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Revealing the Mechanisms That Contribute to Anger Expression Proneness in Patients with Brain Damage: The Importance of Executive Dysfunctions and Alexithymia

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Abstract: Background: The ability of scientists and clinicians to detect the therapeutic needs of patients with brain damage has increased in recent years. In this sense, many studies have signaled that individuals tend to experience an increase in irritability after suffering brain damage, with some patients even showing sudden aggressive outbursts. This increase in anger expression in these patients could be explained by executive functioning alterations (or executive dysfunctions), given their role in goal-oriented behaviors, along with emotional dysregulations such as alexithymia (e.g., difficulties recognizing and verbalizing feelings) and anger rumination (e.g., tendency to recall thoughts regarding experiences of frustration or anger). Therefore, it is essential to understand the mechanisms that contribute to and/or facilitate anger expression in patients with brain damage. **Methods:** In this regard, the main objective of this study is to assess whether executive dysfunctions (assessed with the Frontal Systems Behavior Scale) would explain anger expression (measured with the Reactive and Proactive Aggression Questionnaire) in patients with brain damage (n = 23; mean age: 56.61 ± 10.68; 57% men) compared to controls (n = 24; mean age: 60.96 ± 9.25; 33% men), paying special attention to potential moderators of this association such as alexithymia (analyzed with the Toronto Alexithymia Scale-20) and anger rumination (assessed with the Anger Rumination Scale). **Results:** The results of the current investigation led us to conclude that anger expression in patients with brain damage was partly explained by executive dysfunction, especially in those patients who scored high in alexithymia. This model was not significant among controls. **Conclusions:** Thus, we highlight the importance of targeting certain psychological alterations, such as alexithymia, when implementing psychotherapeutic programs as an adjuvant to cognitive training focused on cognitive deficits (e.g., executive dysfunctions). This, in turn, would support the full recovery of individuals who have experienced brain damage.

Keywords: alexithymia; anger expression; anger rumination; brain damage; executive dysfunction

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1. Introduction

Many studies have observed that different types of brain damage (e.g., traumatic brain injury, stroke, brain tumors, etc.) may cause individuals to exhibit more violent behaviors. These behaviors range from increased irritability or hostility to uncontrollable outbursts of anger, including aggression [1–5]. Taken together, these studies indicate that approximately 30 to 40% of patients with different types of brain damage tend to exhibit aggressive behaviors, particularly within the 12 months after experiencing this type of damage [5–7]. Thus, it is essential to understand the mechanisms that contribute to this intake of violence in order to target them and develop effective intervention programs to reduce these behaviors.

Many authors have suggested that alterations in executive functioning (or executive dysfunctions) could explain the tendency for anger outbursts or other forms of anger expression in patients with brain damage [4,8]. This is because executive functioning comprises a set of top-down mental processes, such as cognitive flexibility, inhibitory control, planning abilities, and decision-making, among others [9]. Therefore, alterations in these cognitive processes entail a reduction in the ability to properly adapt to challenging environments. The consequences of brain damage (e.g., language difficulties, dependence on others, economic stress derived from losing their jobs, etc.) could significantly affect an individual's lifestyle and that of their families. Therefore, these consequences can become an important source of distress and frustration [10]. These sources of distress, in combination with serious difficulties adapting to the demanding context derived from executive dysfunctions, might explain their proneness to anger.

Executive alterations are not the only changes observed in patients with brain damage that might contribute to anger expression. A number of studies have identified a series of symptoms that affect emotional functioning in these patients [11–13]. One of these symptoms affects the ability to identify or recognize and verbalize feelings, which is known as alexithymia [14]. As mentioned earlier, individuals who have experienced brain damage, particularly in the ventromedial prefrontal cortex but not limited to this brain area, tend to present significant difficulties identifying and verbalizing emotions [15]. This might explain their reduced ability to control certain harmful behaviors, which could be due to their lack of concern for others' wellbeing or their failure to consider the consequences of their actions because of their serious difficulty recognizing emotional states [16–19]. Therefore, it makes sense to conclude that alexithymia traits may be clearly involved in the tendency for aggressive behavior, specifically emotionally driven violence, among patients with brain damage.

Another potential candidate to explain anger proneness in patients with brain damage is anger rumination. Sukhodolsky et al. [20] defined this as the tendency of some individuals to recreate or recall thoughts regarding frustrating or anger-inducing experiences. In fact, several authors have established that the tendency to ruminate about angry thoughts was a good predictor of violence, even when controlling the role of potential confounding variables [20–24]. Taken together, it could be suggested that struggling with recognizing and expressing emotions, along with the tendency to ruminate, may contribute to the intake of violence in specific frustrating circumstances.

With all this in mind, the main objective of this study is to assess whether executive dysfunctions would explain anger expression in two samples (patients with brain damage and controls), paying special attention to potential moderators of this association, such as alexithymia and anger rumination. Taking into account previous scientific literature showing the association between the variables that explain violence proneness [9–11,13,18,24–29], we hypothesized that severe alterations in executive functioning (or executive dysfunctions) would explain higher anger expression in individuals with high levels of alexithymia, with anger rumination only being significant in patients with brain damage.

2. Materials and Methods

2.1. Participants

After screening 30 potential participants with brain damage of both genders at the Neural S.L. clinic. (Valencia, Spain), only 23 of these patients met all the inclusion criteria of this study and agreed to participate. Most of the adult patients with acquired brain damage were referred to the neural clinic from Valencian public hospitals, where a neurorehabilitation plan is implemented based on the patients' needs. In fact, it should be noted that cognitive rehabilitation interventions were complemented by other types of intervention (e.g., physiotherapy, speech therapy, etc.).

To take part in this study, participants had to be over 18 years old, have a good knowledge of Spanish (oral and written), and have a high degree of independence and understanding to complete the questionnaires (alone or with the help of the interviewer/researcher). Additionally, it was necessary for patients to be oriented (e.g., personal, spatial, and temporal) and without anosognosia, given that it was crucial to have a good understanding of the severity of the symptoms impacting the patient's life.

Regarding the control group, advertisements were posted in the city of Valencia, on social networks, and/or through word-of-mouth. To take part in the study, participants needed to have similar sociodemographic characteristics as the patients with brain damage but without brain damage or psychopathological symptoms, over 18 years old, and a good understanding of Spanish.

2.2. Procedure

Patients with different types of acquired brain damage were transferred from Valencian hospitals to the neural clinic to receive neurorehabilitation treatment. After being transferred from the hospitals, all of them were interviewed by a group of professionals (e.g., medical doctors, physiotherapists, speech therapists, neuropsychologists, among others) to determine their therapeutic needs and then implement the appropriate rehabilitation program for each patient. After the interview, they were asked if they would like to participate in our study. The researcher ensured that potential participants or their legal tutors understood the objectives of the study. Those who voluntarily agreed to participate in the research were scheduled in the clinic, after signing the informed consent, for a semi-structured interview which included questions about different sociodemographic variables (e.g., education, marital status, among others) and psychopathology (e.g., anxiety disorders, depression, etc.) together with a set of self-reports.

In relation to the control group, people who expressed interest were contacted by telephone to evaluate compliance with the inclusion criteria. Subsequently, they were scheduled at the Faculty of Psychology to collect sociodemographic variables and to administer the self-reports.

This project adapts to the ethical standards for research with humans agreed upon in the Declaration of Helsinki and approved by the ethical committee of the University of Valencia (assigned code: 2023-PSILOG-3169095).

2.3. Psychological Instruments

To measure executive dysfunctions, the Frontal Systems Behavior Scale (FrSBe; [30] conveniently adapted to Spanish [31]) was administered. This self-report consists of 46 items on a 5-point Likert scale ranging from 1 (almost never) to 5 (almost always), evaluating different symptoms that have generally been related to frontal alterations, specifically apathy, disinhibition, and executive dysfunction. It is important to note that this questionnaire evaluates frontal symptoms before the onset of brain damage or the appearance of a certain disease and/or problem (retrospective) and after the onset of this disorder (current). In our study, we asked each patient for symptoms before the brain damage and six months after it. That is, 6 months before assessment and current levels of executive functioning. This was the same for controls. The higher the score, the greater the indication of the presence of these frontal symptoms or executive dysfunctions. Reliability analysis revealed that Cronbach's alpha for this study was 0.82.

Alexithymia was measured with the Spanish version [32] of the Toronto Alexithymia Scale-20 (TAS-20) developed by Bagby et al. [33]. This instrument consists of 20 items with a 6-point Likert scale that ranges from 1 (strongly disagree) to 6 (strongly agree). The total score is calculated by adding the score on each item. Accordingly, the higher the score, the higher the presence of alexithymia symptoms. Cronbach's alpha for this study was 0.70.

Anger rumination was measured with the Anger Rumination Scale (ARS) [20] adapted to Spanish [34]. This self-report consists of 19 items on a 4-point Likert scale ranging from 1 (almost never) to 4 (almost always). The total score was obtained by adding the score of each item, with a higher total score indicating higher anger rumination. Cronbach's alpha for this study was 0.90.

To measure anger expression, we employed the Reactive and Proactive Aggression Questionnaire (RPQ) [35] adapted to Spanish [36]. This self-report consists of 23 items on a 3-point Likert scale that ranges from 0 (never) to 2 (often). The test combines items assessing reactive and proactive aggression. Furthermore, it offers the total anger expression score as the sum of the score of each item. We used the total score as a dependent variable for this study. Cronbach's alpha for this study was 0.84.

2.4. Data Analysis

First, t-Student's and/or chi-square tests were conducted to check for group differences in terms of demographic variables.

After conducting correlation analysis for each group separately, moderation analysis (model 1; two-way interaction) was performed, focusing only on the variables that were significantly interrelated. This tested the theoretical models that hypothesized violence proneness. In this sense, the dependent variable was total anger expression, and the independent variable was executive dysfunction, including alexithymia and/or anger rumination as potential moderators for each group. The independent variable and the potential moderator were introduced separately in step 1, followed by the two-way interaction between the independent variable and × moderator in step 2. A moderation effect was considered when the explained variance significantly increased from step 1 to step 2. Moderation models 2 and 3 were also checked. Model 2 evaluated the interaction between independent variable × moderator 1 + independent variable × moderator 2, and model 3 assessed the interaction between independent variable × moderator 1 × moderator 2. Again, a moderation effect was considered when the explained variance increased significantly from step 1 to 2.

Finally, following the recommendations by Hayes and Rockwood [37], we also considered simple slopes. Concretely, the interaction between each of the main predictors and the moderator was divided into "low (−1 SD)", "moderate (the mean)", and "high (+1 SD)".

All statistical analyses were performed with the IBM Statistical Package for the Social Sciences (SPSS) for Windows, Version 28.0, with the α level set at 0.05. Moreover, PROCESS 3.5 was used to conduct the moderation analyses.

3. Results

The analysis of group differences between groups did not reveal differences. That is, groups did not differ in age, gender distribution, civil status, educational level, working status, the presence of mental disorders such as anxiety or depression, handedness, and the presence of hemineglect (Table 1).

Table 1. Characteristics of both groups.

		Brain Damage (*n* = 23)	**Controls (*n* = 24)**	***t*-Test/ Chi-Square**	***p*-Value**
Age		56.61 ± 10.68	60.96 ± 9.25	−1.50	0.142
Gender	Men	57%	33%	2.06	0.152
	Women	43%	67%		
Civil status	Single	14%	13%	0.506	0.918
	Married	62%	58%		
	Divorced	14%	13%		
	Widower	10%	16%		

Table 1. *Cont.*

		Brain Damage (n = 23)	Controls (n = 24)	t-Test/ Chi-Square	p-Value
Educational level	Basic	23%	24%	3.28	0.351
	Secondary	13%	29%		
	Professional training	32%	14%		
	College	32%	33%		
Working status	Employed	36%	42%	6.05	0.109
	Unemployed	10%	4%		
	Non-contributory pension	27%	4%		
	Others (e.g., retired, dependency assistance, etc.)	27%	50%		
Mental disorders (e.g., anxiety, depression, etc.)	Yes	26%	8%	2.62	0.109
	No	74%	92%		
Handedness	Right	65%	83%	2.02	0.193
	Left	35%	17%		
Hemineglect	No	77%	-	-	-
	Yes	22%			

3.1. Correlation Analysis

The calculation of the relationships between variables using correlation analysis revealed that total anger expression was positively related to executive dysfunction, alexithymia, and anger rumination in the brain damage group ($p < 0.05$ for all cases) but was only associated with alexithymia and anger rumination in the control group ($p < 0.05$). Furthermore, executive dysfunction was significantly related to both alexithymia and anger rumination in the brain damage group, which also showed a significant relationship between alexithymia and anger rumination ($p < 0.05$) (Table 2). Therefore, moderation models were only conducted for patients with brain damage. In this regard, we checked whether executive dysfunction explained total anger expression and whether this was moderated by alexithymia and/or anger rumination by applying models 1 (executive dysfunction × alexithymia or executive dysfunction × anger rumination) and 2 or 3 (executive dysfunction × alexithymia × anger rumination) of the moderation macros.

Table 2. Correlations between variables in patients with brain damage and controls.

	Total Anger Expression		Executive Dysfunction		Alexithymia	
	Brain Damage	**Controls**	**Brain Damage**	**Controls**	**Brain Damage**	**Controls**
Executive dysfunction	0.672 ***	0.215				
Alexithymia	0.416 *	0.481 *	0.458 *	0.081		
Anger rumination	0.796 ***	0.628 ***	0.735 ***	−0.046	0.569 **	0.400

Note. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

3.2. Moderation Models

The calculation of the interaction between executive dysfunction and alexithymia, as well as between executive dysfunction and anger rumination, revealed that the amount of explained variance increased in step 2 only in the first case. In other words, the amount of explained variance increased from step 1 to step 2 only in the interaction between executive dysfunction and alexithymia (R^2 change = 0.1280, $p = 0.0242$) but not in the case of executive dysfunction and anger rumination (R^2 change = 0.0044, $p = 0.6273$). Table 3 summarizes the coefficients of the moderation model (independent variable, moderator, and the interaction

between both). The bootstrap analysis for the interaction between executive dysfunction and alexithymia revealed the following values (coeff = 0.0043, bootmean = 0.0038, bootSE = 0.0017, 95% CI = 0.0005 to 0.0063). The total model explained 59.46% of total anger expression (F = 9.29, p = 0.0005) in the brain damage group. Furthermore, the simple slopes calculation revealed that the interaction between low executive dysfunction and low alexithymia was not significant (t = 1.17, p = 0.2565), but the moderate and high levels were significant (t = 3.18, p = 0.0049; t = 4.41, p = 0.003, respectively). This means that in both cases, higher levels of executive dysfunction and alexithymia are associated with increased total anger expression (Figure 1).

Table 3. Regression models testing the interaction of the executive dysfunction predictors on alexithymia to predict total anger expression in brain damage patients.

Effect	Estimate	SE	95% CI		p
			LL	UL	
Executive dysfunction	−0.261	0.166	−0.610	0.088	0.134
Alexithymia	−0.557	0.261	−1.10	−0.011	0.045
Executive dysfunction × Alexithymia	0.005	0.002	0.001	0.009	0.024

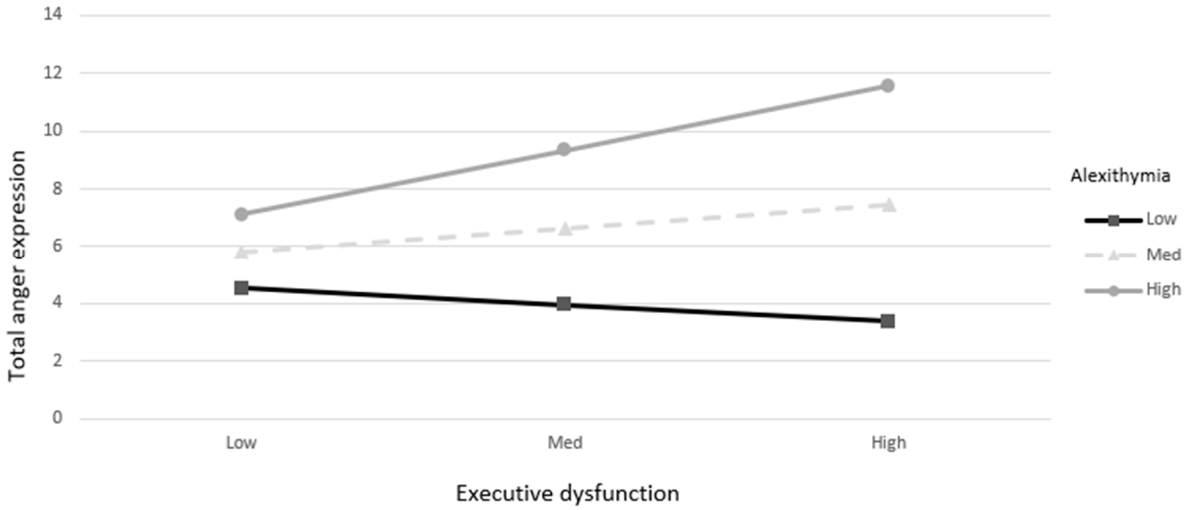

Figure 1. Total anger expression explained by the interaction between executive dysfunction and alexithymia in patients with brain damage.

Moderation models 2 and 3 were not significant. In fact, combining both interactions (executive dysfunction × alexithymia + executive dysfunction × anger rumination) did not increase the amount of explained variance of total anger expression (R^2 change = 0.0297, p = 0.4670). Furthermore, the triple interaction between the independent variable and both potential moderators (executive dysfunction × alexithymia × anger rumination) did not increase the amount of explained variance of total anger expression (R^2 change = 0.0529, p = 0.1035).

4. Discussion

Our data indicate that anger expression proneness is associated with high executive dysfunction, alexithymia, and anger rumination only in patients who have experienced some form of brain damage. However, the calculation of moderation models revealed that anger expression was only linked to executive dysfunctions, specifically among patients with brain damage who scored high in alexithymia.

The main objective of the study focused on developing a complex model that would explain anger expression in patients with brain damage. We initially hypothesized that anger expression would be explained by executive dysfunction, specifically in patients scoring high in alexithymia and anger rumination. In this sense, our results partly supported that model, given that only one significant model was observed. The variables of interest were only interrelated in the group of patients with brain damage when assessed through correlation analyses. However, only one significant moderation model, including executive dysfunction and alexithymia, was observed. This model suggests that patients with brain damage who also have executive dysfunctions may experience challenges in recognizing and expressing their feelings, leading to potential anger expression. These results support previous conclusions in this field of research, indicating that patients with significant alterations in executive functioning due to damage in the prefrontal brain also exhibit high scores in alexithymia [38]. In this regard, alexithymia has been identified as an important factor in facilitating the intake of violence, as previously indicated [39]. However, the absence of a significant association between both these variables in the control group did not support the findings of a previous study with a larger sample size that found an association between executive functioning and alexithymia over time [40]. Discrepancies between studies could be explained by the sample size used in our study, as well as the relative homogeneity of the control group. This is something that should be explored in future research to understand the link between these variables better, which would help develop effective intervention programs to improve the quality of life in patients with brain damage.

The main moderation model in our study highlights the variables to be considered when developing coadjutant psychotherapeutic interventions. As recommended, to reduce alexithymia, psychotherapy programs could incorporate different techniques focused on improving emotional awareness (e.g., establishing links between emotions and specific events, working on adopting other's perspective, employing pictograms in cases with significant brain damage or language difficulties), and/or training in emotional expression techniques (e.g., role-playing games), among others [41]. This would help patients' recovery from brain damage sequelae by combining cognitive training focused on executive dysfunctions or other cognitive deficits with therapies focused on the patient's emotional needs. However, the combination of therapies should be adapted to the patient's needs, as sometimes single therapies could offer greater advantages compared to combined programs in certain patients [42]. Hence, it is necessary to be cautious when applying these therapies without considering other patient characteristics (e.g., educational level, economic needs, personality traits, among others).

Despite the interest of our conclusions, we also highlight several limitations to consider when trying to replicate or complement our current conclusions in future research. First, the limited sample size combined with the cross-sectional nature of the study affected the reliability of our conclusions. Thus, not only is it necessary to increase the number of participants and their heterogeneity but also to assess whether those results are maintained over a sustained period of time. Second, the conclusions of this study were mainly based on self-reports, which, despite having good psychometric properties, could significantly affect the interpretation of the results due to biases such as social desirability, exaggeration, or downplaying of symptoms, among others. Third, we did not differentiate or control participant characteristics (e.g., brain damage type, sociodemographic variables, motor impairment, neuropsychological assessment, etc.), which may diminish the reliability of the results and should be amended in future studies. However, due to the small sample size, we defend the importance of not employing a large number of statistical analyses, which would increase the risk of type I errors. It is important to keep in mind that psychological symptoms resulting from brain damage may be temporary rather than irreversible. Therefore, it is crucial to assess them over time to determine if they are a temporary result of the reconfiguration of the brain to overcome the initial alterations in neurological functioning after the damage [43].

5. Conclusions

In summary, our results allow us to assert that anger expression proneness in patients with brain damage may be partly explained by the combination of executive dysfunctions and alexithymia. This can help guide therapists in developing interventions that focus not only on cognitive deficits but also on other symptoms that tend to reduce the effectiveness of the interventions. That is, it would be important to implement therapeutic programs that address both the cognitive functioning and the clinical characteristics of these patients (e.g., alexithymia, anger management techniques, the tendency to ruminate anger thoughts, among others) simultaneously. In this sense, the broader our knowledge regarding the needs of these patients, the better our ability to design interventions that are more effective in overcoming current limitations.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

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