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The mediating role of emotional symptomatology between anticipatory fatigue and the perception of fatigue

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Abstract

Background: Clinical research stresses the importance of cognitive variables for predisposition, onset, and especially, perpetuation of perceived fatigue. The aim was to analyze the mediating effects of emotional symptomatology (somatic, depressive and anxiety) between anticipatory fatigue and perception of physical and cognitive fatigue. **Methods:** The sample was composed of 317 participants (29% from a clinical population) aged 18 to 76. Anticipatory fatigue and perception of fatigue were measured by fatigue scales. Emotional symptoms were assessed by the General Health Questionnaire, GHQ-28. **Results:** Depressive symptomatology mediated the relationship between anticipatory fatigue and cognitive fatigue in both groups, and also somatic symptoms/somatization in patients. The indirect effect of physical fatigue was observed only in the clinical group, with depressive symptoms partially mediating the anticipatory fatigue and cognitive fatigue relationship. **Conclusions:** Anticipatory fatigue has a partial indirect effect on total physical fatigue, and full indirect effect on cognitive fatigue, mediated by depressive and somatic symptoms. Anticipatory fatigue is a relevant cognitive factor in the design of psychological intervention for improvement of cognitive and physical fatigue.

Keywords: Anticipatory fatigue, anxiety symptoms, cognitive fatigue, depressive symptoms, physical fatigue, somatic symptoms.

Resumen

***El papel mediador de la sintomatología emocional entre la fatiga anticipatoria y la percepción de la fatiga*. Antecedentes:** la investigación clínica resalta la importancia de variables cognitivas en la predisposición, inicio y mantenimiento de la fatiga percibida. Se analizan los efectos de mediación de la sintomatología emocional entre la fatiga anticipatoria y la percepción de la fatiga física y cognitiva. **Métodos:** la muestra se compone de 317 participantes (29% de población clínica) de 18 a 76 años. La fatiga anticipatoria y la percepción de la fatiga se midieron a través de escalas de fatiga y los síntomas emocionales a través del Cuestionario GHQ-28. **Resultados:** la sintomatología depresiva media la relación entre fatiga anticipatoria y la fatiga cognitiva en ambos grupos, y también para el grupo de pacientes cuando además los síntomas son somáticos. En el caso de la fatiga física, el efecto indirecto se da solo para el grupo clínico, siendo la sintomatología depresiva la variable que media parcialmente la relación. **Conclusiones:** la fatiga anticipatoria tiene un efecto indirecto parcial sobre la fatiga física e indirecto total sobre la fatiga cognitiva, mediado por los síntomas depresivos y somáticos. Este factor cognitivo es relevante en el diseño de la intervención psicológica para la disminución de la fatiga cognitiva y física.

Palabras clave: fatiga anticipatoria, síntomas ansiosos, fatiga cognitiva, síntomas depresivos, fatiga física, síntomas somáticos.

Clinical research stresses the importance of cognitive variables in addition to mood and motivation factors for predisposition, onset and perpetuation of fatigue (perceived fatigue) (Kluger, Krupp, & Enoka, 2013; Sáez-Francàs et al., 2014). Fry and Martin (1996) showed that, in chronic fatigue syndrome (CFS), distortion of the perception of physical activity, the discrepancy between how much the patient thinks he/she did and reality, favored perception of overexertion.

Among CFS maintenance factors are inadequate coping with symptoms (activity avoidance or restriction), personality variables (negative affect/neuroticism, perfectionism), or anxiety and depressive symptomology (Valero, Sáez-Francàs, Calvo, Alegre, & Casas, 2013). Cognitive factors such as distorted perception of physical disability, worry about illness and expectation of appearance of more or more severe symptoms, helplessness and somatic attributions are also significant because they amplify physical and emotional symptoms, favor illness behavior and avoidance of activity, more disability and social impairment in a recurrent cycle (Wearden, Dunn, Dowrick, & Morriss, 2012).

The importance of cognitive factors in therapeutic intervention of CFS is evident. Christensen, Frostholm, Ørnbøl, and Schröder (2015) observed that illness perception mediates the benefits of cognitive therapy. More specifically, Wearden and Emsley (2013) observed the long-term importance of beliefs about the meaning of the symptoms (catastrophism), even to a greater extent than increased activity or improved cardiovascular and muscular deconditioning. However, as Cella, Chalder, and White (2011)

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suggest, the heterogeneity of these patients is the norm, and focus on bodily symptoms is the most important cognitive symptom.

The cognitive factors seem clearer in CFS than other impairments, such as multiple sclerosis (MS; Bol, Duits, Hupperts, Vlaeyen, & Verhey, 2009). But even in this disease, the cognitive variables (tendency to catastrophize, feeling embarrassed about MS symptoms, physical attributes of symptoms, beliefs that symptoms signal injury, and tendency to focus on MS symptoms), emotional states (depression/anxiety), and certain behaviors (overexertion, avoidance, excessive rest, etc.) worsen or maintain fatigue. Changes in perception of fatigue (as at least partly controllable) mediate in the effect of therapeutic intervention for fatigue, even more than behavioral changes (Knoop, van Kessel, & Moss-Morris, 2012).

Elements in common with the above may be observed. Cognitive factors like illness/symptom perception (as unpredictable and uncontrollable, or poor sense of personal control), excessive focusing on bodily sensations, or somatic attribution of their cause more than their real cause, have been suggested. Such factors seem to determine the patient's reaction (e.g., inactivity, excessive rest, all-or-nothing behavior patterns), and have a significant effect on fatigue and social impairment. The importance of aversive perception, beliefs about fatigue (as a cognitive attentional process, not of specific beliefs) and focusing on it may be deduced from this. It has been suggested that excessive focusing functions like hypervigilance in anxiety or rumination in depression (Wiborg, Knoop, Prins, & Bleijenberg, 2011).

The importance of such cognitive factors places cognitive expectation in a prime position in psychopathology (Rief et al., 2015), so expectation or anticipation of the fatigue (physical/cognitive) might be a relevant process not yet specifically analyzed in research.

Anticipatory fatigue may be similar to the anxious/apprehensive expectation described for anxiety (Barlow, 2002). Anticipatory anxiety becomes stronger after a panic attack, and also precedes it, while other indicators such as sensitivity to anxiety better explain its onset (Helbig-Lang, Lang, Petermann, & Hoyer, 2012). Generalized anxiety, where anxious expectation marks the onset and chronic maintenance of anxiety, involving increased reactivity to threat uncertainty (unpredictability), is somewhat similar (Grupe & Nitschke, 2013).

Anticipation is also important in depressive states, in this case as expectation of failure, and is relevant to the duration of distress, consolidation of cognitive indicators, and the fatigue itself, or anergy (Koval, Kuppens, Allen, & Sheeber, 2012).

In both depression and anxiety, physical (bodily symptoms) and cognitive discomfort (fears, rumination, etc.) with a tendency to inflate the negative consequences heighten distress, leading to maintenance of discomfort (Ruscio, Seitchik, Gentes, Jones, & Hallion, 2011), diminishing the probability of anticipating reward or change (Olino et al., 2011), and therefore, strengthening functional impairment.

In view of the above, in both CFS and MS, there may be difficulty in processing and identifying emotions, favoring anxious or depressed responses in situations causing fatigue (Bol et al., 2009). Discrimination of emotional states and the fatigue itself may be impeded by their shared characteristics (Caseras et al., 2008). Cognitive factors have been shown to be very relevant to reducing fatigue, while emotional symptoms could have a mediating role (Knoop, Prins, Moss-Morris, & Bleijenberg, 2010). However, the importance of somatization (somatic symptoms) in the relationship between cognitive and emotional variables has not yet been explored.

Previous studies have suggested considering anticipatory fatigue and a possible mediating role of mood symptomology in fatigue (Fuentes-Márquez, Senín-Calderón, Rodríguez-Testal, & Carrasco, 2015). This is new in the literature. Anticipatory fatigue would then be conceptually similar to anxious or depressive expectation. Thus symptoms (somatic, anxiety and depressive) would mediate the relationship between anticipatory fatigue and perceived physical and cognitive fatigue, decreasing the direct negative impact of the anticipatory fatigue on the current experience (perceived fatigue). Furthermore, this mediating role was predicted to be moderated by the clinical versus nonclinical quality of the symptomatology as indicative of severity. This functional description would not be exclusive to any particular pathology, but would deal with a general process demonstrable in various different impairments.

Method

Participants

The sample consisted of two groups, clinical (CG) ($n = 92$; 33.6% males; mean, $M_{age} = 38.87$, $SD = 14.31$) and nonclinical (NCG) ($n = 225$; 40% males; $M_{age} = 32.45$, $SD = 12.94$). CG participants were recruited by non-random sampling from a Public Mental Health Service and a private psychology center, and the NCG by snowball sampling from a population of undergraduate and postgraduate students.

Both groups were high-middle class, $t(315) = 1.17$, $p = .24$, following Hollingshead (1975), and both had a similar gender distribution, $\chi^2(1) = 1.10$, $p = .31$. Nevertheless, the CG was significantly, $t(315) = -3.88$, $p = .000$, older than the NCG. Age variance (Levene's $F = 1.120$, $p = .291$) and social class (Levene's $F = .938$, $p = .334$) were homogeneous.

CG participants were diagnosed according to the *Diagnostic and Statistical Manual of Mental Disorders-Text Revision- 4th edition* ([DSM-IV-TR] American Psychiatric Association [APA], 2000) with depressive (27.2%), anxiety (25%), adjustment (17.4%), psychotic (7.6%), personality (6.5%), and somatoform (11.9%) disorders, which will give an indication of the severity of the participants in the CG.

Instruments

Personal Information Form (PIF). Questionnaire designed to assess demographic information on the index of social rank (Hollingshead, 1975), medical conditions, psychosocial and environmental problems.

Anticipatory Fatigue. Ad hoc scale in validation process. It was evaluated by an aggregate score on three items: "I get tired before doing something that comes to my mind," "I do not feel strong enough to start anything , and I get tired easily . The items are scored on a 5-point Likert scale: 0 = *Never*, 1 = *Almost never*, 2 = *Occasionally*, 3 = *Often*, and 4 = *Always* . An ordinal alpha was calculated following Elosua and Zumbo (2008). This measure was .97 in the CG and .98 in the NCG.

Fatigue Scale (Chalder et al., 1993). This scale is a 14-item questionnaire designed to assess the intensity of perceived fatigue. It is composed of two factors: physical fatigue (eight items; e.g., "Do you have problems with tiredness?") and cognitive fatigue (six

items, e.g., "Do you have difficulty concentrating?"). Respondents answer questions about the symptoms of their fatigue during the last 15 days before evaluation on a 4-point Likert scale (from 0 = *Better than usual* to 3 = *Much worse than usual*). The Cronbach's alpha was calculated for each group and factor: a) In the CG, .88 for physical fatigue and .85 for cognitive fatigue; b) in the NCG, .82 for physical fatigue and .79 for cognitive fatigue. This scale is a valid tool for differentiating between clinical and nonclinical fatigue (with 14 or 11 items: 96-100%), with 75.5% sensitivity and 74.5% specificity, and adequate concurrent validity with a measure of social adjustment (Chalder et al., 1993; Cella & Chalder, 2010).

Goldberg's General Health Questionnaire (GHQ-28; Spanish version (Goldberg, 1996). It is a 28-item measure of emotional distress and symptoms, divided into four subscales: somatic symptoms/somatization, anxiety/insomnia, social dysfunction, and depression. Each item is scored on a four-point Likert scale: 1 = *Not at all* to 4 = *Much more than usual*. Numerous studies have been done on the reliability and validity of the GHQ-28 in clinical populations. Internal consistency and test-retest reliability are reported to be high (.78 to .90) (Robinson & Price, 1982), and excellent interrater reliability (Cronbach's α = .90–.95) (Failde & Ramos, 2000). This study used three scales: somatic (α_{CG} = .80, α_{NCG} = .75), anxiety (α_{CG} = .84, α_{NCG} = .80), and depressive (α_{CG} = .77, α_{NCG} = .70) symptoms.

Procedure

After the sample was recruited, nonclinical participants completed the questionnaires as a group at the university during a class period, and the clinical group was evaluated individually during the first and second therapy session. Assessment was done by a psychologist with clinical experience. All participants were volunteers and previously gave their written informed consent. This study was approved by the University of Seville Mental Health Service Ethic Committee.

Data analysis

Pearson's correlations (two-tailed) were calculated for variables, means and standard deviations. The Preacher and Hayes (Hayes & Preacher, 2013; Preacher & Hayes, 2004) nonparametric resampling approach to mediation, or bias-corrected bootstrapping, was performed based on 10,000 bootstrap iterations at a CI of 95%, as recommended by Mallinckrodt, Abraham, Wei, and Russell (2006). Finally, new path analysis by structural equations was carried out to test global fit of the final model, including all significant effects from previous results. The Hayes macro (Hayes & Preacher, 2013) for IBM SPSS Statistics ver. 21 was used, and LISREL 8.54 software (Jöreskog & Sörbom, 2003) to fit the final model with unweighted least squares (ULS) estimation.

Results

Preliminary analyses

Means, standard deviations, and correlations of variables are presented in Table 1. Physical and cognitive fatigue were both significantly associated with symptoms (i.e., somatic, anxiety and depressive). Anticipatory fatigue was also significantly related to fatigue (cognitive and physical) and emotional symptoms.

Table 1
Descriptive statistics and intercorrelations of variables

	1	2	3	4	5	6	7	*M*	*SD*
1. Cognitive fatigue	–							7.01	3.39
2. Physical fatigue	.60**	–						8.97	4.32
3. Depressive symptoms	.59**	.57**	–					4.44	5.93
4. Anxiety symptoms	.47**	.46**	.68**	–				8.81	6.00
5. Somatic symptoms	.51**	.50**	.65**	.75**	–			7.49	4.75
6. Anticipatory fatigue	.30**	.30**	.36**	.36**	.47**	–		1.08	1.04
7. Age	.26**	.15**	.23**	.16**	.19**	.02	–	34.32	13.64

** *p*<.01

Mediational analysis: Direct and indirect effects

The hypothesis was tested with a mediation model (see Figure 1) in which anticipatory fatigue (predictor variable) positively affects perception of cognitive and physical fatigue (dependent variables) mediated by emotional symptoms (somatic, anxiety and depressive symptoms). According to this model, symptoms have a stronger effect on perception of fatigue in the CG than in the NCG. This conditional process model contains a mediation process combined with a moderating effect by a participant group (clinical versus non-clinical). The regression coefficients were estimated using eight ordinary least square (OLS) regressions (four for cognitive fatigue, and four for physical fatigue), in line with contemporary conventional standards of mediation analysis (see Baron & Kenny, 1986; Müller & Wytykowska, 2005; Shrout & Bolger, 2002), and a bias-corrected bootstrap confidence interval for the indirect effect with 10000 bootstrap samples (Preacher & Hayes, 2004). The assumptions of OLS regressions were explored for dependent and mediating variables. The a priori sample size required for multiple regression with an anticipated large effect size (.35) and desired statistical power (.80) for eight predictors at a .05 probability level was a minimum of 52 participants.

The resulting coefficients and model information are summarized in Table 2. The more anticipatory fatigue participants show, the more somatic (a_1 = .76), anxiety (a_2 = .74) and depressive (a_3 = .73) symptoms they exhibit. Anticipatory fatigue also had a direct significant effect on physical fatigue (c' = .22) (not cognitive fatigue). Furthermore, the conditional effects of symptoms (mediators) are not contingent on group, as shown by

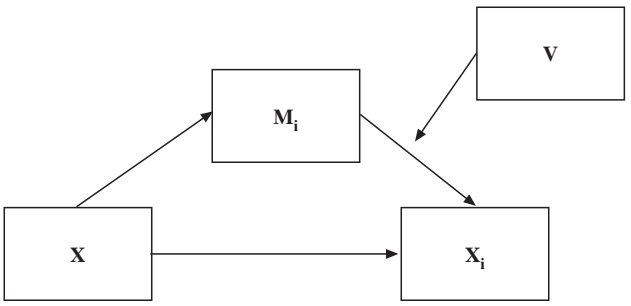

Figure 1. *Hypothesized conceptual model. X (Independent) = anticipatory fatigue; M_i (Mediators) = somatic (M_1), anxiety (M_2) and depressive (M_3) symptoms; Y (dependent) = physical fatigue (Y_1) and cognitive fatigue (Y_2); V (Moderator) = clinical condition*

Table 2
Conditional process model coefficients for cognitive and physical fatigue

	Consequents				
	SOM (M_1)	ANX (M_2)	DEP(M_3)	Cog.Fat. (Y_1)	Phy.Fat. (Y_2)
Antecedents	Beta (SE)	Beta (SE)	Beta (SE)	Beta (SE)	Beta (SE)
Anticipatory Fatigue (X)	.76(.09)**	.74(.12)**	.73(.12)**	.12(.08)	.22(.10)*
SOM (M_1)	–	–	–	-.10(.19)	.02(.25)
ANX(M_2)	–	–	–	.03(.16)	.22(.21)
DEP(M_3)	–	–	–	.23(.19)	-.16(.24)
Group (V)	–	–	–	1.22(1.05)	-.22(1.34)
M_1*V	–	–	–	.16(.12)	.10(.16)
M_2*V	–	–	–	-.05(.11)	-.14(.14)
M_3*V	–	–	–	-.00(.10)	.26(.13)
Constant	1.20(.83)	2.73(1.09)*	1.62(1.08)	3.01(1.39)*	4.87(1.78)**
R^2	.16**	.09**	.09**	.38**	.31**
F (d.f)	61.41(1,311)	33.66(1,311)	33.41(1,311)	23.31(8,304)	17.11(8,304)

Note: SOM (M_1) = somatic symptoms; ANX (M_2) = anxiety symptoms; DEP (M_3) = depressive symptoms; Group (1 "nonclinical participants", 2 "clinical participants"); M = mediator; V = moderator; d.f = degree of freedom; Cog.Fat. = cognitive fatigue; Phy.Fat. = physical fatigue
* $p<.05$
** $p<.01$

the statistically non-significant interactions between symptoms and group (nonclinical/clinical participants). Group regression coefficients showed CG participants perceived more cognitive and less physical fatigue than NCG; however, these coefficients were not significant.

Table 3 shows that the indirect effects of anticipatory fatigue on cognitive and physical fatigue through symptoms were significant for somatic and depressive symptoms. However, these effects were different depending on type of fatigue (cognitive/physical) and group (nonclinical/clinical): (*a*) The effect of anticipatory fatigue on cognitive fatigue was mediated by somatic symptoms in the CG and also by depressive symptoms in both groups. (*b*) The effect of anticipatory fatigue on physical fatigue was only mediated by depressive symptoms in the CG. Therefore, nonclinical participants who anticipated fatigue tended to perceive cognitive fatigue through depressive symptoms, while clinical participants who anticipated fatigue tended to perceive cognitive fatigue through somatic and depressive symptoms, and physical fatigue through depressive symptoms. The effects of anticipatory fatigue on perception of fatigue were mediated by symptoms, and also moderated by whether participants were clinical/nonclinical. In OLS regressions, dependent and mediating variables did not follow a normal distribution (skew and kurtosis z scores outside the -1.96/1.96 range, Kolmogorov-Smirnov and Shapiro-Wilk tests $p<.05$). However, the rest of the assumptions, such as linearity (linear plots between predictor and dependent variables), homoscedasticity (over 95% of all standardized residuals from -1.96/1.96), and independence (Durbin-Watson test 1.72 for cognitive fatigue and 1.99 for physical fatigue, and both within the 1.5 to 2.5 range suggested) were not violated. The post-hoc statistical power for the eight predictors in the different regression analyses was nearly .99.

Table 3
Conditional indirect effects of anticipatory fatigue on the perception of cognitive and physical fatigue in clinical and nonclinical participants

		Cognitive fatigue			Physical fatigue		
			CI 95%			CI 95%	
Mediators	Moderator	Beta (BootSE)	LLCI	ULCI	Beta (BootSE)	LLCI	ULCI
SOM	Nonclinical-G	.04 (.07)	-0.07	0.20	.09 (.09)	-0.07	0.28
	Clinical-G	.17 (.08)	0.00	0.35	.17 (.10)	-0.03	0.38
ANX	Nonclinical-G	-.01 (.05)	-0.13	0.08	.06 (.08)	-0.08	0.24
	Clinical-G	-.05 (.08)	-0.25	0.09	-.04 (.09)	-0.23	0.14
DEP	Nonclinical-G	.16 (.06)	0.04	0.31	.07 (.11)	-0.13	0.33
	Clinical-G	.16 (.05)	0.06	0.28	.26 (.09)	0.11	0.47

Note: G = group of participants; LLCI = lower level of confidence interval; ULCI = upper level of confidence interval; Number of bootstrap samples for bias-corrected bootstrap confidence interval 10000; SOM = somatic symptoms; ANX = anxiety symptoms; DEP = depressive symptoms
* $p<.05$
** $p<.01$

The final model

To estimate and fit the overall model found by mediational analysis, a final model was tested using structural equation modeling (SEM), with only the significant paths (Figure 2). Estimation and fit indices were calculated using unweighted least squares (ULS) (e.g., Bollen, 1989), as the data did not meet the normality assumption. This ULS estimator tends to provide more accurate estimates, standard errors, and goodness-of-fit tests than others such as WLS (weighted least squares) or DWLS (diagonally weighted least squares) (Forero, Maydeu-Olivares, & Gallardo-Pujol, 2009; Muthén, 1993). The hypothesized mediation model shows the following fit indices: $\chi^2(7) = 3.66$, $p = .82$, RMSEA = .00 (90% *CI* = [.00 to .04]), p(RMSEA<.05) = .97, CFI = 1, NNFI = 1, AGFI = 1). According to these indices, model fit to the data is satisfactory. The significant standardized regression coefficients (see Figure 2) show the impact of anticipatory fatigue on perceived physical fatigue partially mediated (the direct path from anticipatory fatigue to perceived physical fatigue was significant) through clinical depressive symptoms. Anticipatory fatigue also had an indirect effect on perceived cognitive fatigue, fully mediated (direct path from anticipatory fatigue to perceived cognitive fatigue does not remain significant) by depressive and clinical somatic symptoms. This model included correlations between factor errors on the same scale. To check that these correlated errors were not overestimating model fit, it was tested by cross validation in two balanced samples (n1 = 158 and n2 = 158). The fit indices for each sample were: $\chi^2(7) = 0.00$, $p = 1$, RMSEA = .00 (90% *CI* = [.00 to .00], p(RMSEA<.05) = 1, CFI = 1, NNFI = 1, AGFI = 0.94 for Sample 1, and $\chi^2(7) = 0.15$, $p = .99$, RMSEA = .00 (90% *CI* = [.00 to .00]), p(RMSEA<.05) = 1.00, CFI = 1, NNFI = 1, AGFI = .93) for Sample 2. Results and conclusions for these samples remained similar to the full sample.

Discussion

This research was based on the important role of cognitive factors in maintaining perceived fatigue (Knoop et al., 2012; Wearden et al., 2012). Expectation plays a major role in psychopathology (Rief et al., 2015), and nevertheless, unlike expectation in anxiety and depressive disorders, the role of expectation/anticipation of fatigue has not been studied in depth. Although it could be relevant in functional somatic syndromes, it is proposed here due to its relevance in various disorders.

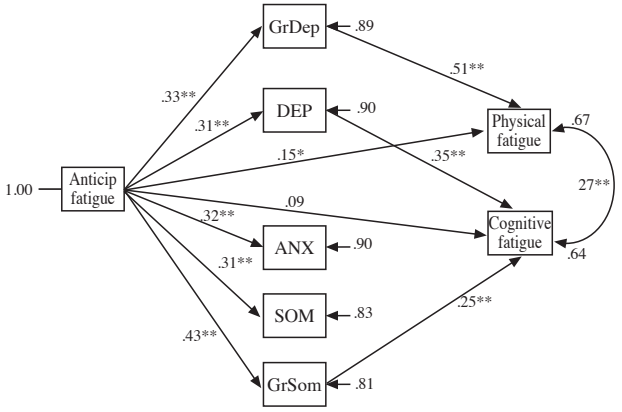

Figure 2. *Final model with standardized parameters testing the mediating and moderating relationships between anticipatory fatigue (X) and, perceived physical and cognitive fatigue through clinical versus non-clinical somatic, anxiety and depressive symptoms. Anticip = anticipatory; GrDep = group of participants*depressive symptoms; DEP = depressive symptoms; ANX = anxiety symptoms; SOM = somatic symptoms; GrSom = group of participants*somatic symptoms. Errors of mediating variables have been correlated¸ * p<.05, ** p<.01*

Some previous results have suggested that the relationship between anticipation and fatigue (physical/cognitive) is mediated by emotional symptomology (Fuentes-Márquez et al., 2015). Results show that anticipation of fatigue has a partial indirect effect on physical fatigue and full indirect effect on cognitive fatigue, mediated by depressive and somatic/somatization symptoms. As a person anticipates fatigue, and his/her emotional symptomology increases, the risk of experiencing fatigue increases significantly. This risk depends on whether the perception of fatigue is physical or cognitive, and is moderated by whether or not there is some pathology involved. Depressive symptomology mediates the relationship between anticipatory fatigue and cognitive fatigue in both groups. However, if the symptomology is related to somatic symptoms, mediation is only in the CG, suggesting a difference in severity because of the two different emotional states mediating in this relationship with cognitive fatigue. In physical fatigue, the indirect effect is only in the CG, where the depressive symptomology variable partly mediates the relationship. Therefore, the most general predictive model for perception of fatigue (physical and cognitive) includes depressive symptomology. The presence of somatic symptoms may show the patient's severity and difficulty in differentiating emotional states (Bol et al., 2009; Caseras et al., 2008). This suggests that this emotional state is as relevant to research as depression and anxiety symptoms. They further suggest that depression and somatic symptoms may be a mechanism (not the only one) explaining the relationship between anticipatory fatigue and fatigue perceived.

The relationships of these variables have not been specifically studied in the literature. The importance of presurgical expectation of symptoms and distress on pain and postsurgical fatigue has been analyzed (Schnur et al., 2007). It is known that in CFS, catastrophism and focusing on symptoms influence fatigue more than kinesiophobia. Expectation of the appearance of more symptoms with more fatigue and repercussions on emotional, physical symptoms and social functioning has been corroborated (Priebe, Fakhoury, & Henningsen, 2008). In patients with CFS, in a physical activity like climbing stairs, anticipation of fatigue explains 21% of variance in its duration (Heins et al., 2013).

In other studies, the emotional variables, covariance in the relationship of cognitive variables, or activity have been considered mediators with therapeutic change as the criterion (Wearden & Emsley, 2013). In those studies, unlike this one, no control groups were used. On the contrary, emotional variables, mainly depression, have been considered the mediator, and cognitive factors lose relevance (Valero et al., 2013). In any case, no specific relationship was found in the literature between anticipatory fatigue and mediation of emotional variables in its relationship with perception of fatigue. In this study, depression and somatic symptoms were outstanding. Some studies have stressed the importance of anxiety to negative results in cognitive therapy (Cella et al., 2011), and as in depression, a relationship with fatigue. However, as Bol et al. (2009) show, the physiological basis of anxiety and depression is partly shared, so it is possible that participation of anxiety as a

mediating variable between anticipatory fatigue and perception of fatigue was masked in the final model.

The relationship between this cognitive factor, expectation/anticipatory fatigue, with perception of fatigue, has been shown to be a useful key to be kept in mind in designing cognitive intervention, both for patients with somatic symptom disorders and more general emotional disorders.

This study had some limitations that should be considered before the results may be generalized. As a cross-study, no causality can be attributed to the relationships between variables. Furthermore, the selection of clinical participants was not random and the diagnostic categories imply clinical heterogeneity. However, this also means that analysis of the relationship of variables is not limited to any concrete disorder, but may be applied to a more general process of cognitive and emotional functioning. In any case, a larger sample size, especially in the clinical group, and more balanced participant age would be advantageous. Moreover, the normality assumption for OLS regression was violated. However, the normality assumption only affects the validity of regression analysis when the sample size is quite small (Hayes, 1996). The bias-corrected bootstrap confidence interval based on 10000 samples and the clinical (non-normal) nature of the main measures of this study lead us to believe that the normal assumption is not especially relevant for these data. Finally, underlying conditions (e.g., personality variables), as well as other cognitive factors could be analyzed to see to what extent they are relevant to anticipatory fatigue. These points should be born in mind for the design of future research.

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