictor variable on the criterion, with the role of the mediator variable, indicated here as a label.

Table 14. Mediation analysis

Criterion	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

Criterion	Predictor	Label	β	*p*	CI	
					LL	UL
ind4		a2*b4	0.10	0.00	0.05	0.16

Note: β = regression coefficient, *p* = threshold of significance (*p* < .05), ind1, ind2, ind3, ind4 = indirect effects, CI=the confidence interval.

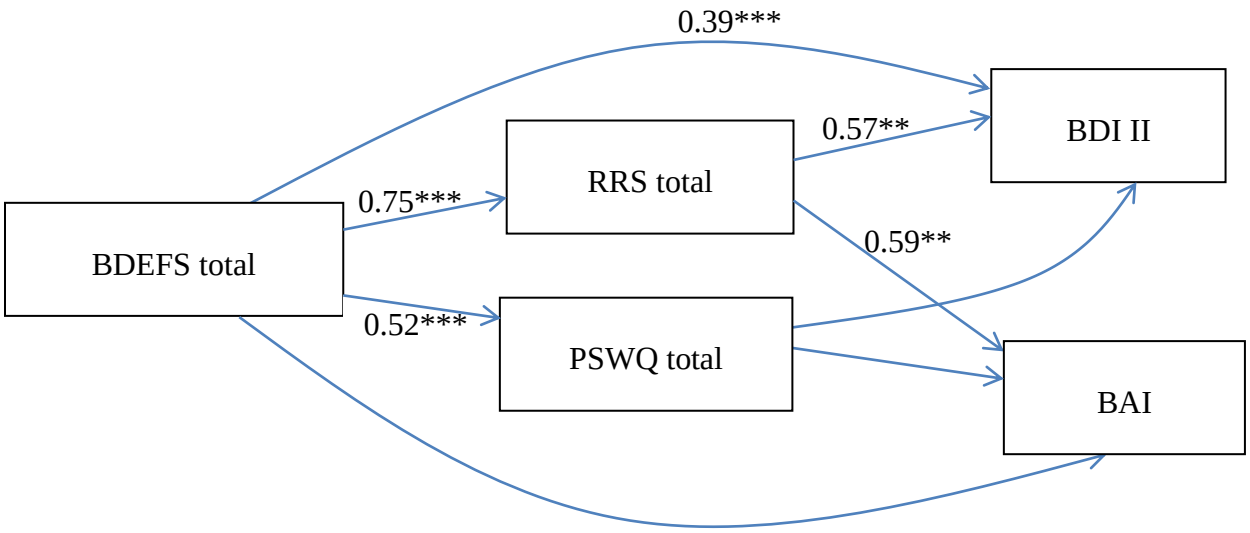

Figure 2. Graphical representation of the mediation model.

Note. The mediation coefficient * indicates p < .05. ** indicates p < .01, *** *p* < .001.

Figure illustrates the mediation model derived from structural equation analysis. Executive dysfunctions, considered as independent variables, influence depression levels, referred to here as the dependent variable, but not anxiety. This influence is both direct for depression but not for anxiety, and mediated by rumination and worry. The model thus suggests that executive dysfunctions have both a direct and an indirect effect on depression levels through rumination and worry.

We observe that there is a direct statistical relationship (direct effect) also between total BDEFS and BDI, but there is no effect between BDEFS and BAI, but RRS mediates the relationship between BDEFS and BAI. This means that the mediator RRS explains the relationship between BDEFS and BAI, but it partially explains the relationship between BDEFS

and BDI II, that there are other mechanisms here that explain the relationship, mechanisms that we did not include in the mediation.

3.2.4 Discussions and conclusions

Starting from the hypothesis of depressive-executive dysfunction of Alexopoulos (2003), and taking into account the increased comorbidity between depression and anxiety, we wanted to investigate and test a mediation model on a sample of inpatients with depression and anxiety, to analyze whether there is a relationship between executive functioning, negative repetitive thinking in this clinical population. The study examined the nature of the relationship between executive functioning and depression, respectively anxiety, with worry and rumination as mediators, and to see if these variables prove to be important mechanisms underlying anxiety and depression disorders.

The study results provide slightly different results from the study hypotheses, which may suggest an interesting pattern of relationships between the underlying mechanisms of these disorders. Thus, the results reveal that rumination, and not worry, is a mediating variable of the relationship between executive functioning and depression, respectively anxiety. Due to the fact that a significant direct effect was also found between EF and depression, a partial mediation between EF and depression and anxiety symptoms is observed.

One possible explanation for these positive and significant correlations between all the studied variables could be the fact that they are all self-rating scale, which makes them susceptible to common bias. More specifically, subjects' responses to these scales may be influenced by a negative cognitive bias, whereby subjects tend to self-evaluate more negatively. This possibility is a limitation of our study. In future research, it would be useful to assess EF using performance tests as well, not just self-report scales. This could help reduce the influence of possible self-report biases and obtain a more accurate picture of the relationship between EF and the other studied variables.

Spearman's correlation analysis supports the hypothesis that executive dysfunction is positively associated with worry and rumination. These results suggest that higher levels of worry and rumination are associated with lower EF.

The data also show that, compared to worry, rumination is more strongly associated with executive dysfunction, which could have implications for the comorbidity of anxiety and

depression disorders that could help develop intervention and treatment methods for anxiety disorders. anxiety and depression.

The overall association between worry, rumination, and executive dysfunction was expected and is consistent with other studies. Geronimi et al. (2016) found that increased worry led to increased executive function deficits. In the mediation model used, rumination was found to function as a mediator between EF and depression and anxiety, respectively. But worry did not show the same mediating effect. A plausible explanation for this could be related to the instrument used to measure anxiety, namely the BAI scale. This scale tends to place greater emphasis on the physiological elements of anxiety, rather than its cognitive components. The BAI has been challenged for its focus on psychophysiological symptoms related to panic. Results from several studies have found that patients with panic disorder score higher on the BAI than patients with generalized anxiety disorder (Cox, Cohen, Direnfeld, & Swinson, 1996).

As a negative thinking style, rumination is difficult to control or stop. This characteristic of rumination has led researchers to suggest that EF deficits may contribute to ruminative thinking (Joormann & Vanderlind, 2014; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). The results of the studies support the idea of difficulty switching attention between different cognitive representations or stopping the prevailing responses, and resisting interference generated by task-irrelevant information. This may be particularly related to this persistent nature of rumination, whereas the ability to integrate and maintain new and old information appears to be less affected by rumination (Miyake et al., 2000) than other aspects of EF.

Although rumination may be explained by EF deficits, the exact nature of this relationship remains unclear. It seems that rumination is related to problems in updating the contents of working memory, a causal relationship between the two has been difficult to prove (Vălenaș & Szentágotai-Tătar, 2017). For example, depressed individuals with high rumination scores have shown difficulty in removing negative task-irrelevant material from working memory (Joormann & Gotlib, 2008).

The present study did not include a comparison sample of adults without psychopathology. Therefore, it cannot be determined whether differences in EF could explain the results in the study.

We often observe in clinical practice, that in addition to the main disorders (main diagnosis as it is called in the medical sheet), patients have a number of secondary diagnoses,

either psychiatric or other somatic conditions. Many of these may intervene as confounding variables in the study. For example, the patients in this study had psychiatric comorbidities such as: personality disorders, somatization disorder, gambling addiction, mild cognitive deficit, and other medical conditions: arterial hypertension, cardiovascular diseases, diabetes, chronic obstructive pulmonary disease. Lower scores on EF may also be due to these comorbidities. The relationship between somatic illnesses and psychiatric outcomes has received much less attention. It is possible that somatic illnesses among psychiatric patients lead to poorer therapeutic outcomes. For example, psychiatric patients who also have other medical problems may have more severe psychiatric symptoms, greater functional deficits, and longer lengths of stay in psychiatric hospitals (Lyketsos, Dunn, Kaminsky, & Breakey, 2002) .

A limitation of the present study is that the sample consists mostly of women. Although this reflects the demographics of these disorders, and their representation in the psychiatric population, future studies should establish that the same patterns of covariates are observed in males. Another limitation is that all data were collected at the same time. Better testing of the hypotheses could be done through a longitudinal study, examining how the causal cascade unfolds over time.

3.3 The relationship between self-related emotions and depression. The influence of rumination on this relationship (*Study 3*)

3.3.1 Introduction

There are studies that have investigated the relationship between shame, guilt, and depressive symptomatology (Kim, Thibodeau, & Jorgensen, 2011). Also, there is already in the specialized literature a clear picture of the distinction between these two emotions, and the question of how shame and guilt are involved in the experience of depressive symptoms has been raised many times. Studies in non-clinical samples have shown that shame, but not guilt, is related to depressive symptomatology. There are few studies that have targeted the psychiatric population. Furthermore, one of the few studies that investigated this relationship in a clinical sample concluded exactly the opposite, that guilt and not shame is related to depressive symptomatology (Alexander, Brewin, Vearnals, Wolff, & Leff, 1999).

Many studies have assessed rumination as a state and investigated its mediating effect on the relationship between shame, guilt, and depression. To our knowledge, we found no studies

investigating the moderating effect of rumination on the shame–depression or guilt–depression relationship in a clinical sample.

Theoretically, shame is seen as a global self-evaluative experience involving the whole self, whereas guilt is specific to committed behaviors (Lutwak et al., 2003). In general, people tend to feel guilt when they focus on the negative aspects of their behaviors, what they did or did not do, but they feel shame when they focus on the negative aspects of them, on the self that did or did not do.

Guilt is less painful and less devastating than shame because when one feels guilty, the primary concern is with a specific behavior, not the whole self (Tangney 1998 apud Eisenberg, 2000). Guilt involves feelings of tension, remorse, and regret, but does not affect the person's core identity.

Self-related emotions are of fundamental importance to a wide range of psychological processes (Tracy & Robins, 2004), are moral emotions (Else-Quest, Higgins, Allison, & Morton, 2012), and have been likened to a moral-emotional barometer that provides information about the social and moral acceptability of human behavior (Tangney, Stuewig, & Mashek, 2007), and their functions remain unclear (Sznycer, 2019). These emotions contribute to the regulation of behavior, adherence to social norms and personal standards, and can influence interpersonal relationships and others' perceptions of us.

Next we will focus on two of the emotions related to the person, namely shame and guilt. This focus is justified by the relevance of these emotions in relation to depression and their importance in understanding the underlying mechanisms of this condition. Shame has been shown to correlate with depression, anxiety, psychoticism, and anger, whereas guilt has been only partially correlated with the same variables (Woien, Ernst, Patock-Peckham, & Nagoshi, 2003).

A large 2011 meta-analysis examined the link between shame, guilt, and depressive symptoms. It included 108 studies and more than 22,000 participants. The results suggest that shame is more closely related to depressive symptoms compared to guilt. Age, gender, or ethnicity did not influence these results. Although these findings do not allow the determination of a causal relationship, they indicate that shame may play a more important role in depression than guilt (Kim, Thibodeau, & Jorgensen, 2011).

However, in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; *DSM–5*; American Psychiatric Association, 2013), inappropriate or excessive guilt is a symptom of depression. Despite the fact that researchers talk about excessive or inappropriate guilt, it is not explicitly assessed (Tilghman-Osborne, Cole, & Felton, 2012). The DSM 5 places guilt as becoming inappropriate when it involves "worries or ruminations about minor failures," indicating that people "have an exaggerated sense of responsibility for unpleasant events." Such a manifestation of guilt occurs when people take responsibility for outcomes over which they have little or no control.

A 1999 study found that in depressed patients, guilt, not shame, was related to depressive symptoms (Alexander et al., 1999). In contrast, other research suggests that only shame has a major impact, causing rumination that can lead to depression (Orth, Berking, & Burkhardt, 2006).

Studies have shown that rumination is associated with lower cognitive control, with impaired EF due to the fact that it depletes executive resources. Watkins and Brown (2002) directly manipulated rumination and assessed the impact on EF in depression. Inducing rumination appears to trigger the continuous generation of ruminative thoughts, which interfere with competing executive processing (Watkins and Brown, 2002).

Rumination consists of two factors, ruminative thinking (brooding) and reflective thinking (reflection) (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). While reflective thinking involves an intentional turn to cognitive problem solving and an attempt to alleviate depressive symptoms, ruminative thinking involves a passive comparison of the current situation with an unattainable standard. Ruminative thinking is maladaptive, unlike reflective thinking, and is associated with relevant cognitive distortions in depression (Joormann, Dkane, & Gotlib, 2006).

Taking all this into consideration, the main objective of the study is to analyze the relationship between shame, respectively guilt and depression. Second, we aimed to test the moderating effect of rumination measured as a trait on the shame-depression and guilt-depression relationship.

3.3.2 Method

Participants

The present study was carried out on a group of N=53 patients with psychiatric pathology (ages between 26 and 79 years, m=53, 100% female). Formal psychiatric diagnosis (recurrent depressive disorder, depressive episode, dysthymia), hospitalization.

Instruments

Sociodemographic characteristics

Demographic data were collected regarding: gender, age, marital status, education level, rural/urban environment, diagnosis and current medication.

Shame and *guilt* variables were assessed with the Test of Self-Conscious Affect (TOSCA-3, Tangney, Wagner, & Gramzow, 1989). The instrument has proven to have good validity and reliability, with numerous studies showing good psychometric properties (scales have internal consistency of α= .74 for the shame scale and α= .69 for the guilt scale (Tangney, Miller, Flicker, & Barlow, 1996) .

Rumination - RRR - the RRS ruminative response scale, which is a subscale of the RSQ ruminative style questionnaire (Nolen-Hoeksema & Morrow, 1991).

Depression - BDI II - Beck Depression Inventory-II (BDI-II; A. T. Beck, R. A. Steer, & G. K. Brown, 1996).

3.3.3 Results

The study participants obtained a mean BDI score of M = 27.70 and a standard deviation SD = 12.23, with a minimum value of 4 and a maximum value of 57 (Table 15).

Table 15. Means and standard deviations for depression (BDI), shame (R TOSCA), guilt (V TOSCA), rumination (total RRS), brooding (B RRS), reflection (R RRS).

Variable	*min*	*max*	*M*	*SD*	*n*
BDI	4	57	27,17	12,14	53
R TOSCA	21	69	46,02	12,44	53
V TOSCA	20	80	62,53	12,88	53
RRS total	31	87	61,57	13,81	53
B RRS	6	20	14,70	4,03	53
R RRS	5	19	13,13	3,26	53

Table 16 shows three Pearson correlation coefficients between depression, shame, and guilt. There is a significant positive relationship between depression and shame. The guilt and depression variables did not correlate significantly.

Table 16. Correlations between depression (BDI), shame (R TOSCA), and guilt (V TOSCA).

Variables	1	2	3
1. BDI	-		
2. R TOSCA	,290*	-	
3. V TOSCA	-,245	,254	-

*$p < .05$(two-tailed)

We additionally analyzed the relationship between brooding, respectively reflection and depression (Table 17). Depression and brooding variables had a significant positive correlation r=.301, p< .05.

On the other hand, there is no significant correlation between the depression and reflection.

Table 17. Correlations between depression (BDI), ruminative and reflective thinking.

Variables	1	2	3
1. BDI	-		
2. brooding	,301*	-	
3. reflection	,258	,666**	-

*$p < .05$, **$p<.01$

The first model tested was the moderation effect that rumination has on the shame-depression relationship *F(3,49)=14.73, p<.001*. Effect of the shame predictor on the dependent variable depression B=.15, 95% CI [-.06, .36], *p=.07*, nonsignificant predictor of depression. For the rumination moderator, the effect on the dependent variable depression B=.35, 95% CI[.01, .69], *p=.04*, significant effect. The predictor-moderator interaction effect on the dependent variable was non-significant B=.02, 95% CI[-.01, .04], *p=.07*, effect size R^2=.07.

The second moderating model tested was the effect that rumination has on the guilt-depression relationship *F(3,49)=11.77, p<.001*. The effect of the guilt predictor on the dependent variable depression B=-.24, 95% CI[-.52, .04], *p=.09*, non-significant predictor of depression. For the rumination moderator, the effect on the dependent variable depression B=.49, 95% CI[.29, .69], *p<.01*, significant effect. The guilt-rumination interaction effect on the dependent variable depression was nonsignificant B=.01, 95% CI[-.01, .04], *p=.15*, effect size R^2=.11.

We note that no statistically significant effect of the interaction between the predictor and moderator variable was revealed in any of the two moderation models. Thus, there was no statistically significant interaction between shame and rumination, or guilt and rumination, respectively. A significant correlation between rumination and depression is observed in both moderation models.

3.3.4 Discussions and conclusions

Findings from previous studies on the relationship between shame, guilt, and depression are mixed. Four studies have shown that shame but not guilt is associated with depression (Orth et al., 2006), but the only study in a clinical sample found the opposite (Alexander et al., 1999). Orth et al. (2006) showed that both shame and guilt were significantly correlated with depression. This discordance among previous studies suggests that further investigation is needed to better understand the relations between shame, guilt, and depression in different contexts and samples.

The main conclusion of the present study is that shame, but not guilt, correlates positively with depression, thus supporting one of the insights from previous studies. The specificity of this study is that it was conducted on a clinical sample of patients with chronic depression, which adds validity and clinical relevance to the results.

There is evidence to suggest that the emotional regulation of shame and the propensity to experience shame may affect the development and maintenance of psychopathology. However, the existing investigations are only the first step in determining how they can be considered the mechanisms of change (Candea and Szentagotai, 2013).

It is interesting to note that the DSM-5 lists guilt, and not shame, as a symptom of depression. This discrepancy could be explained by the imprecise conceptual definition of the two emotions and the tendency to use these terms interchangeably in the past (Alexander et al., 1999).

Based on the results of the present study and the literature review, the overall conclusion is that the relationship between shame, guilt, and depression is complex and requires further research. The current study adds to the understanding of the relationships between these variables by highlighting the importance of shame in the context of clinical depression. In the future, researchers should continue to explore the mechanisms underlying this relationship and

develop therapeutic interventions that specifically address shame and emotion regulation in the treatment of depression.

We aimed to investigate whether rumination, measured here as a trait, has a moderating effect on the relationship between shame and depression, and guilt and depression, respectively. An effect of rumination as a putative moderator was not revealed in the two moderation models. Thus, we did not find a significant interaction between the predictor and moderator variable in any of the two analyzed situations.

Other studies found a mediating effect of rumination (measured as state) in the relationship between shame and depression (Orth et al., 2006). When analyzed separately, both the effects of shame and guilt were substantially mediated by rumination. However, when shame and guilt were analyzed simultaneously, guilt had no direct effect on depression, instead, the direct effect of shame on rumination remained. However, these studies were conducted on non-clinical samples, where both the effect size and the psychological processes involved may be different.

The lack of a moderating effect across the study may indicate that in this specific sample, shame and guilt are consistently associated with depressive symptoms, unaffected by variation in levels of rumination. However, it is important to note that these results should be interpreted with caution, as they may be influenced by sample characteristics or methodological limitations.

Based on these findings, one could further explore the relationship between shame, guilt, and depression by considering other variables that might moderate this relationship. The difference between mediation and moderation is that mediation refers to a mechanism by which an independent variable influences a dependent variable, while moderation refers to the change in the relationship between the independent and dependent variable as a function of a third variable.

Rumination plays a significant role in relation to shame and guilt in the context of depression, but the psychological processes may be different across clinical and non-clinical samples. This is an important aspect that deserves to be investigated in future studies.

From a vulnerability-stress perspective, rumination can be considered a vulnerability factor that amplifies the impact of negative life events on depressive symptoms (Abela & Hankin, 2011). In the present study, a moderating effect of rumination on the relationship between guilt/shame and depression was not found, but a significant correlation between

rumination and depression was revealed. This may indicate that rather than moderating the relationship between guilt/shame and depression, rumination may have a direct effect on depression.

A direct effect of rumination on depression implies that increased levels of ruminative thinking may contribute to the onset and maintenance of depressive symptoms, independent of shame or guilt. In this case, rumination may represent a risk factor for depression in itself that does not necessarily interact with shame and guilt to influence depression.

However, it is important to consider other variables or mechanisms that may be involved in the relationship between rumination, shame, guilt, and depression. For example, one possibility would be that rumination interacts with other variables, such as level of social support or coping skills, in the relationship between shame/guilt and depression.

To better understand the role of rumination in the context of depression and its relationship to shame and guilt, future research could continue to explore these relationships and examine other potentially relevant variables. A possible explanation of the obtained results could be the fact that most of the patients were old, had a low educational level, came from a rural environment and many of them had difficulties in translating themselves into the scenarios presented in TOSCA. Many of these scenarios were hard to imagine for many of the patients. In addition, self-reports, like other assessment methods, can be affected by many measurement artifacts (Orne, 1962). Another aspect that could interfere with the results of the study is the fact that these patients often had comorbidities. The patients were assessed by the researcher in the clinic, and although they had no other psychiatric diagnosis, many met criteria for anxiety spectrum disorders and/or personality disorders. Although these disorders are known to co-exist, the reasons involved in this co-morbidity are not fully understood (Ruscio and Khazanov, 2017). All these aspects, plus the small sample size, a homogenous sample from the point of view of gender, we consider as limitations of the study and may act as confounding variables, and it is necessary to control these variables in the future.

Shame as an emotion has important implications in psychological practice and may have implications in psychopathology and therapy: shame as a predictor, shame as a diagnostic criterion, shame as a mechanism of change, and shame as an outcome (Candea and Szentagotai, 2013). The present study supports the potential role of shame in depression but future investigations in clinical samples are needed.

4. CONCLUSIONS AND GENERAL DISCUSSIONS

4.1 Theoretical objectives

As we have shown in this paper, EF deficits have a critical importance in psychopathology, being associated with most psychiatric disorders (Snyder, Miyake & Hankin, 2015). Deficit in EF predicts rumination (Whitmer & Banich, 2007; De Lissnyder et al., 2012; Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Zetsche, D'Avanzato & Joormann, 2012), worry (Crowe, Matthews, & Walkenhorst, 2007; Snyder et al., 2010, 2014) and poor use of emotion regulation strategies (McRae, Jacobs, Ray, John, & Gross, 2012; Andreotti et al., 2013), which are all transdiagnostic risk factors for multiple forms of psychopathology (Ruscio et al., 2007; Aldao, Nolen-Hoeksema, & Schweizer, 2010; Abela & Hankin, 2011; McLaughlin & Nolen-Hoeksema, 2011).

This paper was born from clinical observations over time and from the difficulties I encountered as a clinical psychologist in my daily work. These practical experiences inspired me to explore the field of EF and cognitive mechanisms more deeply, as I observed the significant impact these aspects have on patients and the therapeutic process. The thesis makes original contributions in the following aspects: the detailed analysis of EF that strengthens the understanding and substantiation of the concept, with attention to the field of psychiatry and clinical psychology; investigation of executive dysfunctions in relevant pathologies, in an ecological environment; proposing a concrete intervention applicable in the immediate clinical context, with the aim of making patients with depression, anxiety and alcohol dependence aware of these aspects; provides practical guidelines and suggestions for clinical psychologists, helping them to better understand the cognitive mechanisms involved and to develop appropriate therapeutic strategies. These contributions strengthen knowledge and support practitioners in the care of patients with affective disorders and addictions, opening new perspectives for research and intervention in the field of clinical psychology.

In the following we will provide a synthesis of the main theoretical and empirical contributions brought by this thesis. This retrospective will highlight the relevance and impact of our research in the wider context of the literature, while highlighting the unique and innovative aspects of our approach.

The theoretical framework provided support for the studies carried out. In this chapter of the thesis I have reviewed the following aspects: the literature review on EF, the investigation of

executive dysfunction in the context of psychopathology, the examination of the specialized literature on negative repetitive thinking (including here rumination and worry, and their connection with EF), the examination of EF in clinical context (methods of measurement, impact of pharmacological treatment on EF and how executive dysfunction is reported), exploring different intervention strategies to address executive deficits and negative repetitive thinking. The literature analysis also highlighted a series of questions that are the target of research in the field.

The present work aimed to explore EF in the clinical population, under conditions of hospitalization and medication, compared to the non-clinical population; comparative examination of EF using performance tests and self-rating scales among the psychiatric population; evaluation of the mediating role of rumination, respectively worry, between deficient EF and psychopathology (depression, anxiety); and investigating the relationship between self-related emotions and depression, and the influence of rumination on this relationship.

Study 1 had as its central objective the assessment of EF by means of self-report methods and performance tests among patients with psychiatric disorders, all in an ecological context, i.e., during hospitalization and under drug treatment. These results were compared with those of a non-clinical sample to discern differences. Furthermore, a comparative analysis of EF was performed between the two predominant psychiatric disorders in the clinical sample - depression and alcohol dependence. As expected based on the specialized literature, we identified significant differences between the performances of the two groups, with the non-clinical showing superior performance compared to the clinical group on both types of assessments. As expected, we did not identify a significant correlation between the two assessment methods - the computerized tasks and the self-rating scale. An innovative element of the research was the comparison of performance on EF tests (in both computerized and self-reported tasks) between depression and alcohol dependence groups. We found that depressed patients tend to self-evaluate in a negative way, perceiving their performance as lower than objectively measured by computerized tasks. In contrast, people with alcohol dependence had better self-ratings, while their performance on computer tasks was more modest. We explored various hypotheses to understand the discrepancies between the performances of the two analyzed groups and the practical implications of these results.

In study 2 we aimed to explore the association between EF, negative repetitive thinking (worry and rumination) and the manifestation of anxiety and depression symptoms, in order to better understand the relationship between these constructs. We focused, on the one hand, on the assessment of bivariate associations between these constructs, and sought to assess the associations between EF, worry, rumination, and depression and anxiety symptoms, on the other hand. A key aspect was to examine the proportion of variance in depression and anxiety symptoms explained by the interaction between EF and worry and rumination, respectively, in order to investigate the possible mediating effects of these variables on psychopathological manifestations, thus contributing to a better understanding of how these variables intervene in the manifestation of psychopathology. The results of this study reveal a mediating role of rumination in the relationship between EF and symptoms of depression and anxiety, whereas worry did not play such a role. In addition, a significant direct relationship was found between EF and depression, suggesting a partial mediation by rumination. We explored potential explanations for the findings obtained, discussed relevant practical applications, and addressed the inherent limitations of this study.

Study 3 explored the relationship between self-related emotions (shame and guilt) and depression, with a particular focus on the possible influence of rumination on this relationship. We focused on highlighting the moderating effect of rumination on the link between shame, respectively guilt, and depression. The study showed that there was a positive correlation between shame and depression, while guilt did not show a similar association. Additionally, we observed that trait rumination did not moderate the relationship between shame, guilt, and depression, contradicting our initial hypothesis. These results emphasize the importance of shame as a potential risk factor in depression and the need to address this emotion in clinical interventions.

4.2 Methodological / practical objectives and applicative prefigurations

Within the work, some methodological and practical objectives are achieved, with the aim of making important contributions in the field of EF and associated pathologies. From a methodological point of view, we proposed to carry out a rigorous analysis of the specialized literature, to identify particularities of these phenomena in the clinical context.

On the other hand, the practical objectives of this work were related to the development and proposal of concrete intervention strategies, which are applicable in clinical practice. We highlighted the importance of adapting these strategies to the specifics of patients and to the resources available in clinical practice, considering the limited period of hospitalization and the need for efficiency of interventions.

We also wanted to raise the awareness of the clinical psychologist community about the importance of assessing cognitive deficits in psychiatric patients. In order to effectively use cognitive test data to make diagnostic and/or interventional decisions, clinicians must ask themselves the following questions: First, does the patient have cognitive decline or not? If so, what is the course of this decline (ie, sudden, gradual, or rapid) and what is the severity of the impairment? Second, is this decline characterized by a particular cognitive profile? The importance of personality as a contributing factor to individual differences in EF, beyond demographic criteria, has been demonstrated. The answers to these questions must then be integrated with the clinician's knowledge of the natural course and pathophysiology of the various disorders, as well as a thorough understanding of the brain-behavior relationship, so as to corroborate the patient's cognitive profile with knowledge of the corresponding neuroanatomical substrates that may characterize a given disorder.

Often, in addition to making diagnostic decisions, clinicians must make judgments about a patient's functioning in daily life. However, clinicians are aware that judgments about the functionality of a given patient are often subjective and full of uncertainty. This difficulty, along with the potentially problematic consequences of misclassifying patients along the functional ability dimension, has led to some considerable criticism of EF tests as lacking ecological validity (Burgess, Alderman, Evans, Emslie, & Wilson, 1998).

Behavioral signs of EF deficits may or may not be evident during the clinical interview and testing, although if they are, they should be noted and documented carefully for later integration with background information and testing data. Most often, however, the signs of EF dysfunction are milder. Some behavioral signs can sometimes be quite obvious. Many of these behavioral signs were observed during testing in the studies presented in this paper. For example, some patients may arrive late, forget to bring requested items (such as reading glasses or hearing aids), need additional prompting to continue with the task, speed up when requested by the examiner, only to slow down again a few seconds later.

Clinicians face a number of associated challenges when assessing EF. The literature recommends (1) using multiple tests of EF in a given test battery, (2) using tests that rely on different sets of component processes, (3) averaging over more tests of EF to improve the fidelity of results, and (4) to enrich assessment batteries with newer instruments that tap subdomains of EF that are not traditionally assessed (Suchy et al., 2017).

The prevalence of EF deficit in inpatient medical services is high - 72% of patients admitted to medical or surgical services have been psychiatrically evaluated, did not perform well on EF tests (Schillerstrom, Horton, & Royall, 2005). In clinical practice, I have met patients with disorders such as frontal lobe lesions, Alzheimer's disease, Parkinson's disease, Huntington's disease, multiple sclerosis, Korsakoff's syndrome. A better understanding of EF, especially the aspects of EF that are affected in each disorder, helps to find better treatments and intervention or rehabilitation programs. More specifically, aspects related to aphasia and/or other language pathologies can be observed, for which knowledge of the current theoretical information regarding EF is very useful, especially when examining patients' communication skills in everyday situations (Miyake, Emerson, & Friedman, 2000).

Moreover, patients with comorbidities such as hypertension, chronic obstructive pulmonary disease, and diabetes perform significantly worse on EF assessments compared to individuals without pathology, comorbidities very commonly associated with executive dysfunction (Schillerstrom, Horton, and Royall, 2005). These comorbidities are frequently associated with patients who call on various medical services. All of these aspects are important when clinicians make assessment and intervention decisions.

Applicative prefigurations

Taking into account the findings from the three studies, we propose as an applicative projection the development of an intervention program within the Department of Psychiatry and Ergotherapy. This program would aim to specifically address the issues identified through our studies, with the aim of improving the clinical management of patients.

Proposal for an ergotherapy intervention program:

The proposed intervention consists in making an intervention plan in the form of an informative brochure, to be distributed within the hospital to patients with difficulties in EF. The information contained in the brochure is based on the relevant conclusions from the specialized

literature and is substantiated in accordance with the results obtained within the studies carried out in this doctoral thesis, as follows:

Psychoeducation (program adapted from Naismith et al., 2011, Lehtisalo et al., 2019) – booklet that will contain the following information: brain and neuropsychological assessment; preserving brain health and controlling cardiovascular health; healthy sleep; diet and exercise; awareness of risk factors; attention and information processing speed; learning and memory; EF; depression and pharmacological treatments; non-pharmacological treatments for depression; anxiety; rumination; shame and guilt; mindfulness. We propose a future study to test the effect of this booklet on patients with EF difficulties.

4.3 Limitations

This thesis makes an important contribution to understanding the role of EF, negative repetitive thinking, anxiety and depression in the context of clinical populations. However, it is important to keep in mind that these results must be interpreted in light of several limitations.

One of the important limitations of the research is the small sample size. It is known that in studies that include psychopathology, i.e. psychiatric samples, sample sizes are often small and therefore the coefficient of determination R2 is likely to be substantial (when working with a small sample, the results might indicate that the predictors have a greater impact on the dependent variable than they actually have). Likewise, estimating the relative importance of predictors can be somewhat risky (Koretz, 1979). To alleviate these problems, it would be necessary to increase the sample size in future research, so that it is possible to use a larger number of predictors and obtain more stable results.

Another limitation would be the potential influences of uncontrolled variables that were not investigated or controlled for in depth in our studies. Variables such as the patients' medication, their level of education, as well as the testing environment - in this case, the hospital - can have a significant impact on the variables being measured. This specific environment, although necessary for the study of clinical populations, may add additional variables that are difficult to control in such a context. Other variables such as patients' level of distress, interactions with hospital staff, changes in their daily routine can influence performance on cognitive tests. This variety of conditions can have a significant impact on the results. These aspects could limit their generalizability to other contexts. Future research should consider all these variables.

Executive functions, repetitive thinking, rumination, anxiety, and depression are complex, multidimensional, and interconnected concepts. There may have been limitations in how these concepts were defined and measured (eg, the BAI Anxiety Questionnaire), and this may lead to measurement errors and distort the true links between variables. Because the mentioned concepts are interconnected, it is possible that the measures capture an overlap between them. Therefore, there is a risk that some of the observed relationships are in fact the result of this overlap, rather than a direct causal link between the variables of interest.

Despite these limitations, the thesis aims not only to improve the theoretical understanding of the studied aspects, but also to bring awareness on the part of the professional community regarding the reality faced by psychiatric patients. By exploring the relationships between EF, repetitive thinking, rumination in patients with alcohol dependence, depression and anxiety, the paper opens up possibilities for the development and improvement of clinical interventions, also proposing such an intervention. So, even if there are limitations, we want this thesis to contribute to clinical practice and the development of specific interventions for our patients.

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