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Cognitive, behavioral and physiological reactivity to pain as a predictor of long-term pain in rheumatoid arthritis patients

Andrea W.M. Evers[a,*], Floris W. Kraaimaat[a], Piet L.C.M. van Riel[b], Johannes W.J. Bijlsma[c]

[a]*Department of Medical Psychology, University of Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands*
[b]*Department of Rheumatology, University Medical Center St. Radboud, Nijmegen, The Netherlands*
[c]*Department of Rheumatology and Clinical Immunology, University Medical Center Utrecht, Utrecht, The Netherlands*

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Abstract

A heightened reactivity to pain is assumed to play a significant role in the maintenance and exacerbation of pain in patients with chronic pain. In a prospective study involving 95 rheumatoid arthritis (RA) patients, the relative contribution of self-reported cognitive, behavioral and physiological components of pain reactivity were examined for a change in pain within 1 year. Regression analyses indicated that self-reported physiological reactivity predicted an increase in clinical and self-reported pain after 1 year, but not cognitive and behavioral reactivity. Neither disease activity nor neuroticism mediated or moderated the relationship of pain reactivity to long-term pain. However, structural equation modeling revealed that neuroticism directly affected physiological reactivity to pain, which in turn was the only significant predictor for subsequent pain. The results of this study underline the crucial role of physiological pain reactivity for exacerbation of pain in RA patients and are indicative for a symptom-specific pattern of physiological pain reactivity that is sustained by psychological predisposition and respondent learning processes.

Keywords: Pain reactivity; Cognitive; Behavioral; Physiological; Response systems; Chronic pain

1. Introduction

Pain is the most prominent physical complaint in rheumatoid arthritis (RA), a chronic inflammatory disease that primarily affects the joints. Although pain is a direct consequence of the disease process, patients' pain reports are usually only moderately related to the underlying pathology, and pain frequently becomes a problem in its own right. Variability in biomedical and psychosocial treatment outcomes, primarily geared to reducing the aversive consequences of pain instead of eliminating it, also indicate that pain remains one of the most complex factors in chronically painful disorders such as RA.

In recent decades, biopsychosocial approaches have conceptualized pain as a multifaceted phenomenon that consists of at least three response systems, i.e. motor-behavioral, subjective-cognitive, and sensory-physiological components (Philips, 1977; Epstein et al., 1978; Lethem et al., 1983; Flor et al., 1990). In line with theories of emotion (e.g. Lang, 1968; Rachman and Hodgson, 1974; Borkovec et al., 1977), it is assumed that the degree of synchrony of these response systems varies: they are not necessarily commonly activated, might be maintained by different factors and might have differential effects on treatment outcomes. Studying their functional interrelationships and effects on long-term outcomes could provide a better understanding of the specificity of processes responsible for the maintenance and exacerbation of chronic pain.

In acute pain, pain responses involve the behavioral reaction of interrupting activity, cognitive attempts to direct attention to the aversive experiences to find a reasonable cause for the pain and prevent further damage, as well as physiological processes of heightened autonomic, somatosensory and central nervous system activity (e.g. Flor et al., 1990). In as much as these responses are immediately triggered and functional for survival in instances of acute pain, they may be more loosely related and less protective in the event of chronic pain. In fact, maintenance of these reactions is thought to sustain and exacerbate pain and related outcomes, such as functional disability and depression. Based on predisposition and learning mechanisms, a habitual pattern of reactivity to pain, including avoidance behavior, cognitive preoccupation with bodily signals and

* Corresponding author. Tel.: +31-24-3613608; fax: +31-24-3613425.
E-mail address: a.evers@cksmps.azn.nl (A.W.M. Evers).

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heightened physiological arousal, might become increasingly chronic. This habitual pattern might be generalized to various stimuli associated with pain, function relatively independently of objective pathology and intensity of pain, and subsequently affect long-term pain outcomes (e.g. Lethem et al., 1983; Linton, 1985; Philips, 1987; Flor et al., 1990; Turk and Flor, 1999). Experimental and quasi-experimental studies have provided considerable evidence for the existence of these response patterns in chronic pain patients and for their maladaptive effects on pain outcomes, including pain itself.

Behavioral approaches predominantly focus on the prominent role of avoidance behavior in the maintenance and exacerbation of chronic pain through processes of external reinforcement or anticipatory anxiety (Fordyce, 1976; Lethem et al., 1983; Linton, 1985; Philips, 1987). A considerable amount of research assessing avoidance behavior on the basis of observed or self-reported pain behavior (e.g. Philips and Jahanshahi, 1985b; Vlaeyen et al., 1990; Jensen et al., 1995) has demonstrated the major role of avoidance and its relationship to worse long-term outcomes in various chronic pain populations, including RA (Evers et al., 1998a; van Lankveld et al., 1999, 2000). Experimental studies have also supplied preliminary evidence for the maladaptive effects of avoidance on pain, demonstrating, for example, that avoidance of exposure can lead to decreased tolerance of stressful stimulation in migraine patients (Philips and Jahanshahi, 1985a).

Research on cognitive factors has emphasized the role of negative outcome expectancies with concepts such as catastrophizing or excessive worrying in the face of pain (see Keefe et al., 1989; Jensen et al., 1991; Turk and Rudy, 1992; Aldrich et al., 2000). Results from pain-coping and cognition measures that assess the extent that patients tend to catastrophize in the face of pain (e.g. Rosenstiel and Keefe, 1983; Sullivan et al., 1995; Kraaimaat et al., 1997) have provided considerable evidence for the prominent role of these cognitions in various chronic pain patients and their relationships to unfavorable pain outcomes (e.g. Keefe et al., 1989; Affleck et al., 1992; Martin et al., 1996). Moreover, experimental and longitudinal studies support the unfavorable effects of catastrophic thoughts on pain. For example, catastrophizing has been demonstrated to affect pain tolerance and pain intensity in experimentally-induced pain (e.g. Spanos et al., 1979; Geisser et al., 1992; Sullivan et al., 1995). In prospective studies, catastrophizing or worrying in the face of pain predicted a worsening of various pain outcomes in RA patients (Keefe et al., 1989; Evers et al., 1998a), including pain itself (Keefe et al., 1989).

On the sensory-physiological level, reactivity to pain has been assumed to be particularly manifest in increased autonomic and muscular reactivity as well as the sensitization of central structures (Flor et al., 1990; Turk and Flor, 1999). Based on predisposition and/or respondent learning processes, these responses might develop in a chronic condition into a consistent, habitual pattern of reactivity to pain and pain-related stimuli that affect pain and related outcomes (e.g. Flor et al., 1990; Turk and Flor, 1999). Evidence for stress- or pain-related patterns of heightened autonomic, somatosensory and/or central responses has been provided among various chronic pain patients, including those with RA (Salamy et al., 1983; Flor et al., 1985, 1992a, 1997; Jamner and Tursky, 1987; Lutzenburger et al., 1997; see also Anderson et al., 1985; Flor and Turk, 1989). Preliminary support also exists for the maladaptive function of physiological reactivity patterns on pain outcomes. For example, stress-induced increases in symptom-specific muscular tension predicted greater pain severity in depressed patients with chronic low back pain (Burns et al., 1997). In addition, self-reported autonomic arousal in the face of pain, assessed as pain-related fears, predicted greater pain severity and more physical complaints in heterogeneous groups of chronic pain patients (McCracken et al., 1996, 1998).

In conclusion, there is empirical support for a multidimensional reactivity pattern to pain and its maladaptive effects in chronic pain patients. However, research usually focuses on just one component of response systems, such as pain behaviors, cognitive constructs or physiological responses (see Philips, 1987; Flor et al., 1990; McCracken et al., 1996). Other conceptualizations assess reactivity to pain as composite constructs without differentiating between response systems (see Jensen et al., 1991), such as pain-coping measures with confounding behavioral and cognitive responses (e.g. Brown and Nicassio, 1987). Both approaches preclude information about the possibly variable interrelationship between the response systems under different conditions and their relative contribution to pain outcomes. Integration and systematic comparison of these response systems could possibly clarify their common and independent response effects on chronic pain and provide a better understanding of the specificity of mechanisms underlying long-term pain.

Stress-vulnerability models suggest that the possible independent effects of the response systems might be the result of being differently determined by biomedical pathology and psychological vulnerability factors. It is usually assumed that pain reactivity is initiated and maintained by biomedical factors in an acute stage, but functions increasingly independently in a chronic stage (e.g. Lethem et al., 1983; Flor et al., 1985, 1990; Philips, 1987). However, since most of the research has been conducted with benign pain syndromes, where there is no biomedical indicator of pathology, the role of biomedical factors may be systematically underestimated. In pain syndromes with an underlying pathology of inflammatory activity, such as RA, where patients are recurrently confronted with unpredictable pain flare-ups, a habitual pattern of pain reactivity may be directly triggered and maintained by the disease process. In addition, pain reactivity has been demonstrated to be affected by psychological vulnerability factors, such as

neuroticism or negative affectivity. Neuroticism and negative affectivity have been demonstrated to be related to avoidance behavior (Harkins et al., 1989; Wade et al., 1992), catastrophizing (Affleck et al., 1992; Martin et al., 1996) and physiological reactivity in chronic pain patients (Vlaeyen et al., 1999), suggesting a common underlying predisposition that possibly mediates or moderates the effect of pain reactivity on long-term pain (e.g. Affleck et al., 1992; Martin et al., 1996; Burns et al., 1997; Vlaeyen et al., 1999).

The purpose of the present study was to study the interrelationships of cognitive, behavioral and physiological response systems of pain reactivity in patients with RA and their concurrent relationships to disease severity and neuroticism. In addition, our object was to prospectively determine the role of these response systems for the long-term prediction of pain and study possible mediating or moderating effects of disease activity and neuroticism on this relationship. It was hypothesized (1) that the three response systems would demonstrate closer relationships to neuroticism than to measures of disease severity, and (2) that initially higher levels of the response systems would predict an increase in pain within 1 year, after controlling for disease severity and neuroticism.

2. Methods

2.1. Participants

The sample consisted of 95 outpatients with RA from two participating hospitals in the Netherlands. Inclusion criteria were a minimum age of 18 years and a diagnosis of RA according to American College of Rheumatology criteria (Arnett et al., 1988). The sample was predominantly female (61%) and married (85%) with at least a primary or secondary level of education (28 and 63%, respectively). The mean age was 58.9 years (SD 11.6, range 33–82 years). The mean time since diagnosis was 15.9 years (SD 9.2, range 4–45 years).

2.2. Measures

Several clinical and self-report measures were assessed in the sample at two assessment points, with a mean time interval of 1 year.

(1) Pain was assessed with a composite score of both clinical and self-report measures. Clinical pain ratings comprised the number of painful joints (Fuchs et al., 1989). Self-reports of pain were assessed with the Impact of Rheumatic Diseases on General Health and Lifestyle (IRGL) Pain Scale (six items), a disease-specific scale for arthritis patients that assesses the severity and frequency of painful episodes and swollen joints and the duration of morning stiffness in the past month (Huiskes et al., 1990; Evers et al., 1998b). Previous research showed the reliability and validity of the IRGL to be highly satisfactory (Huiskes et al., 1990; Evers et al., 1998b). Cronbach's alpha of the pain scale in the present study was 0.86.

(2) Disease activity was assessed with standardized erythrocyte sedimentation rate (ESR) laboratory measurements, which is an indicator of inflammatory activity in RA.

(3) Cognitive and behavioral reactivity to pain were assessed with the Pain Coping Inventory (PCI; Kraaimaat et al., 1997; Evers et al., 1998a), a pain-coping instrument which measures different cognitive and behavioral ways of dealing with pain on a four-point Likert scale, ranging from 'rarely or never' (1) to 'very frequently' (4). Cognitive reactivity was assessed with the passive pain-coping scale Worrying (nine items), which measures negative pain cognitions. Representative items were: 'I start worrying when in pain' or 'I think that the pain will worsen'. Behavioral reactivity was assessed with a composite score of the passive pain-coping scales Resting and Retreating (12 items), measuring behavioral tendencies to restrict functioning and avoid environmental stimuli, respectively. Representative items of these scales were: 'I quit my activities', 'I rest by sitting or lying down' or 'If I am outdoors, I try to return home as soon as possible'. The reliability and validity of the PCI was supported by previous research on patients with RA, patients with chronic headache pain and patients attending pain clinics (Kraaimaat et al., 1997; Evers et al., 1998a). Cronbach's alpha in the present study was 0.74 for the cognitive and 0.77 for the behavioral reactivity to pain.

(4) Physiological reactivity to pain was measured by various self-reported physiological reactions to pain, partly derived from the Physiological Anxiety Scale of the Pain Anxiety Symptoms Scale (McCracken et al., 1992). Respondents were asked to indicate how frequently they experience physiological reactions in the face of pain on a four-point Likert scale, ranging from 'rarely or never' (1) to 'very frequently' (4). From a total pool of eight items, four items (i.e. trouble catching breath, heart racing, pressure in chest and panicking) had to be eliminated due to the infrequency of responses endorsed (skewness or kurtosis >1.5). The items retained were: 'When in pain, I become dizzy or weak', 'I start sweating when in pain', 'I become restless when in pain' and 'When in pain, I have a tight or tense feeling in my body'. Internal scale consistency proved to be sufficient, as indicated by Cronbach's alpha of 0.71.

(5) Neuroticism was measured by a Dutch version of the Eysenck Personality Questionnaire (Wilde, 1963; Eysenck and Eysenck, 1992). Cronbach's alpha in the present sample was 0.85.

3. Results

3.1. Patient characteristics

Regardless of the long-term duration of arthritis in our sample (the duration of disease was approximately 16 years), disease activity and pain levels were comparable to

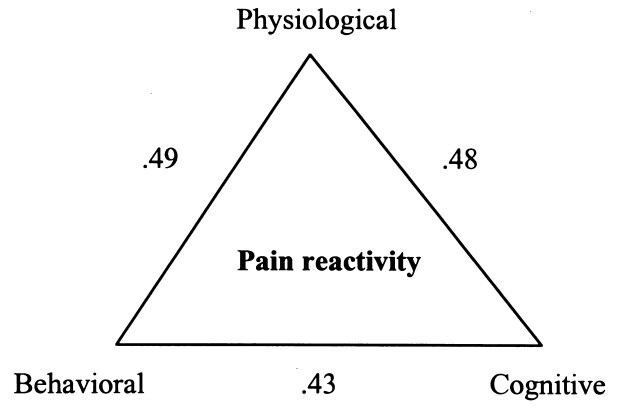

Fig. 1. Interrelationships between response systems of pain reactivity. All correlations are significant at $P < 0.001$.

what have been previously reported in representative RA samples (Huiskes et al., 1990; Evers et al., 1998b). On average, the moderate level of disease severity remained after 1 year, as indicated by non-significant changes in disease activity and pain. In addition, mean scores of the pain reactivity response systems were relatively stable and did not change within 1 year.

In terms of individual changes in the dependent variable, however, a review of the scatter plot indicated that there was considerable individual variation in pain, and 28% ($n = 26$) and 62% ($n = 58$) of the patients, respectively, showed a worsening or improvement in pain of 1 SD and 0.5 SD during the study period.

3.2. Correlates of pain and pain reactivity

Pearson correlation coefficients between the cognitive, behavioral and physiological components of pain reactivity at first assessment indicated a moderate correlation between the different response systems (between 0.43 and 0.49; see Fig. 1). In addition, all response systems demonstrated similar correlations with disease activity, pain and neuroticism (see Table 1). While correlations with disease activity were all non-significant, the response systems were weakly related or tended to be related to pain and all were moderately related to neuroticism, indicating more pain reactivity in patients with higher levels of pain and neuroticism. Finally, pain was also weakly related to higher levels of disease activity and neuroticism (for both $r = 0.28$, $P < 0.01$).

Table 1
Correlates of response systems of pain reactivity

	Behavioral	Cognitive	Physiological
Disease activity	0.07	0.02	0.16
Pain	0.18	0.23*	0.23*
Neuroticism	0.42***	0.49***	0.35**

***$P < 0.001$, **$P < 0.01$, *$P < 0.05$.

3.3. Predictors of long-term pain

Pearson correlation coefficients between the pain reactivity response systems at first assessment and residual gain scores of pain were calculated to explore the relationship between pain reactivity and long-term pain. Results indicated that one of the three response systems was significantly related to an increase in pain, i.e. physiological reactivity ($r = 0.40$, $P < 0.001$). In addition, behavioral reactivity tended to correlate with an increase in pain ($r = 0.20$, $P < 0.10$), while the correlation with cognitive reactivity was non-significant ($r = 0.07$, NS).

Stepwise multiple regression analyses were then performed to examine the relative contribution of pain reactivity to the change in pain within 1 year, after controlling for possible confounding variables. Pain at second assessment was used as the dependent variable, controlling for the baseline scores of pain in the first step. The other control variables were entered in step 2, i.e. demographic variables (gender, age and educational level), disease activity and neuroticism, all measured at first assessment. In step 3, the different components of pain reactivity at first assessment were entered in the regression analyses. Results indicated that the best predictor for pain at second assessment was the initial level of pain, explaining 32% of the total variance. The control variables in step 2 did not add any variance. Pain reactivity in step 3, however, added 10% of the variance. Beta coefficients demonstrated that physiological reactivity significantly predicted an increase in pain after 1 year, but not cognitive and behavioral reactivity (see Table 2). When entering the different pain reactivity response systems separately in the regression analyses, again only physiological reactivity explained significant variance in long-term pain.

As visible from the results of Table 2, neither disease activity nor neuroticism mediated the effects of pain reactivity on long-term pain. To study possible moderator effects of disease activity and neuroticism, centered interaction terms with all pain reactivity components were entered in the regression analyses in step 4. Results again indicated that neither disease activity nor neuroticism

Table 2
Multiple regression analysis predicting long-term pain

	Beta	Adjusted R^2
1. Pain T1[a]	0.38***	0.32***
2. Control variables T1[b]		0.00
3. Pain reactivity T1		0.10***
Behavioral	0.08	
Cognitive	0.10	
Physiological	0.33**	
Total adjusted R^2		0.42***

[a] T1, first assessment.
[b] Control variables: demographic variables (gender, age and educational level), neuroticism T1 and disease activity T1.
***$P < 0.001$, **$P < 0.01$.

moderated the relationship between pain reactivity and long-term pain.

The relative contribution of pain reactivity to subsequent pain was then tested in structural equation modeling, when taking the effects of disease activity and neuroticism into account (AMOS 4.0, Arbuckle, 1994). The same models were set up for all pain reactivity components in which the effects of pain reactivity at first assessment were tested against subsequent pain, controlling for pain, neuroticism and disease activity at first assessment. As in the regression analyses, only the model for physiological reactivity provided a significant path to long-term pain. After omitting non-significant paths from this model (from disease activity to physiological pain reactivity, as well as from disease activity and neuroticism to subsequent pain), an excellent fit was revealed for the final model, in which neuroticism directly affected the physiological reactivity to pain. In turn, physiological reactivity was the only significant predictor of subsequent pain ($\chi^2(2) = 1.89$, $P = 0.39$; Goodness of Fit Index (GFI) = 0.99; Tucker Lewis Index (TLI) = 1.00; Incremental Goodness of Fit Index (IFI) = 1.00; see Fig. 2).

4. Discussion

A heightened reactivity to pain is assumed to contribute to the maintenance or exacerbation of pain in patients with chronic pain (e.g. Flor et al., 1990; Turk and Flor, 1999). However, little systematic research has been conducted on the different pain reactivity response systems (cognitive, behavioral and physiological) and their predictive value for pain in chronic pain patients. The focus of our study was to examine interrelationships of the pain reactivity response systems, their relationship to biomedical and psychological vulnerability factors and their effects on long-term pain in RA patients.

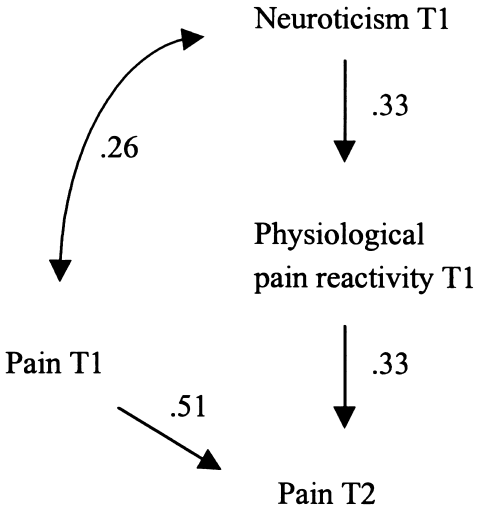

Fig. 2. The significant paths (standardized regression coefficients) of the structural equation modeling, testing effects of pain reactivity at first assessment (T1) against pain at second assessment (T2), controlling for pain and neuroticism at first assessment (T1). Non-significant paths are omitted from the analyses. In addition, error variances are omitted from the figure for convenience of presentation. All paths are significant at $P < 0.01$.

According to the assumed desynchrony of the response systems, the self-reported behavioral, cognitive and physiological components were moderately intercorrelated. This moderate degree of interdependence indicates that the response systems are not necessarily commonly activated and represent different dimensions of the pain experience, although they affect and probably enhance each other. In addition, all response systems demonstrated relatively uniform relationships to measures of disease severity and neuroticism. In accordance with what has previously been proposed on the basis of stress-vulnerability models (e.g. Flor et al., 1985, 1990), pain reactivity was hardly affected by disease severity, suggesting that it becomes a habitual response pattern in chronic pain and functions independently of actual pathology. In addition, the uniform relationship to neuroticism indicates a common underlying predisposition for vulnerability to stress. This psychological diathesis seems to be a relatively general predispositional factor for heightened reactivity to pain, since correlations between neuroticism and avoidance behavior (Harkins et al., 1989; Wade et al., 1992), catastrophizing (Affleck et al., 1992; Martin et al., 1996) and physiological reactivity (Vlaeyen et al., 1999) have previously been reported in various chronic pain populations. Regardless of the similar relationships to these stress-vulnerability factors, response systems differently affected long-term pain. Results of multiple regression and structural equation modeling clearly indicated that the self-reported physiological reactivity to pain was the only significant predictor of subsequent pain, independent of the effect of initial pain, disease activity, neuroticism and the other response systems. These results are in line with a previous cross-sectional study in which the self-reported physiological responses to pain predicted pain severity in a heterogeneous group of chronic pain patients, but not the behavioral or cognitive responses (McCracken et al., 1996). However, as far as we know, this is the first study that has compared response system effects on long-term outcomes and demonstrated maladaptive effects of self-reported physiological reactivity on chronic pain. Different physiological and/or cognitive-attentional mechanisms may account for these results.

Since the self-reported physiological reactivity was not related to disease activity and only very modestly to the intensity of present pain, it is unlikely that it represents symptomatic manifestations of the RA disease process. This lack of relationship to disease severity and the positive relationship to neuroticism instead suggest that it may be part of a psychophysiological response pattern. Peripheral physiological reactivity patterns in response to stressful and painful events as well as delayed return to baseline responses have previously been reported in various chronic pain patients (see Flor and Turk, 1989), including those with RA (e.g. Fisher and Cleveland, 1960; Moos and Engel,

1962; Walker and Sandman, 1977; Anderson et al., 1982), indicating heightened and/or prolonged muscular and autonomic reactivity when exposed to pain or stress.

The issue arises in so far as this self-reported physiological reactivity pattern reflects a symptom-specific physiological response in RA patients, as repeatedly reported in research on chronic benign pain (see Flor and Turk, 1989). Heightened EMG levels found only near painful joints support such a response specificity for RA patients as well (Moos and Engel, 1962; Walker and Sandman, 1977). In fact, the distribution of the self-reported physiological reactions initially assessed in our sample may indicate a symptom-specific pattern. Four items that primarily reflected respiratory and cardiovascular reactions had to be eliminated, due to the infrequency of endorsed responses. In contrast, this differentiation of the response pattern has not been reported in other chronic pain patients, where an adjusted version of the physiological reactivity scale (including respiratory and cardiovascular responses) has been used (McCracken et al., 1992, 1996, 1998; Larsen et al., 1997). To further explore the issue of response specificity, post-hoc item analyses of the physiological reactivity scale were performed. Results indicated that all items demonstrated similar relationships to subsequent pain, suggesting a physiological response pattern consisting of both autonomic and somatosensory components that affect long-term pain. This pattern may be in agreement with the heightened and prolonged EMG and skin conductance levels that have been reported most consistently in RA patients when comparing physiological reactivity patterns to other chronic diseases (Fisher and Cleveland, 1960; Moos and Engel, 1962; Walker and Sandman, 1977; Anderson et al., 1982; see Anderson et al., 1985). Assuming a physiological basis for our self-report scale, comparisons of psychophysiological response patterns between patients with RA and other chronic pain populations may clarify whether this reflects a RA-specific pattern or is part of a general reactivity pattern in chronic pain disorders.

Symptom-specific physiological patterns have frequently been found as reactions to pain-related or personally relevant stressors, suggesting that they may be enhanced by respondent learning processes (Flor et al., 1990; Turk and Flor, 1999). Respondent learning processes may also explain the great individual differences in self-reported physiological pain reactivity in our sample. Only 40% of the patients reported the physiological reactions to pain at least sometimes. In this subgroup, autonomic and somatosensory reactivity might have become a conditioned response to pain that maintains a pain-tension circle and exacerbates pain in the long run (Flor et al., 1990; Knost et al., 1999; Turk and Flor, 1999). However, it could also be argued on the basis of respondent learning processes that the self-reported physiological reactivity reflects general anxiety arousal, as suggested by research on pain-related fears (e.g. McCracken et al., 1992, 1996). Pain may then be enhanced due to anxiety-related autonomic and somatosensory activation in anticipation and as a consequence of pain (Flor et al., 1990; Turk and Flor, 1999). Although the physiological responses of both pain and anxiety have been demonstrated to be highly confounding and have considerable overlap (Gross and Collins, 1981), the kind of self-reported physiological reactivity in our sample, lacking cardiovascular and respiratory responses that are typical for the presence of general anxiety syndromes (Borkovec et al., 1977), do not solely support anxiety-related physiological reactions.

Admitting the limitation of self-report measures, the self-reported physiological reactions may also reflect a bias in attentional and interpretational processes, for example a tendency to amplify pain-related responses, as suggested by research on hypochondria and hypervigilance (Pennebaker and Skelton, 1978; Barsky et al., 1988; Chapman, 1986; Rollman and Lautenbacher, 1993). Chronic pain patients have been shown to overemphasize physical symptoms (Flor et al., 1992b, 1999), and the high level of chronic pain patients' physical complaints in general and of those with high negative affectivity in particular have been frequently ascribed to attentional and interpretational biases (Harkins et al., 1989; Watson and Pennebaker, 1989; Affleck et al., 1992; Larsen, 1992; Wade et al., 1992; Smith et al., 1995). However, as pain-related fears, attentional and interpretational processes can not sufficiently explain the response specificity of the self-reported physiological pain reactivity, suggesting that these processes may only indirectly affect long-term pain by their relationship to patterns of physiological reactivity.

In contrast to physiological reactivity, behavioral and cognitive reactivity failed to affect long-term pain. It could be argued that the lack of effects for cognitive and behavioral reactivity might be due to the limited assessment of these response systems with worry and avoidance behavior constructs. However, the selected constructs are theoretically grounded, and the present and similar assessments of avoidance behavior and worry have previously been demonstrated as predictive of various long-term outcomes in arthritis patients (Keefe et al., 1989; Evers et al., 1998a; van Lankveld et al., 1999, 2000; Steultjens et al., 2001). Preliminary evidence also supports specific modality-related effects, depending on the type of response system: behavioral responses might most directly affect activity-related outcomes; cognitive responses, subjective-cognitive outcomes; and physiological responses, sensory-related outcomes. For example, avoidance behavior has most consistently been shown to predict functional disability and use of medication in prospective and treatment studies (e.g. Linton, 1986; Evers et al., 1998a; van Lankveld et al., 1999; Steultjens et al., 2001). Cognitive constructs of worrying or catastrophizing, although related to various long-term outcomes, including pain (Keefe et al., 1989), have been shown to affect the affective and evaluative components of pain, but not the sensory component (Geisser et al., 1994). In contrast to the study by Keefe et al. (1989),

where a visual analog scale pain measure was used, our pain assessment consisted of both clinical and comprehensive self-report measurements that mainly reflected sensory aspects of pain (Fuchs et al., 1989; Evers et al., 1998b), suggesting – in agreement with previous cross-sectional research (McCracken et al., 1996) – that the sensory pain aspects are most directly affected by physiological reactivity patterns.

The results of our study, demonstrating effects for physiological pain reactivity on long-term RA pain, but not for the behavioral and cognitive reactivity, underscore the importance of assessing the response systems separately and studying their relative contribution to different pain outcomes. Increasing knowledge of the underlying mechanisms could eventually provide a better understanding of active treatment components and lead to more effective chronic pain modification procedures. For example, a response-specific pattern of physiological pain reactivity brings into question the sole application of anxiety-based treatments for reducing pain, since they might mainly affect the anxiety aspect of pain instead of the actual pain symptoms (Gross and Collins, 1981). A better understanding of the specificity of these mechanisms seems to be particularly useful for predicting long-term pain, since multidisciplinary treatments are currently primarily aimed at modifying secondary outcomes, such as depression and disability, instead of pain itself.

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