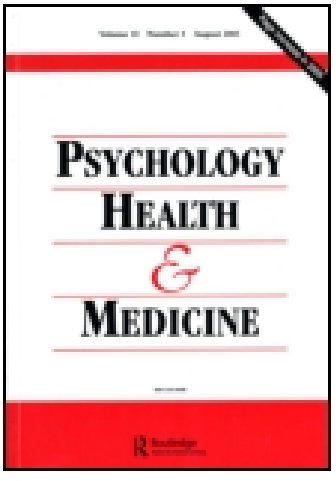

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Trait emotional intelligence and inflammatory diseases

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Trait emotional intelligence and inflammatory diseases

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Researchers have become increasingly interested in the psychological aspects of inflammatory disorders. Within this line of research, the present study compares the trait emotional intelligence (trait EI) profiles of 827 individuals with various inflammatory conditions (rheumatoid arthritis [RA], ankylosing spondylitis, multiple sclerosis, and RA plus one comorbidity) against 496 healthy controls. Global trait EI scores did not show significant differences between these groups, although some differences were observed when comparisons were carried out against alternative control groups. Significant differences were found on the trait EI factors of Well-being (where the healthy group scored higher than the RA group) and Sociability (where the healthy group scored higher than both the RA group and the RA plus one comorbidity group). The discussion centers on the multifarious links and interplay between emotions and inflammatory conditions.

Keywords: trait emotional self-efficacy; inflammation; rheumatoid arthritis; ankylosing spondylitis; multiple sclerosis; TEIQue

Introduction

There is clear evidence that psychological factors are implicated in the progression of a range of inflammatory conditions, whose impact on psychological well-being and quality of life they moderate through a variety of paths. The present study is an empirical investigation seeking to explore psychological factors in inflammatory conditions. The study reports profile differences between various inflammatory groups and healthy controls on trait emotional intelligence (trait EI).

Inflammation is a central process of the innate immune system. It protects organisms from the harmful effects of various physical, chemical, and biological pathogens. The inflammatory process confines, dilutes, and destroys the offending agents and aids the repair of damaged tissue. Chronic inflammation plays an important role in the pathogenesis of many common and debilitating illnesses, ranging from atherosclerotic cardiovascular diseases, to rheumatic diseases of the joints, and nervous system disorders like multiple sclerosis (MS).

Researchers have become increasingly interested in the psychological aspects of inflammatory disease. Several studies have demonstrated how the interactions between psychosocial and immune factors are relevant to various such diseases (Kiecolt-Glaser,

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McGuire, Robles, & Glaser, 2002a; O'Donovan et al., 2009). Kiecolt-Glaser et al. (2002a) show how negative emotions can enhance the production of proinflammatory cytokines. The overproduction of proinflammatory cytokines can contribute to immune dysregulation, which increases the risk of various diseases. Negative emotions may also contribute to prolonged and chronic infections, and to delayed wound healing, which can stimulate proinflammatory cytokine production.

Other studies have shown that negative emotions play a role in improving or worsening the course of inflammatory conditions. Indeed, the pathology of these conditions can often be very disabling and cause more stress and negative feelings than most non-inflammatory diseases. Studies have shown that pain, stiffness, fatigue, physical disability, and controllability by self-care are all relevant aspects that can modulate inflammatory diseases, and can thus influence quality of life for those living with a stressful condition (Chorus, Miedema, Boonen, & van der Linden, 2003; Fournier, de Ridder, & Bensing, 2002). MS, for e.g. is a largely uncontrollable inflammatory disease with limited self-care options. In this disease, emotional distress is more prevalent than in other chronic illnesses (Fischer & Crawford, 1994; Fournier et al., 2002).

Rheumatoid arthritis (RA) is characterized by symmetric and erosive synovitis of peripheral joints, and a progressive and unpredictable disease course that is punctuated by intermittent periods of disease flare and remission. The disease has a bigger impact on all aspects of quality of life than most other inflammatory diseases (Chorus et al., 2003; Pincus, 1996). For e.g. RA patients report lower levels of vitality, general health, and physical function compared to psoriatic arthritis, osteoarthritis, and ankylosing spondylitis (AS) patients (Chorus et al., 2003; Salaffi et al., 2009; Slatkowsky-Christensen, Mowinckel, Loge, & Kvien, 2007).

AS is a chronic disease that predominantly affects the pelvic skeleton, causing decreased spinal mobility. It appears to be less debilitating than RA (Boonen, de Vet, van der Heijde, & van der Linden, 2001; Chorus et al., 2003). Even if it seems less debilitating than other diseases, it can still create daily pain, stiffness, fatigue, and physical disability (Calin, 1995; Ward, 1999). Several studies have shown that AS patients have higher physician ratings of disease activity and severity compared to psoriatic arthritis patients (Zink et al., 2006). However, AS patients generally have higher pain thresholds compared to osteoarthritis patients (Gerecz-Simon, Tunks, Heale, Kean, & Buchanan, 1989).

Compared to healthy people, all patients with inflammatory conditions tend to score lower on quality of life and controllability, and higher on pain, anxiety, and depression (Kalia & O'Connor, 2005; Salaffi et al., 2009; Slatkowsky-Christensen et al., 2007; Tander et al., 2008). Fournier et al. (2002) suggested that individual differences in stress and negative emotion in patients with inflammatory diseases may be moderated by, amongst other variables, coping styles, social support, and personality traits. Adequate coping strategies, social support, and good social relations are themselves associated with better adjustment to disease, better quality of life, and reduced stress and depression (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002b; McCabe, 2006; Pakenham, 2006).

Other psychological aspects that can moderate stress and negative emotions are emotion control and emotion management. There is a growing consensus that better emotion control is associated with less pain and improved health in RA specifically (Hamilton, Zautra, & Reich, 2005; van Middendorp et al., 2005) and in chronic diseases more generally (de Ridder, Geenen, Kuijer, & van Middendorp, 2008). There is clear evidence that emotion-related variables are implicated in the progression of a range of inflammatory conditions, whose impact on patients' psychological well-being

and quality of life they moderate through a variety of paths. Hence, it is worthwhile to investigate the role of the affective aspects of personality in the context of inflammatory disease.

Trait emotional intelligence

Trait EI refers to a constellation of emotional self-perceptions located at the lower levels of personality hierarchies (Petrides, Pita, & Kokkinaki, 2007). Essentially, the construct concerns people's perceptions of their emotional abilities, which is why it has also been labeled as "trait emotional self-efficacy". Trait EI integrates the affective aspects of personality through 15 distinct facets that are combined into four broad factors (see Table 1).

An expanding body of evidence, including quantitative genetic investigations (Vernon, Petrides, Bratko, & Schermer, 2008) gives grounds for conceptualizing trait EI as part of human personality. Multiple studies have demonstrated that the construct is implicated in many important life domains, including medicine (e.g. Smith et al., 2012), stress (e.g. Laborde, Brüll, Weber, & Anders, 2011), mental health (Martins, Ramalho, & Morin, 2010; Petrides, Hudry, Michalaria, Swami, & Sevdalis, 2011), neuropsychology (Mikolajczak, Roy, Luminet, Fillée, & de Timary, 2007), academic performance (Sanchez-Ruiz, Mavroveli, & Poullis, 2013), decision-making (Telle, Senior, & Butler, 2011), and nursing (Quoidbach & Hansenne, 2009).

Table 1. The Sampling domain of trait EI in adults.

	High scorers perceive themselves as …
Well-being	
Self-esteem	… successful and self-confident
Trait happiness	… cheerful and satisfied with their lives
Trait optimism	… confident and likely to "look on the bright side" of life
Self-control	
Emotion control	… capable of controlling their emotions
Stress management	… capable of withstanding pressure and regulating stress
Impulsiveness (low)	… reflective and less likely to give into their urges
Emotionality	
Emotion perception (self and others)	… clear about their own and other people's feelings
Emotion expression	… capable of communicating their feelings to others
Relationships	… capable of having fulfilling personal relationships
Trait empathy	… capable of taking someone else's perspective
Sociability	
Social awareness	… accomplished networkers with excellent social skills
Emotion management (others)	… capable of influencing other people's feelings
Assertiveness	… forthright, frank, and willing to stand up for their rights
Adaptability*	… flexible and willing to adapt to new conditions
Self-motivation*	… driven and unlikely to give up in the face of adversity

Note: *This facet is not keyed to any factor, but feeds directly into the global trait EI score.

The present study

Given the strong influence of emotions on most aspects of everyday life, it is not surprising that trait EI plays a role across many different life domains. Especially, robust links exist between trait EI, coping styles, and emotions (e.g. Mikolajczak, Nelis, Hansenne, & Quoidbach, 2008; Mikolajczak, Petrides, & Hurry, 2009), which gives further and sufficient reason to investigate the construct's impact on inflammatory diseases.

The aim of the present study is to contribute in that direction. As previously mentioned, there is evidence that people with inflammatory diseases have elevated levels of anxiety, stress, and depression and that there are significant psychophysiological differences between different types of inflammatory disease. Our study explores the broader question of whether differences exist in the trait EI profiles of patients with various inflammatory conditions and healthy controls.

Method

Participants

Patient societies were approached in countries of comparable lifestyle (UK, Ireland, USA, Canada, Australia, and New Zealand). Societies in the UK and Canada volunteered to participate. Two societies emailed approximately 3000 invitations to their members to complete our online questionnaire and two others posted an advertisement on their website, leading to a slow "trickle" of additional participants. In total, 873 adults diagnosed with a range of several inflammatory diseases took part in the study. They were subsequently divided into groups according to their primary disease type: RA: 648 subjects, MS: 101 subjects, PA: 21 subjects, and AS: 31 subjects. Two further groups were created according to the presence of other inflammatory comorbidities: RA with one comorbidity (RA + 1: 47 subjects) and RA with two or more comorbidities (RA + 2: 25 subjects). Last, a healthy group of 496 participants was randomly drawn from the TEIQue normative database. The size of the control group was chosen to approximate the size of the RA group, which was the largest in the study.

Following preliminary analyses, the two smallest groups ("Psoriatic Arthritis": 21 subjects; "RA with two or more other diseases in comorbidity": 25 subjects) were excluded from the overall sample of 1369 participants due to their small size, which was further reduced because of missing data on relevant covariates. Thus, the final sample comprised 1323 participants (305 male, 820 female, and 198 not indicated). It was ethnically diverse, with 64% of participants being White-UK, 13% White-other, 12% White-Canadian, 2% Black-Caribbean, 2% Chinese, and a further 7% from other backgrounds.

Measures

Trait EI questionnaire

The TEIQue v. 1.50 (Petrides, 2009) was used to measure trait EI. This is a 153-item inventory that assesses 15 trait EI facets, four higher-order factors (well-being, self-control, emotionality, and sociability) and a global trait EI score combing scores on the 15 facets. It utilizes a 7-point Likert scale, ranging from completely disagree to completely agree. Raw scores on all of its variables range from a low "1" to a high "7". Internal consistencies (Cronbach's α) for the four factors and global trait EI in the present study were high in all cases (see Table 2).

Socio-demographic and disease details

Participants were asked questions about age, gender, ethnic background, type of disease, and age at onset of symptoms.

Results

Table 2 summarizes the means and standard deviations for the five trait EI variables (four factors plus global trait EI) and the demographics of age and age of onset of symptoms.

ANOVA and ANCOVA

First, we computed a univariate ANOVA with global trait EI as the dependent variable and participant group (RA, AS, MS, RA plus one comorbidity, and healthy controls) as the independent variable. This returned a nonsignificant main effect of group, $F_{(4,\ 1318)} = .996$, $p = .409$. An identical ANCOVA, with participant age as the covariate, was also nonsignificant, $F_{(4,\ 1117)} = 1.874$, $p = .113$.

MANOVA and MANCOVA

A MANOVA was carried out with the four trait EI factors as the dependent variables and the five aforementioned participant groups as the independent variable. There was a significant multivariate main effect of group [Wilks' Lambda = .925, $F_{(16,\ 4018)} = 6.45$, $p < .001$, $\eta_p^2 = .019$] originating from significant differences on Well-being, $F_{(4,\ 1318)} = 3.592$, $p = .006$, $\eta_p^2 = .011$, and Sociability $F_{(4,\ 1318)} = 8.34$, $p < .001$, $\eta_p^2 = .025$, as revealed by the follow-up ANOVAs. Hochberg's GT2 post hoc tests indicated that the control group scored significantly higher on Well-being ($p = .014$) and Sociability ($p < .01$) compared to the RA group as well as on Sociability compared to the "RA plus one comorbidity" group ($p = .017$).

Table 2. Descriptive statistics for the key variables in the study broken down across the inflammatory and control groups.

		RA ($n = 648$)		MS ($n = 101$)		AS ($n = 31$)		RA + 1 ($n = 47$)		Controls ($n = 496$)	
	α	*M*	SD	*M*	SD	*M*	SD	*M*	SD	*M*	SD
Age	–	51.02	11.39	44.43	10.46	46.12	10.99	50.53	12.97	41.54	10.04
Age at onset of symptoms	–	38.65	13.35	31.75	8.25	25.35	11.68	34.51	15.19	–	–
No. of years since symptoms began	–	11.35	9.86	11.68	8.96	19.77	9.75	14.41	14.16	–	–
Well being	.866	5.08	1.06	5.00	1.07	5.33	1.15	5.24	.92	5.27	.82
Self-control	.794	4.65	.86	4.60	.76	4.64	.92	4.65	.93	4.66	.80
Emotionality	.809	5.18	.81	5.14	.76	5.27	.84	5.01	.83	5.08	.74
Sociability	.817	4.70	.82	4.88	.88	4.75	.99	4.57	.90	4.95	.75
Global trait EI	.917	4.90	.71	4.88	.69	4.98	.78	4.88	.72	4.97	.61

Notes: RA: rheumatoid arthritis; MS: multiple sclerosis; AS: ankylosing spondylitis; RA + 1: rheumatoid arthritis plus one comorbidity.

Subsequently, an identical MANCOVA was performed, controlling for participant age. It showed that the differences between the RA and control groups persist even after participant age has been controlled for. Thus, significant multivariate effects were found both for group [Wilks' Lambda = .928, $F_{(16,\ 3404)} = 5.24$, $p < .01$, $\eta_p^2 = .018$] and for the covariate (participant age) [Wilks' Lambda = .944, $F_{(4,\ 1114)} = 16.56$, $p < .001$, $\eta_p^2 = .056$]. The follow-up ANCOVAs revealed that the multivariate effects stemmed mainly from significant differences in the adjusted means of Well-being $F_{(4,\ 1117)} = 3.012$, $p = .017$, $\eta_p^2 = .011$ and Sociability, $F_{(4,\ 1117)} = 7.205$, $p < .001$, $\eta_p^2 = .025$.

The second MANCOVA was carried out, with the four trait EI factors as the dependent variables, the four inflammation groups as the independent variable, and participant age and age at onset of symptoms as the covariates. Age had a significant main effect [Wilks' Lambda = .967, $F_{(4,\ 614)} = 5.28$, $p < .001$, $\eta_p^2 = .033$], while group [Wilks' Lambda = .972, $F_{(12,\ 1624)} = 1.45$, $p < .136$, $\eta_p^2 = .009$] and age at onset of symptoms [Wilks' Lambda = .990, $F_{(4,\ 614)} = 1.55$, $p = .185$, $\eta_p^2 = .010$] did not reach significance. We also ran a similar MANCOVA with number of years since symptoms began instead of age at onset of symptoms as a covariate, which yielded substantively identical results.

Additional analyses

In order to establish the veracity of our findings, a number of additional analyses were carried out, comparing the inflammatory groups against two different control groups:

(a) a group comprising the entire normative sample of the TEIQue ($n = 2254$; 1086 males, 1104 female, and 64 missing). All the differences reported in the main analyses were replicated. In addition, there were significant RA vs control differences in global trait EI and MS vs. control differences in the well-being factor. In all cases, the inflammatory groups scored lower than this control group;

(b) a group more closely matched on age with the inflammatory groups ($n = 538$; age: $M = 47.54$, SD = 8.56). This control group was drawn from the TEIQue UK normative database. To align the age distribution of the groups, as suggested by a reviewer, we categorized participants in three age bands: below 22 years ($n = 556$); between 22 and 44 years ($n = 1340$); and above 44 years ($n = 358$). All participants aged below 22 years were excluded. From the age group between 22 and 44, we randomly selected approximately 15% of cases that were subsequently added to the subjects aged above 44 years. All the differences reported in the main analyses were replicated. In addition, the RA, MS, and RA + 1 groups differed significantly from controls in global trait EI, the RA group differed from controls in Self-control, the MS group differed from controls in Well-being, Self-control, and Sociability, and the AS group differed from controls in Sociability. In all cases, the inflammatory groups scored lower than this control group. More detailed comparisons at the level of the 15 facets are reported in a separate paper (Tillmann, Krishnadas, Cavanagh, & Petrides, 2013).

Discussion

With respect to global trait EI scores, our main analyses did not show any significant differences between healthy participants and participants with inflammatory diseases, although some of the additional analyses with alternative control groups suggested such

differences. There were significant differences in the trait EI factors of Well-being and Sociability. These differences persisted after controlling for age. *Posthoc* tests indicated that individuals with RA reported lower levels of Well-being and Sociability than healthy controls. The group with RA and one other comorbidity also reported lower levels of Sociability than the control group. These findings confirm previous impressions that individuals with inflammatory diseases, such as RA, tend to score lower than healthy controls on several aspects of mental health. Additional analyses conducted with alternative control groups verified these profile differences and also suggested further differences between the groups.

The findings raise an interesting question about causality. Were the observed differences in trait EI present before disease onset or did they develop as a response to the disease? Without longitudinal data it is not possible to provide a definitive answer to this question. If the differences arose as a response to illness, reduced Sociability may be the end result of physical pain, functional immobility, and fear of social disapproval. On the other hand, if decreased socialization was present *before* disease onset, then it may itself constitute an additive risk factor in the pathogenesis of inflammatory diseases.

The fact that time since onset of symptoms did not reach significance in a MANCOVA reduces the likelihood that the observed differences were a response to illness. Furthermore, the fact that personality is generally stable over time (Terracciano, McCrae, & Costa, 2006) also suggests that the trait EI differences could have been present before disease onset and may have rendered low trait EI individuals more susceptible to the effects of chronic stress. Chronic stress leads to over-activation and dysregulation of the Hypothalamus-Pituitary Axis, which in turn dysregulates inflammatory systems and perpetuates diseases like RA in certain people (Imrich, 2002; Sternberg, Silverman, & Cizza, 2007). Longitudinal studies investigating the causes of inflammatory conditions will, therefore, benefit from incorporating trait EI measures alongside other primary variables of interest.

A limitation of our study is the fairly small size of certain subgroups, which may have resulted in relatively low statistical power, and which also prevented us from including other demographic characteristics, such as gender or socioeconomic status, as additional independent variables that would have allowed for the modeling of interaction effects. Furthermore, due to the limit on the number of questions we could ask and incorporate into the analyses, we did not examine any possible effects of educational differences in our study. This omission is worth noting because research has shown that more educated participants are more likely to seek care and counselling (Erlyana, Damrongplasit, & Melnick, 2011; Patti et al., 2007).

Future research should continue to explore how various aspects of inflammatory disease can be alleviated as well as how they impact on quality of life, particularly during times of emotional turmoil. While our design cannot give an exhaustive account of how psychology is implicated in inflammatory diseases, it does add to a growing body of data revealing robust links between physical and mental (especially emotional) health.

Conclusion

Trait EI provides a useful framework within which to organize our understanding of the interplay between affective and immune factors in the cause and course of inflammatory diseases. Our findings represent a timely contribution to the literature in the light of recent descriptions of how negative emotions, such as depression, anxiety, and chronic

stress, can affect inflammatory processes. At this stage, we have only seen snapshots of the dynamic impact of trait EI on inflammatory conditions, and thus are not in a position to propose any casual mechanisms or models. It remains a possibility that trait EI differences may have been present before disease onset. On this ground alone, trait EI should be investigated further in relation to its role in the process of inflammation. As a promising first step, trait EI measures could be included as baselines in prospective longitudinal studies attempting to chart the individual's affective development alongside the course of their condition.

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