Self-critical Perfectionism Predicts Lower Cortisol Response to Experimental Stress in Patients with Chronic Fatigue Syndrome

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Abstract

Previous studies have shown that self-critical perfectionism (SCP) is implicated in Chronic Fatigue Syndrome (CFS). However, to date, no studies exist that have examined whether SCP is related to a hypofunction of the hypothalamic-pituitary-adrenal (HPA) axis, which has been shown to be a key factor in the pathophysiology of CFS. We conducted a quasi-experimental study to examine the effects of SCP (as measured with the Depressive Experiences Questionnaire) on stress reactivity in a sample of 41 female CFS patients. Participants were exposed to the Trier Social Stress Test (TSST). Both subjective stress and salivary cortisol levels were measured until 90 minutes after the TSST. We also examined the relationship between stress reactivity and illness characteristics (i.e. duration and severity of symptoms). The results showed that SCP was associated with increased subjective stress reactivity, but with decreased HPA-axis reactivity as indicated by a blunted cortisol response to the TSST. Reduced cortisol reactivity was associated with greater symptom severity. There was no relationship between cortisol reactivity and illness duration. Our findings suggest that SCP is associated with loss of resilience of the neurobiological stress response system in CFS.

1. Introduction

Chronic fatigue syndrome (CFS) is part of a larger group of functional somatic syndromes, mainly defined by severe and medically unexplained fatigue and post-exertional malaise (Carruthers et al., 2011; Fukuda et al., 1994). The condition predominantly affects women (Jason et al., 1999). A considerable proportion of CFS patients also presents with chronic pain, and there are high rates of comorbidity between CFS and fibromyalgia (FM) syndrome (Ablin et al., 2011; Meeus, Nijs, & Meirleir, 2007; Van Houdenhove, Kempke, & Luyten, 2010). Besides chronic pain, psychiatric conditions often co-occur with CFS, most notably depression (Van Houdenhove et al., 2010). Evidence-based treatments for CFS include cognitive-behavioral therapy (CBT) and graded exercise therapy (GET) (Knoop, Prins, Moss-Morris, & Bleijenberg, 2010; Van Houdenhove & Luyten, 2008).

A growing body of research suggests that CFS is related to loss of resilience of the stress response system, reflecting a dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis after a prolonged state of hyperactivity due to chronic stress (i.e. childhood trauma and/or cumulative life stress) or overexertion (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Heim, Ehlert, & Hellhammer, 2000; Kempke, Luyten, Claes, Goossens et al., 2013; Kempke, Luyten, Claes, van Wambeke et al., 2013; Kempke, Luyten, De Coninck et al., 2015; Luyten et al., 2011; Nater, Maloney, Heim, & Reeves, 2011; Van Houdenhove & Luyten, 2010; Van Houdenhove, Luyten, & Kempke, 2013; Van Houdenhove, Van Den Eede, & Luyten, 2009). Indeed, a number of studies have shown that stress may play a crucial role in the onset of CFS (Nater et al., 2011; Van Houdenhove, Egle, & Luyten, 2005). Moreover, a recent meta-analysis and systematic reviews suggest that at least in a subgroup of patients, CFS is associated with a hypofunction of the HPA axis as evidenced by mild hypocortisolism, attenuated diurnal variation of cortisol, blunted cortisol stress reactivity, and enhanced

negative feedback sensitivity (i.e. enhanced suppression of cortisol via the negative feedback loop of the HPA axis) (Cleare, 2003; Papadopoulos & Cleare, 2011; Tak et al., 2011; Tomas, Newton, & Watson, in press; Van Den Eede, Moorkens, Van Houdenhove, Cosyns, & Claes, 2007). Congruent with these findings, elevated levels of inflammatory markers have been reported in CFS, such as increased proinflammatory cytokines, probably as a result of insufficient glucocorticoid signaling to the HPA axis due to hypocortisolism (Craddock et al., 2014; Klimas, Broderick, & Fletcher, 2012; Silverman, Heim, Nater, Marques, & Sternberg, 2010).

Although previous studies have provided evidence of altered HPA axis function in CFS, the factors that may underlie these dysfunctions are not yet understood. It has been suggested that chronic stress and early life stress in particular may at least in part explain HPA-axis hyporeactivity in CFS (Heim et al., 2009; Kempke, Luyten, De Coninck et al., 2015). Indeed, there is a growing body of evidence that chronic exposure to severe stress may eventually result in a switch from HPA-axis hyperreactivity to hyporeactivity (Bosch et al., 2012; Heim et al., 2000; Lupien, McEwen, Gunnar, & Heim, 2009; Luyten, Vliegen, Van Houdenhove, & Blatt, 2008; Trickett, Noll, Susman, Shenk, & Putnam, 2010; Van Houdenhove et al., 2009).

Elsewhere, we and others have postulated that chronic stress and subsequent neurobiological alterations in CFS might result, at least in part, from elevated levels of self-critical or maladaptive perfectionism (Kempke, Luyten, Claes, Goossens et al., 2013; Luyten et al., 2011; Kempke, Van Houdenhove, Claes, & Luyten, in press; Van Houdenhove et al., 2013). Self-critical perfectionism (SCP) has been shown to be a vulnerability factor for affective spectrum conditions, and has demonstrated incremental validity above and beyond higher-order traits such as neuroticism (Luyten, Blatt, Van Houdenhove, & Corveleyn, 2006; Luyten & Blatt, 2011; Sherry, Gautreau, Mushquash, Sherry, & Allen, 2014). It refers to a combination of having both high personal standards and a tendency to make overly critical

self-evaluations (Kempke, Luyten, Claes, Goossens et al., 2013). Studies suggest that this personality dimension has its roots in early attachment relationships, and develops as a result of critical and intrusive parenting (Blatt, 2004; Soenens, Vansteenkiste, & Luyten, 2010).

Regardless of its origins, evidence is accumulating from both cross-sectional and prospective studies that SCP may represent a risk factor for CFS (Deary & Chalder, 2010; Dittner, Rimes, & Thorpe, 2011; Kempke, Luyten, Claes, Goossens et al., 2013; Kempke, Luyten et al., 2011; Kempke, Van Den Eede et al., 2013; Kempke, Van Houdenhove et al., 2011; Luyten, Van Houdenhove et al., 2006; Magnusson, Nias, & White, 1996; Moss-Morris, Spence, & Hou, 2010; Sirois & Molnar, in press; White & Schweitzer, 2000). Deary and Chalder (2010), for instance, found higher levels of maladaptive perfectionism in CFS patients, but not in healthy controls, replicating earlier findings (see White & Schweitzer, 2000). Moss-Morris et al. (2010), in turn, found that maladaptive perfectionism was significantly related to the onset of CFS in patients with glandular fever (infectious mononucleosis). Kempke, Van Houdenhove et al. (2011), in a cross-sectional study, found that maladaptive perfectionism was significantly associated with fatigue in a large sample of CFS patients. In a subsequent study (Kempke, Luyten, Claes, Goossens et al., 2013), these authors demonstrated a prospective relationship between SCP and daily symptoms in CFS independently from severity of depression. Recently, Sirois and Molnar (in press) found higher levels of maladaptive perfectionism in CFS patients compared to normal controls, patients with irritable bowel syndrome, and patients with fibromyalgia (FM) or arthritis.

Furthermore, SCP has been consistently associated with increased subjective stress reactivity and with the generation of so-called fateful or person-dependent stressors (e.g., interpersonal distress) (Luyten et al., 2006; Luyten, Corveleyn, & Blatt, 2005; Shahar, Joiner Jr, Zuroff, & Blatt, 2004; Shahar & Priel, 2003; Zuroff, Mongrain, & Santor, 2004). Hence, SCP may give rise to repeated or chronic stress, leading to what McEwen (1998, 2007; see

also Arroll, 2013) termed “allostatic load” and loss of resilience of the neurobiological stress system in the long run. An association between SCP and stress has also been demonstrated in patients with CFS. In a diary method study, we showed that SCP was associated with increased stress sensitivity and with more daily hassles assessed over a 14-day period in a sample of 57 patients diagnosed with CFS (Luyten et al., 2011).

Yet, to the best of our knowledge, there are no studies that have investigated the relationship between SCP and HPA axis function in CFS patients. In a study of normal participants (i.e. 50 middle-aged men), Wirtz et al. (2007) reported that maladaptive perfectionism (concern over mistakes and doubts) predicted increased cortisol reactivity to experimental stress. However, as recently outlined by Richardson, Rice and Devine (2014), this study was limited because the authors did not take into account the “personal standards” dimension of perfectionism (i.e. extreme achievement striving), although it has been recognized as an important aspect of SCP. In doing so, Richardson and colleagues found a lower cortisol response to experimental stress among maladaptive perfectionists defined as having both high self-criticism and elevated standards. Indeed, because of the “wear and tear” caused by chronic stress (McEwen, 1998, 2007), and congruent with the idea of a switch from HPA-axis hyperreactivity to hyporeactivity (Van Houdenhove et al., 2009), it can be expected that SCP in CFS is associated with increased subjective stress reactivity, but with reduced cortisol reactivity to psychosocial stress. This hypothesis was tested in the present study in a sample of well-screened CFS patients. In addition, we investigated whether stress reactivity (i.e. cortisol and subjective stress responses) was related to illness characteristics (i.e. severity and duration of symptoms).

2. Method

2.1 Participants

Because CFS predominantly affects women (Jason et al., 1999), only female patients were enrolled in the study. A total of 43 consecutive female patients with CFS were recruited from the University Hospitals of Leuven (Belgium). CFS was diagnosed according to the Centre for Disease Control and Prevention (CDC) criteria (Fukuda et al., 1994) after multidisciplinary screening. Two patients did not complete the study. One patient refused to be exposed to experimental stress, and one patient withdrew for personal reasons. Thus, the final study sample consisted of 41 patients. All patients underwent an extensive medical and psychiatric screening procedure to exclude other causes of chronic fatigue. As in our previous study (Kempke, Luyten, Claes, Goossens et al., 2013; Kempke, Luyten, Claes, van Wambeke et al., 2013), this medical evaluation consisted of physical examination and laboratory tests (i.e., full blood count, sedimentation rate and C-reactive protein, protein electrophoresis ionogram if 40 years old, calcium and phosphorus, renal function, liver function, glycemia, muscle enzymes, antinuclear factor, cortisol, thyroid function, hepatitis B and C serology, HIV serology, urine microscopy, chest radiograph, and abdominal ultrasound). Exclusion criteria included age under 18 years, severe substance abuse, pregnant or breast-feeding, postpartum depression, major medical illness, the use of medication that has been shown to influence HPA axis function (e.g., corticosteroids), hyperthyroidism and hypothyroidism, and mental retardation (based on communication skills). The mean age of the participants was 41.95 years (S.D = 7.89) and the majority (85%) of participants had completed higher secondary school. Descriptive statistics are displayed in Table 1.

Please insert Table 1

2.2 Procedure and materials

After informed consent was obtained, patients were asked to complete a set of self-report questionnaires as part of a larger study on the neurobiology of CFS. The Depressive Experiences Questionnaire (DEQ) (Blatt, D'Afflitti, & Quinlan, 1976) was used as a measure of self-critical perfectionism. The DEQ is a well-validated 66-item self-report instrument developed to measure self-critical perfectionism and interpersonal dependency (Blatt et al., 1976; Luyten et al., 2007). In this study, only the self-criticism scale was used. The depression subscale of the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983) was used to measure severity of depression. The HADS is developed to screen for depression in medical populations, and has demonstrated good reliability and validity (Bjelland, Dahl, Haug, & Neckelmann, 2002; Zigmond & Snaith, 1983). In addition, the depression module of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (First, Spitzer, Gibbon & Williams, 1996) was administered to assess current and lifetime diagnoses of Major Depressive Disorder (MDD). Patients were also asked to complete (paper-and-pencil) daily diaries (at awakening and at 8 p.m.) for 7 consecutive days, consisting of Visual Analogue Scales (VAS) assessing severity of fatigue (0 – not at all fatigued and 100 – extremely fatigued) and pain (0 – no pain and 100 – extreme pain).

Next, individual appointments were made for the stress test. All participants were subjected to the Trier Social Stress Test (TSST), a widely used standardized psychosocial stress test that involves a free speech (5 min) and mental arithmetic (5 min) task in front of an evaluative audience instructed to maintain a neutral facial expression (Dickerson & Kemeny, 2004; Kirschbaum, Pirke, & Hellhammer, 1993; Kudielka & Wüst, 2010). In this study, the audience typically consisted of two MSc or PhD students dressed in white lab coats. Participants received only limited information about the nature of the TSST. They were told they would participate in a mental exercise task. Numerous studies have shown that exposure to the TSST is associated with a significant increase in subjective stress and salivary cortisol

as an index of HPA axis reactivity (Dickerson & Kemeny, 2004; Kudielka & Wüst, 2010). Since individual differences in stress reactivity may result from a slower recovery from acute stress, stress responses were measured up to 90 min after the TSST. Hence, salivary cortisol samples were collected at arrival, after a 30-min rest period/2 min before the onset of the TSST (baseline), immediately after the TSST (0), and at +10, +20, +30, +45 and + 90 min relative to the end of the TSST. Additionally, patients were asked to complete Visual Analogue Scales (VAS) ranging from 0 (not stressed at all) to 100 (extremely stressed) at each time point measuring subjective (self-reported) stress. The stress test was performed between 2:45pm and 3:15pm in order to minimize confounding effects of diurnal variation in cortisol levels. All TSST sessions took place in two rooms in our lab that were specifically set up for the TSST, with a waiting room and a separate room in which the stress test took place. Participants were instructed to refrain from eating, drinking (other than water), or smoking for at least 1 hour prior to the TSST. Saliva samples were obtained with Salivette (Sarstedt, Numbrecht-Rommelsdorf, Germany) and stored at −20°C at the lab until biochemical analysis. At the end of the study, participants received a financial compensation of 70 EUR for the complete study. The study was approved by the Ethics Committee of the University of Leuven (Belgium).

2.3 Data analysis strategies

For subjective stress response (VAS ratings of the TSST procedure), maximum post-test VAS scores were calculated. Using the trapezoid formula described by Pruessner, Kirschbaum, Meinlschmid and Hellhammer (2003), the area under the curve was calculated for cortisol, i.e. the area under the curve with respect to the ground (AUC_G), as an indicator of total cortisol output, and the area under the curve with respect to increase (AUC_I) as a measure of change in cortisol over time. We also calculated mean cortisol values (average of post-test samples)

and delta (peak minus baseline) cortisol (Fekedulegn et al., 2007; Heim, Mletzko, Purselle, Musselman, & Nemeroff, 2008). To assess symptom severity (i.e. fatigue and pain), VAS ratings were averaged over the 7-day period. A total symptom severity score (the average of morning and evening ratings) was then calculated for each participant. The average daily fatigue rating was 70.10 (SD = 15.35) and the average daily pain rating was 53.84 (SD = 24.20).

A series of regression analyses were conducted with SCP as independent variable and stress parameters as dependent variable after controlling for variables that have consistently been shown to be associated with the stress response (i.e. age, body mass index, and severity of depression) (Kudielka & Wüst, 2010). We also used a person-centered approach to examine the effects of SCP on stress reactivity. Specifically, we divided the sample into a low (n=21, range: −.01-−2.12) and high (n=20, range: .02-1.74) SCP group based on a median split of SCP scores on the DEQ (see also Kempke, Luyten, Van Wambeke, Coppens, & Morlion, 2014). Chi square and *t* tests were used to test whether there were any differences in demographic and health variables (i.e. age, body mass index, and depression) between the low and high SCP group. Repeated measures analysis of variance (Greenhouse-Geisser corrected) was used to determine the effects of SCP on subjective and neuroendocrine stress reactivity to experimental stress with sample time (from baseline to 90 min follow up: −2, 0, +10, +20, +30, +45, and +90 min) as the within-subject factor and high and low SCP as the between-subject factor. Significant results were further analyzed by simple contrasts. Independent-samples *t* tests were used to compare mean values of both groups at single time points. To correct for skewness, raw cortisol values (µg/L) were natural log-transformed prior to analysis. All analyses were performed using SPSS 20.0. Results were considered statistically significant at the $p < .05$ level.

3. Results

3.1. The association between SCP and stress reactivity

SCP was significantly positively associated with maximum subjective stress ratings (β= .331, *p*=.043). Figure 1 illustrates the positive relationship between SCP and subjective stress. An inverse relationship, however, was found for cortisol. SCP was significantly negatively associated with measures of increase in cortisol, i.e. AUC_I (β= − .350, *p*=.030) and delta cortisol (β= − .333, *p*=.041). Although not statistically significant, results showed a negative relationship with AUC_G (β= − .139, *p*=.390) and mean cortisol (β= − .164, *p*=.310). The negative relationship between SCP and cortisol is illustrated in Figure 2.

Please insert Figure 1 and Figure 2

3.2. Effects of high and low SCP on stress reactivity

Results showed no significant differences between the high and the low SCP group in age (*t* =.39, *p*=.70) and BMI (*t* = −.22, *p*=.83). Likewise, no significant group differences were detected in the distribution of current (χ^2 =1.73, *p*=.19) and lifetime (χ^2 =2.47, *p*=.12) depression as determined by the SCID or in severity of depression (*t* =1.17, *p*=.25) as measured with the HADS. Therefore, these variables were not included as covariates in the ANOVA analyses.

Results revealed no significant group differences in subjective and cortisol stress reactivity upon arrival (*t*=1.542, *p*=.131; *t*=.096, *p*=.924, respectively) and at baseline (Time -2) (*t*=1.307, *p*=.199; *t*=−.497, *p*=.622, respectively). Yet, results of the repeated measures ANOVA showed a highly significant between-group effect for subjective stress (F(1,39) = 10.552, *p*=.002) (see Figure 3). A series of *t* tests showed that post-test subjective stress ratings were significantly higher in the high SCP group as compared to the low SCP group

(all *p*'s <.05). Likewise, maximum post-test VAS scores were significantly higher in the high SCP group than in the low SCP group (M=66.48, S.D.=17.97 vs. M=51.43, S.D.=21.68; *t*=2.376, *p*=.023, Cohen's d =.77, representing a medium to large effect size).

Results also revealed a significant time by group interaction effect for cortisol stress response (F(1,39) = 3.337, *p*=.018). Simple contrasts showed that this interaction effect resulted from a different response pattern from Time −2 (baseline) to Time 0 (immediately after the test) (F(1,39) = 9.402, *p*=.004). Specifically, the high SCP group showed a blunted cortisol response, whereas the low SCP group showed an increase in cortisol secretion immediately after exposure to the TSST (see Figure 3). Independent samples *t*-tests showed that post-test cortisol concentrations were significantly lower in the high SCP group than in the low SCP group (all *p*'s <.05).

Please insert Figure 3

Similarly, AUC_G (*t*=−2.244, *p*=.033), AUC_I (t=−2.877, *p*=.006), mean cortisol (t=−2.409, *p*=.023), and delta (peak minus baseline) cortisol (*t*=−3.085, *p*=.004) were significantly lower in the high SCP group relative to the low SCP group. Mean values and effect sizes are presented in Table 2.

Please insert Table 2

3.3. The association between stress reactivity and illness characteristics

Illness duration was not significantly related to maximum subjective stress ratings (r= .116, *p*=.507), AUC_I (r= − .002, *p*=.991), delta cortisol (r= − .064, *p*=.711), AUC_G (r= − .071, *p*=.683) or mean cortisol (r= − .076, *p*=.661). However, symptom severity was significantly associated with cortisol reactivity. Specifically, fatigue and pain levels were negatively associated with delta cortisol (r= −.357, *p*=.024; r= −.430, *p*=.006, respectively) and AUC_I

(r= −.307, *p*=.054; r= −.358, *p*=.023, respectively), although the association between fatigue and AUC_I did not reach statistical significance. AUC_G was significantly correlated with pain (r= −.410, *p*=.009), but not with fatigue (r= −.184, *p*=.256). No significant correlations were found between subjective stress ratings and symptoms (fatigue: r= .152, *p*=.357; pain: r= .234, *p*=.151).

4. Discussion

Previous research has demonstrated that self-critical perfectionism (SCP) is implicated in the onset and maintenance of CFS. The present study investigated the effects of SCP on both subjective and neuroendocrine stress response in a sample of 41 CFS patients using the Trier Social Stress Test, a well-validated laboratory stress reactivity paradigm. Results revealed that patients with higher levels of SCP exhibited increased subjective stress reactivity to psychosocial stress. This is consistent with a previous studies showing that SCP is related to higher subjective stress sensitivity in CFS patients in the daily flow of life (Luyten et al., 2011). Most importantly, we found that SCP was related to blunted cortisol reactivity to psychosocial stress in CFS, congruent with our assumption that this personality dimension is associated with loss of resilience of the stress system because of the "wear and tear" of chronic stress.

Our findings are in line with studies demonstrating a relationship between early life stress (i.e. childhood trauma exposure) and reduced cortisol in CFS (Heim et al., 2009; Kempke, Luyten, De Coninck et al., 2015). It has been suggested that SCP may be a coping mechanism to compensate for negative self-feelings associated with childhood trauma and emotional trauma in particular (for an overview see Kempke et al., in press). However, empirical evidence in support of this hypothesis is limited. For instance, in analyses reported elsewhere

(Kempke, Luyten, De Coninck et al., 2015), we found no evidence that SCP mediated the relationship between early childhood trauma (i.e. emotional neglect) and cortisol reactivity.

Interestingly, we also found that cortisol reactivity, but not subjective stress reactivity, was associated with symptom severity. Specifically, we found an inverse relationship between cortisol reactivity and symptom severity, suggesting that reduced HPA axis reactivity is associated with greater symptom severity in CFS. In this regard, a recent study by Hall et al. (2014) revealed that higher HPA activity (i.e. cortisol awakening response) was associated with less post-exertional malaise severity in CFS patients. Furthermore, we did not find a relationship between illness duration and stress reactivity measures.

Results of this study should be interpreted in the light of several limitations. First, the lack of a control group limits the conclusions of the results. The current study does not clarify whether cortisol levels are significantly decreased compared to normal controls. Yet, the main aim of this study was to test whether there is an inverse relationship between SCP and cortisol reactivity in CFS as suggested by recent findings on the effects of chronic stress on HPA axis function. Relatedly, it is not clear whether these findings can be generalized to other functional somatic conditions. Given that CFS is part of a larger spectrum of disorders, it is rather unlikely that these findings are specific for CFS. Riva and colleagues (Riva, Mork, Westgaard, & Lundberg, 2011; Riva, Mork, Westgaard, Ro, & Lundberg, 2010), for instance, demonstrated significantly lower levels of cortisol in fibromyalgia patients as compared to controls, and there is increasing empirical evidence that SCP may also be implicated in these patients (Lerman, Rudich, & Shahar, 2010; Lerman, Shahar, & Rudich, 2012). However, further controlled research is needed to substantiate these assumptions. Second, we did not measure the presence of daily hassles or life events prior to the TSST which may have affected subjective and neuroendocrine stress levels. However, our analyses showed no significant group differences in self-reported stress or cortisol secretion at arrival. Third, the

high lifetime prevalence of depression in our sample may have influenced the results. Yet, comorbidity between CFS and depression is the rule rather than the exception, although prevalence rates for current depression were rather low in this study as compared to findings in other studies (Kempke et al., 2010; Kempke, Luyten et al., 2011). Finally, since we only included female patients, findings cannot be generalized to all CFS patients. Therefore, although CFS most often affects women (Jason et al., 1999), future studies should investigate the relationship between SCP and stress reactivity in both female and male patients with CFS.

The current findings may have important implications for the treatment of CFS, particularly since previous studies have shown that SCP may impede treatment response (Blatt, Zuroff, Hawley, & Auerbach, 2010). Moreover, hypocortisolism has been shown to predict poor treatment response in CFS patients (Roberts et al., 2010). In this regard, we recently showed that pre-treatment SCP negatively impacts treatment response in patients with chronic pain following a psycho-educational CBT-based treatment (Kempke et al., 2014). Furthermore, there is evidence for a significant increase in cortisol output after psychological treatment in both adult and adolescent CFS patients (Nijhof et al., 2014; Roberts, Papadopoulos, Wessely, Chalder, & Cleare, 2009) and a normalization of activity levels after multidisciplinary treatment (Van Houdenhove, Bruyninckx, & Luyten, 2006). Hence, treatment of CFS should include a focus on SCP and related cognitive-behavioural factors (e.g., "persistence") in at least a subset of patients. A wide range of specialized interventions exist to treat self-critical perfectionistic tendencies, including cognitive-behavioural and psychodynamic approaches (Blatt, 2004; Flett & Hewitt, 2008; Glover, Brown, Fairburn, & Shafran, 2007; Luyten, Van Houdenhove, Lemma, Target, & Fonagy, 2012), which can be easily integrated in multidisciplinary treatment for CFS (Kempke et al., 2010). However, it should be noted that there is currently no evidence-based treatment for SCP (Kempke, Luyten, Claes, Goossens et al., 2013).

In summary, this is the first study to show that SCP is associated with increased subjective stress responses, but decreased HPA-axis reactivity in CFS as indicated by lower salivary cortisol responses to experimentally-induced psychosocial stress. Future studies should examine whether chronic stress plays a mediating role in the relationship between SCP and cortisol stress reactivity in CFS. Furthermore, future research should elucidate the epigenetic mechanisms underlying the link between SCP and reduced cortisol reactivity in CFS. Indeed, recent studies have demonstrated that chronic exposure to severe stress, and early life stress in particular, is associated with methylation of the glucocorticoid receptor (GR) gene (Bick et al., 2012; Szyf & Bick, 2013), which is considered to be the key regulator of the HPA axis (Claes, 2009). Hence, it is reasonable to suggest that SCP along with other factors may be significantly related to epigenetic alterations of stress-related genes in CFS (Van Houdenhove et al., 2013).

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Table 1. Descriptive characteristics of the study sample (*N*=41).

Age	*M* (range)	41.95 (28-58)
	S.D.	7.89
Level of education	Primary education	2 (5.0%)
	Lower secondary education	4 (10.0%)
	Higher secondary education	16 (40.0%)
	Undergraduate degree	17 (42.5%)
	University degree	1 (2.5%)
Body mass index (BMI)	*M* (range)	25.48 (19-36)
	S.D.	4.48
SCID-I diagnoses	Current depression	7 (17.1%)
	Lifetime depression	28 (68.3%)
Severity of depression (HADS)	*M* (range)	8.63 (2-17)
	S.D.	3.85
Duration of symptoms (in years)	*M*	4.37
	S.D.	3.69

Table 2. Mean, S.D., and effect sizes of log-transformed cortisol levels for the high and low SCP group.

Parameter	High SCP	Low SCP	Effect size (Cohen's d)
AUC_G	*M*= 108.517	*M*= 145.715	d= .71
	S.D.= 28.290	S.D.= 70.207	
AUC_I	*M*= −18.858	*M*= 10.820	d= .92
	S.D.= 29.777	S.D.= 35.831	
Mean cortisol	*M*= 1.065	*M*= 1.451	d=.76
	S.D.= .291	S.D.= .671	
Delta cortisol	*M*= .147	*M*= .528	d= .99
	S.D.= .385	S.D.= .404	

Index: SCP=self-critical perfectionism; AUCG= Area under the curve with respect to the ground; AUCI= Area under the curve with respect to increase; Delta cortisol=peak minus baseline cortisol.

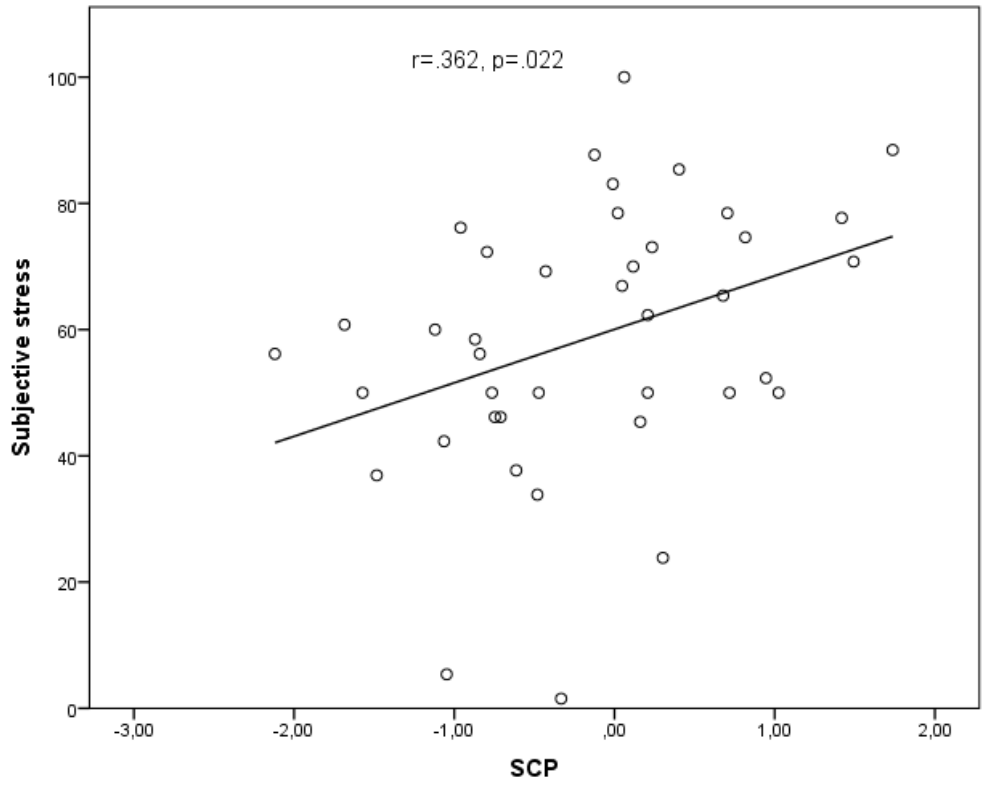

Figure 1. Scatterplot showing the positive correlation between self-critical perfectionism (SCP) and subjective (maximum) stress ratings.