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Ulcerative colitis symptomatology and depression:

The exacerbator role of maladaptive psychological processes

Inês A. Trindade, MSc, PhD student [1] *

Cláudia Ferreira, PhD [1]

José Pinto-Gouveia, MD, PhD [1]

[1] University of Coimbra, Portugal

Faculty of Psychology and Education Sciences

CINEICC – Cognitive-Behavioural Research Centre

* Correspondence concerning this article should be addressed to:

Inês A. Trindade

Email: ines.almeidatrindade@gmail.com

Abstract

Background: Several studies have indicated that depressive symptomatology plays a pertinent role in the clinical recurrences of ulcerative colitis (UC). Due to the self-perpetuating cycle between UC symptomatology and depressive mood, it is considered that more investment should be given to the study of factors that influence depressive symptomatology in UC patients.

Aims: This study aimed therefore at analyzing the exacerbator effect of maladaptive psychological strategies, such as cognitive fusion and brooding, on the relationship between UC symptomatology and depressive symptoms.

Methods: The sample of the current study included 84 Portuguese patients with UC that completed an Internet-based survey (comprising demographic and medical questions, and self-report measures of depression, cognitive fusion, and brooding).

Results: Results showed that UC symptomatology explained 21 % of depression severity's variance. In addition, a significant interaction between UC symptomatology and cognitive fusion was found and explained 50 % of depressive symptoms' severity. A similar interaction was revealed between UC symptomatology and brooding, which accounted for 42 % of depression's variance. These findings demonstrated that, for the same level of UC symptomatology, those participants who revealed more cognitive fusion or more brooding presented significant higher levels of depression.

Conclusions: The present study revealed cognitive fusion and brooding as moderators that exacerbate the impact of UC symptomatology on reported levels of depression. Psychological interventions that focus on the promotion of adaptive emotion regulation strategies to deal with adverse and stressful events should therefore be developed and implemented in UC patients' health care.

Keywords: Ulcerative colitis Inflammatory bowel disease Depression Cognitive fusion Brooding

Introduction

Ulcerative colitis (UC) is a form of inflammatory bowel disease (IBD) characterized by chronic and relapsing inflammation of the large intestine [1]. Although individuals with UC present a near-normal life expectancy, their psychosocial outcomes tend to be poor [2–5]. Dealing with a chronic illness such as UC can indeed be a major life stressor [6], and several accounts show that psychological disturbance is a common comorbidity. Indeed, depression rates in UC patients are higher comparing to the general population even when the illness is inactive [7]. Literature has pointed that IBD patients present a rate of 27 % of depressive disorder compared to the 12 % rate presented by nonclinical controls with similar characteristics [8, 9]. Even comparing to other chronic illnesses (e.g., colorectal cancer), UC presents higher depression rates [10].

There is general consensus that depressive symptoms are not only explained by stressful and difficult external events (such as an illness) but also depend on individual differences to responses to stress and life demands [11]. In fact, when faced with stress and negative experiences, individuals may engage in different emotion regulation processes [11, 12]. Emotion regulation processes may be conceptualized as strategies one uses to manage internal experiences (e.g., sensations, thoughts, or emotions) aiming to modify the type, frequency, magnitude, or duration of their emotional experience [12]. Emotion regulation processes may be adaptive or maladaptive; these lastly mentioned processes usually aim to avoid, eliminate, or attenuate adverse experiences but are paradoxically highly associated with the etiology and maintenance of several psychiatric conditions, namely depression [13, 14].

Cognitive fusion is one maladaptive emotion regulation process linked to depression [15]. This process refers to an excessive attachment to the content of one's

thoughts (cognitions). That is, cognitive fusion involves believing that thoughts translate reality (e.g., believing the thought ‘‘I’m getting worse day by day’’; ‘‘This fatigue is not going to end’’) and consequently responding to them as if they were literally true (e.g., giving up on meaningful activities) [16]. People with higher tendencies to engage in cognitive fusion tend to fail to see their thoughts’ content as a transitory, automatic, and idiosyncratic reaction to events and to behave adaptively [17].

Another maladaptive regulation process refers to rumination, defined as the repetitive focused attention on one’s distress and on its possible causes and consequences [13, 18]. Although individuals use this process to try to understand and resolve adverse events, it usually has a paradoxical effect as it immobilizes the individual in contexts of distress [19]. Accordingly, several studies have demonstrated that rumination predicts the onset, severity, and maintenance of depression [20, 21]. The most depressogenic form of rumination refers to brooding [14], described as the focused attention on negative or self-blaming thoughts such as ‘‘What have I done to deserve this?’’, ‘‘Why do I have problems that other people do not have?’’, or desiring a situation had gone better [22].

The study of depression in UC and the factors that may explain higher severity of depressive symptomatology are most pertinent due to the known self-perpetuating cycle of depression and relapses [3]. Indeed, depressive symptomatology has been highlighted as a risk factor for clinical recurrences of UC and has been found to have a detrimental effect in disease course [23]. The majority of UC patients with depression, however, do not receive psychological or psychiatric treatment [24]. Overall, given the relevant role of depression on physical and psychological health in UC, literature has emphasized that more consideration to this issue should be given [23]. Furthermore, although literature has pointed that maladaptive emotion regulation processes (namely

cognitive fusion and brooding) may play a pernicious effect on depressive symptomatology in different nonclinical and clinical populations, these emotion regulation processes remain unexamined in UC patients. The aim of the present study is therefore to explore associations between phenomenological variables of UC, depressive symptomatology, cognitive fusion, and brooding, and also to examine whether the adoption of these maladaptive processes exacerbates the impact of UC symptomatology on reported depressive symptoms.

Methods

Procedures.

This study is part of a broader investigation concerning the role of psychological regulatory processes in physical and psychological health of IBD patients. For this investigation, a wide sample of IBD patients was electronically collected through the Portuguese Association for IBD (APDI), which ethically approved the research methods. Participants were informed about the aims and confidential nature of the investigation, signed an informed consent, and completed a test battery on an online platform The sample of the present study was composed according to the following inclusion criteria: (1) 18 years old or older; (2) diagnosis of UC; (3) absence of another chronic or severe physical or mental illness (e.g., breast cancer, tuberculosis, generalized anxiety disorder, panic disorder); (4) absence of pregnancy.

Participants.

The final sample of the current paper included 84 Portuguese patients with UC. Participants presented ages varying from 18 to 64 years old (M = 34.89; SD = 9.69) and completed years of education between 7 and 22 (M = 14.73; SD = 2.64). The majority of

the sample was employed (85.71 %), while 11.90 % were college students and 2.38 % were unemployed. Concerning marital status, 58.3 % were married (or living together), 34.5 % were single, and 7.1 % were divorced.

Measures.

Participants reported demographic and medical information. The medical information regarded time since diagnosis, medical complications associated with UC, number of hospitalizations and surgeries, and the frequency of UC symptoms present in the last month. This lastly referred variable, UC symptomatology, was assessed using a 7-point Likert scale (0: Never; 6: Always) regarding each given UC symptom (e.g., fatigue, abdominal pain, diarrhea, bloody stools, nausea, fever). In addition, participants completed the following self-report measures.

Cognitive Fusion Questionnaire-7 (CFQ-7) ([25], Pinto- Gouveia, Dinis, Gregório and Pinto, 2014). The CFQ-7 assesses general cognitive fusion, evaluating how much the participant gets entangled with the content of his or her thoughts. This instrument presents 7 items (e.g., ‘‘I tend to get very entangled in my thoughts’’) that the participant rates on a 7-point Likert scale (1: Never true; 7: Always true) and has shown very good psychometric properties in its original and Portuguese validation studies. In the present study, this scale presented a Cronbach’s alpha of .97.

Ruminative Response Scale-10 (RRS-10) [22, 26]. This questionnaire measures the degree to which the participant engages in rumination when feeling sad and is rated on a 4-point Likert scale (0: Almost Never; 3: Almost Always). Due to the objectives of the present study, only the brooding subscale was considered (e.g., ‘‘What have I done to deserve this?’’). The RRS-10 has shown good psychometric properties in the original

and Portuguese studies. In the present study, the brooding subscale presented a Cronbach's alpha of .86.

Depression Anxiety Stress Scales (DASS-21) [27, 28]. The DASS-21 is a well-known instrument that evaluates the participant's level of depression, anxiety, and stress during the preceding week in a 4-point Likert scale (0: It did not apply to me at all; 3: It applied to me the majority of the time). Given the aims of this study, only the depression subscale was used. This subscale has presented good Cronbach's alphas in the original study (.88), Portuguese validation (.85), and present study (.92). Furthermore, the psychometric properties of the DASS-21 were sound in clinically depressed samples [29] and in chronically ill patients (e.g., [30]).

Analytic Strategy.

IBM SPSS Statistics 20 [31] was used to perform the data analyses. Preliminary data analyses were conducted to examine the adequacy of the data for further analysis. To explore the correlations between study variables, Pearson's correlation coefficients were conducted [32].

Two different models of a series of hierarchical multiple regressions were performed to examine whether the relationship between UC symptomatology and depression is moderated by cognitive fusion (Model 1) and brooding (Model 2) [32]. It is considered that a moderator effect is present when the interaction between the predictor (UC symptomatology) and the moderator (cognitive fusion or brooding) is significant (p\.05) [32]. This statistical procedure is presented with more detail in the Results section. In addition, for each model, a graphic was plotted using ModGraph [33], considering one curve for each of the three levels of the moderator (low, medium, and high) to better understand the associations between the independent variable (UC

symptomatology) and the dependent variable (depression) with different levels of the moderator variables (cognitive fusion and brooding).

Results

Preliminary Analyses.

Data were firstly evaluated for its suitability for regression analyses. Skewness and kurtosis values did not demonstrate a serious bias to normal distribution (Sk < |3| and Ku < |8–10|). Furthermore, residuals presented variance inflation factor (VIF) values inferior to 5, indicating the absence of b estimation problems, and that multicollinearity was not present. Residuals were also analyzed for independence of errors through graphic analyses and the value of Durbin–Watson. Data were thus considered adequate for further analyses.

Descriptive Analyses.

The reported frequency of UC symptomatology was revealed to be in the following order (Fig. 1): flatulence (*M* = 3.85; *SD* = 1.48), fatigue (*M* = 3.54; *SD* = 1.75), bloating (*M* = 3.39; *SD* = 2.11), abdominal pain (*M* = 2.83; *SD* = 1.56), tenesmus (*M* = 2.42; *SD* = 1.82), diarrhea (*M* = 2.18; *SD* = 1.92), difficulty in gaining or maintaining weight (*M* = 2.13; *SD* = 1.87), blood in stools (*M* = 1.80; *SD* = 1.96), vomiting (*M* = .65; *SD* = 1.07), fever (*M* = .59; *SD* = 1.18). Twenty-four patients (28.57 %) reported having active UC.

Time since diagnosis varied between two and half months and 27 years, with a mean of 6.77 years (*SD* = 5.27). Moreover, 34.52 % of the sample reported presenting one or more medical complications associated with UC (*M* = .44; *SD* = .72), such as

osteoarticular complaints (20.23 % of the total sample), anorectal pathology (3.57 %), and anemia (3.57 %). The number of hospital admissions was comprised of between 0 and 20 (M = 1.45; SD = 3.17), and the number of surgeries between 0 and 2 (M = .06; SD = .28). Detailed information regarding the participants' medical information is available in Table 1.

Regarding the other studied variables, results showed that cognitive fusion presented a mean of 26.29 (SD = 11.55), brooding a mean of 7.68 (SD = 3.84), and depressive symptomatology a mean of 5.88 (SD = 5.06).

Correlations.

Results (see Table 2) revealed that depressive symptomatology was not correlated with phenomenological variables of UC (such as time since diagnosis, existence of associated medical complications, number of hospital admissions, and number of UC-related surgeries), except with UC symptomatology. Symptomatology related to UC was also moderately associated with maladaptive psychological processes (cognitive fusion and brooding). Finally, a strong correlation was found between depressive symptoms and these maladaptive emotion regulation processes.

A partial correlation analysis controlling for active disease was performed, which demonstrated that the direction and magnitude of the associations remained the same. This variable was thus not included in the further analyses.

Moderation Analyses.

Two moderator analyses were conducted to explore whether cognitive fusion (Model 1) and brooding (Model 2) exacerbate the relationship between UC symptomatology and depressive symptoms (Table 3).

1. The moderator effect of cognitive fusion on the association between UC symptomatology and depression

UC symptomatology was firstly entered as a predictor in the first step of the regression model, which was revealed to be significant [Step 1: $F_{(2, 82)} = 21.23$, $p < .001$]. On the next step, cognitive fusion was also included as a predictor variable, and a statistically significant model was also obtained [Step 2: $F_{(2, 81)} = 36.03$, $p < .001$]. In the last step, the interaction term (UC symptomatology * cognitive fusion) was further entered and the final model explained 50 % of the severity of depression [Step 3: $F_{(3, 80)} = 26.90$; $p < .001$]. The regression coefficients showed that the interaction between these two variables was significant [$\beta = .65$; $t = 2.25$; $p < .05$]. These results indicate the existence of a moderator effect of cognitive fusion on the link between UC symptomatology and depression severity.

2. The moderator effect of brooding on the association between UC symptomatology and depression

In this model, the same procedure was performed. Thus, UC symptomatology was entered in the first step as a predictor, and brooding was added in the second step [Step 1: $F_{(1, 82)} = 21.23$, $p < .001$; Step 2: $F_{(2, 81)} = 18.16$, $p < .001$]. In the third step, the interaction variable (UC symptomatology * brooding) was also entered in the model, which explained 41 % of depression severity [$F_{(3, 80)} = 18.16$, $p < .001$]. The regression coefficients revealed that this interaction was significant ($\beta = .69$; $t = 2.88$, $p < .05$). Therefore, the moderator effect of brooding on the relationship between UC symptomatology and depression was confirmed.

To sum up, concerning both models, when the interaction terms were added in the regression there was a substantial increase in R^2, that is, in the explanation of depression. The interaction terms also presented significant and strong effects on the severity of depression, indicating that the association between UC symptomatology and depression is significantly moderated by cognitive fusion and brooding.

Two graphics were plotted to better understand the association between UC symptomatology and depression with different levels of cognitive fusion (Fig. 2) and brooding (Fig. 3), considering one curve for each of the moderators' three levels (low, medium, and high).

The graphic representation of Model 1 revealed that, for the same level of UC symptomatology, those participants who revealed more cognitive fusion showed considerable higher levels of depression. As shown in Fig. 2, it was also possible to observe that the moderator effect of cognitive fusion on the prediction of depression is stronger when UC symptoms are more frequent.

Likewise, for the same level of UC symptomatology, participants who presented higher tendencies to engage in brooding showed more depressive symptoms. The graphic representation of the model (Fig. 2) shows that the moderator effect of brooding is stronger when UC symptoms are more frequent. Also, it is interesting to observe that individuals who presented high levels of UC symptomatology but low levels of brooding demonstrated lower levels of depressive symptoms than those individuals who showed low levels of UC symptomatology and medium or high levels of brooding.

Discussion

Literature has pointed that depressive symptomatology plays a pertinent role in the course and clinical recurrences of UC [23]. Due to the self-perpetuating cycle

between physical impairment and depressive mood [3], it is considered that more investment should be given to the study of depressive symptomatology in UC patients [23]. Several studies have pointed that maladaptive emotion regulation processes may play a central role in depression severity in different nonclinical and clinical populations. Specifically, brooding and cognitive fusion have been consistently pointed as highly associated with depressive mood [15, 20]; nevertheless, these emotion regulation processes remained unexplored in UC patients. Therefore, this study aimed at analyzing the moderator effect of the maladaptive emotion regulation processes of cognitive fusion and brooding on the relationship between UC symptomatology and reported depressive symptoms.

Results showed that depressive symptoms were not associated with phenomenological variables of UC. In fact, it was interesting to observe that time since diagnosis, existence of associated medical complications, number of hospital admissions, and number of UC-related surgeries were not significantly linked to higher levels of depression. However, UC symptomatology (e.g., flatulence, fatigue, bloating, abdominal pain) was positive and moderately associated with depression severity. Furthermore, the present study extends previous literature, showing that in UC patients depressive symptoms are strongly associated with the engagement in cognitive fusion and brooding.

Results from the regression analyses showed that UC symptomatology explained 21 % of depression severity's variance. In addition, a significant interaction between UC symptomatology and cognitive fusion was found and explained 50 % of depressive symptoms. A similar interaction was revealed between UC symptomatology and brooding, which accounted for 42 % of depression's variance. These findings suggest that the association between UC symptomatology and these maladaptive regulation

processes presents significant and strong effects on the severity of depression. Indeed, cognitive fusion and brooding were revealed as moderators that exacerbate the impact of UC symptomatology on depression.

These findings demonstrate that, for the same level of UC symptomatology, those participants who revealed more cognitive fusion or more brooding presented significant higher levels of depression. That is, of the patients that report high frequency of UC symptomatology, those who present medium or high levels of cognitive fusion or brooding report considerable higher levels of depressive symptoms, comparatively with those that (with the same level of UC symptomatology) demonstrate low levels of these maladaptive regulation processes. In particular, it was possible to observe that individuals who presented high levels of UC symptomatology but low levels of brooding demonstrated lower levels of depressive symptoms than those individuals who showed low levels of UC symptomatology and medium or high levels of brooding. Furthermore, it was also interesting to examine that the moderator effects of cognitive fusion and brooding on the prediction of depression are stronger when UC symptoms are more frequent. These findings seem to reveal that, for the determination of patients' depression level, more important than specific UC symptomatology is the interaction found between UC symptomatology and maladaptive emotion regulation processes. The present study thus seems to offer relevant contributions to clinical work. In accordance, rather than focusing solely on a physical and objective evaluation and approach of patients' UC symptomatology, data seem to highlight that clinicians should also focus on the way patients deal with their symptoms, in order to be able to identify maladaptive emotion regulation processes (e.g., persistent patterns of inflexible thoughts relating to the limitations and consequences of the disease and/or its symptoms). This study may also represent an avenue to the development of

psychological interventions aiming to develop adaptive emotion regulation processes (such as cognitive defusion and decentering), that may be especially useful in the promotion of mental health of UC patients as well as other chronic patients.

It is nevertheless pertinent to acknowledge the limitations of the present study. Firstly, the sample was recruited through an Internet-based survey using self-report measures, a cost–benefit method that may have compromised the collection of a representative sample of Portuguese UC patients and the reliability of the data. Future studies should therefore confirm the findings in larger samples with other characteristics (e.g., patients recruited in clinical settings, patients of other cultures, and nationalities) using different methods (e.g., clinical interviews). In this line, another limitation of the study lies on its reliance on self-reports by the patients; in future studies, it would be interesting to integrate a clinical perspective (by the doctor in charge). Finally, since the present study's main limitation lies on its cross-sectional nature (not allowing causal interpretations), future longitudinal and experimental studies should be developed to examine the role of maladaptive emotion regulation processes in UC patients' depressive symptoms, namely other maladaptive processes that have been pointed as relevant to explain depression (e.g., self-judgment, experiential avoidance, and uncommitted action with a meaningful life).

Conclusions

The findings of the present study seem to highlight the importance of the maladaptive emotion regulation processes cognitive fusion and brooding as exacerbators of the known link between UC symptomatology and reported levels of depression. Therefore, these results suggest that psychological interventions that focus

on the promotion of adaptive emotion regulation processes to deal with adverse and stressful events should be developed and implemented in UC patients' health care.

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Conflict of interest

The authors declare no conflict of interest.

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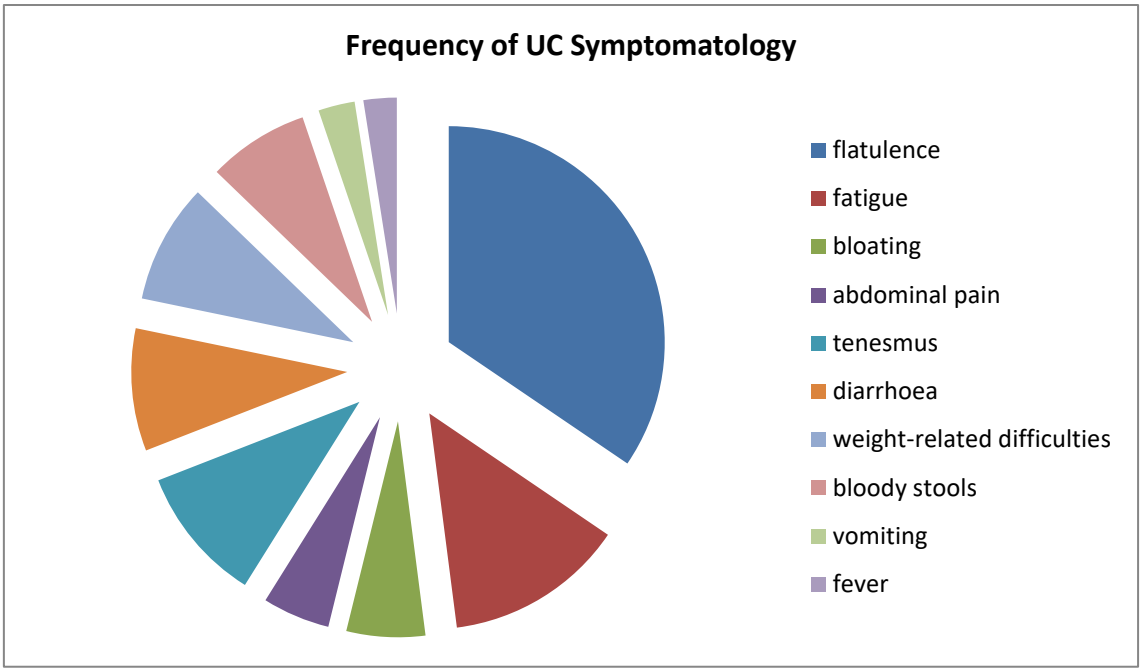


Figure 1.Frequency of UC symptoms during the preceding month (*N* = 84).

Table 1

Sample's medical characteristics (N = 84)

		n	%
Time since diagnosis	< 1 year	5	5.95
	1-4 years	32	38.10
	5-10 years	31	36.90
	> 10 years	16	19.05
Associated medical complications	osteoarticular complaints	17	20.23
	anorectal pathology	3	3.57
	anaemia	3	3.57
	dermatological complaints	2	2.38
	gingival complaints	2	2.38
	optical problems	1	1.19
	diverticula	1	1.19
	high-grade dysplasia of the colon	1	1.19
Number of hospital admissions due to UC	0	42	50
	1	21	25
	> 2	21	25
Number of surgeries due to UC	0	80	95.24
	1	3	3.57
	2	1	1.19

Table 2

Means (M), Standard Deviations (SD), Cronbach's alphas and Intercorrelation scores on self-report measures and self-reported medical data (N = 84)

	1	2	3	4	5	6	7	8
1. Age	-							
2. Time since diagnosis	.25*	-						
3. As. medical complications	.10	.23*	-					
4. N. of hospital admissions	-.18	.09	.29**	-				
5. N. of surgeries	.13	.36**	.19	.21	-			
6. UC symptomatology	-.20	-.18	.16	.28*	-.24*	-		
7. Cognitive Fusion	-.13	-.07	.14	.04	-.05	.34**	-	
8. Brooding	-.23*	-.02	.17	.21	-.07	.34**	.70***	-
9. Depression	-.06	-.01	.12	.20	.01	.45***	.64***	.53***

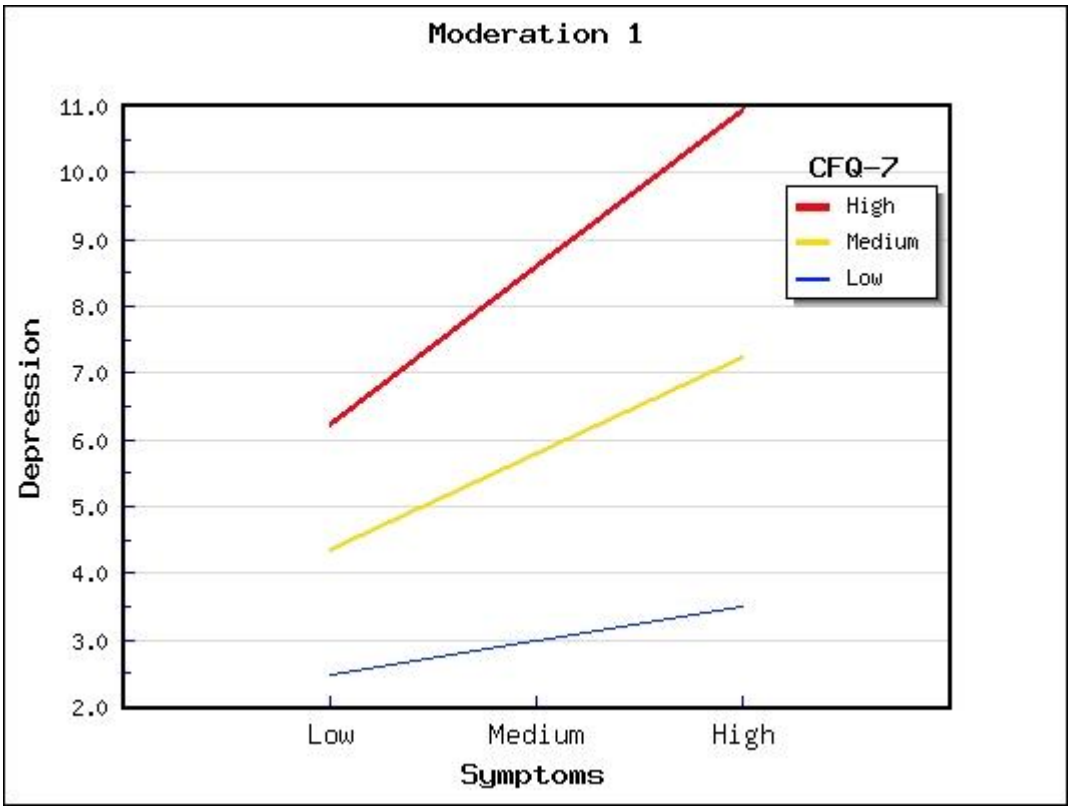
Note. * $p < .05$; ** $p < .01$; *** $p < .001$

Table 3

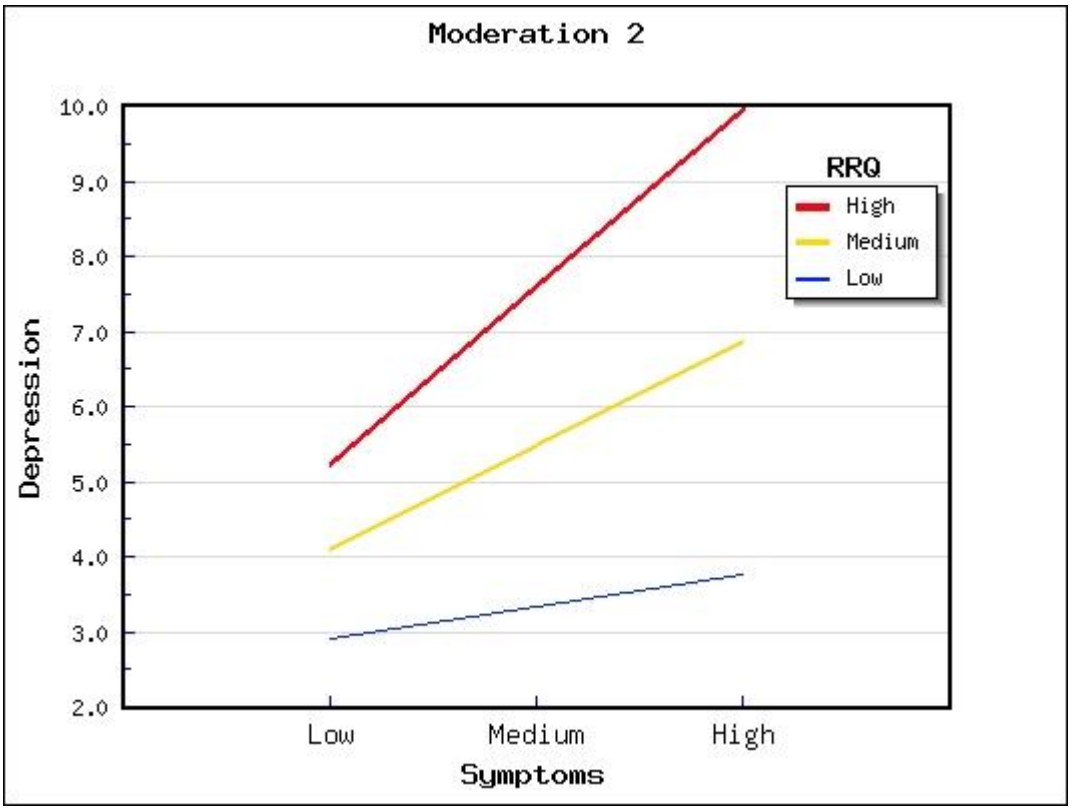
Hierarchical multiple regressions to analyse cognitive fusion's (Model 1) and brooding's (Model 2) moderator effect on depressive symptomatology (N = 84)

	Depression	
Predictor	ΔR^2	β
Model 1		
Step 1	.20***	
UC Symptoms		.45***
Step 2	.46***	
UC Symptoms		.27***
Cognitive fusion		.55***
Step 3	.48***	
UC Symptoms		-.13 ns
Cognitive fusion		.18 ns
UC_Symptoms * cognitive fusion		.65*
Total R^2	.50***	
Model 2		
Step 1	.20***	
UC Symptoms		.45***
Step 2	.35***	
UC Symptoms		.31**
Brooding		.43***
Step 3	.38***	
UC Symptoms		-.11 ns
Brooding		.03 ns
UC_Symptoms * Brooding		.69*
Total R^2	.41***	

* $p < .05$; ** $p < .01$; *** $p < .001$.



Graphic 1. Representation of the moderator effect of cognitive fusion the association between UC symptomatology and depression (N = 84).



Graphic 2. Representation of the moderator effect of cognitive fusion the association between UC symptomatology and depression (N = 84).