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Fear of movement/(re)injury and muscular reactivity in chronic low back pain patients: an experimental investigation

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Abstract

This experiment was set up to test the hypothesis that confrontation with feared movements would lead to symptom-specific muscular reactivity in chronic low back pain patients who report high fear of movement/(re)injury. Thirty-one chronic low back pain patients were asked to watch a neutral nature documentary, followed by a fear-eliciting video-presentation, while surface electromyography (EMG) recordings were made from the lower paraspinal and the tibialis anterior muscles. It was further hypothesized that negative affectivity (NA) would moderate the effects of fear on symptom-specific muscular reactivity, as well as the effects of muscular reactivity on pain report. The results were partly as predicted. Unexpectedly, paraspinal EMG-readings decreased during video-exposure but this decrement tended to be less in fearful patients than in the non-fearful patients. Negative affectivity did not moderate this effect, but moderated the effect of pain-related fear on muscular reactivity of lower leg muscles. In addition, NA directly predicted muscular reactivity in the right tibialis anterior muscle. As predicted, there was a significant covariation between left paralumbar muscular activity and pain report. This association was moderated by NA, but in the opposite direction. The findings extend the symptom-specificity model of psychophysiological reactivity, and support the idea that pain-related fear perpetuates pain and pain disability through muscular reactivity. © 1999 International Association for the Study of Pain. Published by Elsevier Science B.V.

Keywords: Low back pain; Muscular reactivity; Electromyography; Negative affectivity; Fear of movement/(re)injury

1. Introduction

In an attempt to explain how and why some individuals with musculoskeletal pain develop a chronic pain syndrome, several models have been developed including the ‘fear-avoidance model of exaggerated pain perception’ (Lethem et al., 1983), and more recently, a cognitive model of fear of movement/(re)injury (Vlaeyen et al., 1995a). The central concept of these models is fear of pain, or the more specific fear that physical activity will cause (re)injury. Patients may react to these fears either with ‘confrontation’ or ‘avoidance’. In the absence of a serious somatic pathology, confrontation with activities is conceptualized as an adaptive response, eventually leading to the reduction of fear and to recovery. In contrast, avoidance leads to the maintenance

or exacerbation of fear, possibly resulting in a phobic state. Further avoidance of both social and physical activities may finally lead to physical and psychological consequences augmenting disability. In 1990, Kori et al. introduced the term ‘kinesiophobia’ (kinesis = movement) for the condition in which a patient has ‘an excessive, irrational, and debilitating fear of physical movement and activity resulting from a feeling of vulnerability to painful injury or reinjury’.

When a fearful subject is exposed to the feared situation, a behavioral response (escape and avoidance), a cognitive response (worry), as well as a psychophysiological response (increased physiological arousal) can typically be observed (Lang, 1968). Does this triple response mode model also hold for pain-related fear? It has repeatedly been shown that pain-related fear is at least associated with escape/avoidance behaviors. That is, poor behavioral performance (Vlaeyen et al., 1995a; Crombez et al., 1998; 1999) and self-reported disability (McCracken et al., 1992; Waddell et al., 1993; Vlaeyen et al., 1995b; Asmundson et al., 1997; Crom-

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bez et al., 1999) are more strongly associated with pain-related fear than with pain severity or biomedical findings. There is also evidence that pain-related fear is associated with specific worries. McCracken and Gross (1993) and Vlaeyen et al. (1995a,b) found a strong association between pain-related fear and pain catastrophizing in chronic pain patients.

In a previous study (Vlaeyen et al., 1995a), it was tested whether pain-related fear in chronic low back pain patients not only would lead to behavioral and cognitive reactivity during physical activity but also to heightened psychophysiological responses. During exposure to a lifting task, heart rate (HR) and skin conductance levels (SCL) were continuously measured. In line with the prediction, fearful patients reported more concern about damage to their back and they terminated the lifting task significantly sooner than the non-fearful patients. However, no clear evidence was found for increased physiological reactivity in terms of HR and SCL changes in the fearful patients.

One reason for the absence of an effect on psychophysiological parameters in the previous study may be that physiological parameters included were not specific enough. According to the diathesis stress model, chronic pain may develop from, and be exacerbated by the interaction of a predisposing organic or psychological condition (diathesis), and threatening environmental events (stress) (Flor et al., 1990). In a series of well controlled laboratory studies, Flor and Turk (1989) and Flor et al. (1991) demonstrated that under personally relevant stressors, chronic low back pain patients show relatively large increases in electromyography (EMG) readings specifically in the lower left paraspinal muscles, when compared to healthy controls and patients with pain complaints other than back pain. In other words, psychophysiological reactivity to personal stressors appears to be symptom-specific. To further explore the relationship between pain-related fear and specific psychophysiological reactivity, the present experiment measured EMG of the lower paraspinal muscles in chronic low back pain patients during video-exposure of physical activity, while they were anticipating that they had to perform these activities after termination of the video session.

Extending on the original diathesis stress model, negative affectivity (NA) – a general tendency to experience subjective distress and dissatisfaction (Watson and Clark, 1984) – may be considered as an additional vulnerability factor. It is postulated that high NA subjects are hypervigilant, constantly scan their environment for signs of trouble, and interpret ambiguous stimuli in a negative and threatening manner (Watson and Pennebaker, 1989). Because of their selective and anxious attention to minor physical symptoms, the same stress stimuli are likely to have more impact in high NA patients as compared to low NA patients and thus, the preexisting tendency to react with muscular reactivity to stressors might be more pronounced in these patients. Applied to pain-related fear, this would mean that in fearful chronic low back pain patients muscular reactivity in the

presence of a specific fear-eliciting stimulus would be more pronounced in high NA patients.

Negative affectivity may also moderate the effects of muscular reactivity on pain report. There is now ample evidence that NA is associated with elevated symptom reporting (Vassend et al., 1995; McCrae and Lumley, 1998) and that high NA patients report more pain in response to the same stimuli than low NA patients (Morgan and Horstman, 1987). Germane to this issue, a recent study found that stress-induced increases in lower paraspinal muscular reactivity in low back pain patients predicted greater pain severity, and that this occurred more strongly in depressed patients (Burns et al., 1997).

The hypotheses of this study are the following: (a) high fearful patients report significantly higher levels of subjective tension to the video-exposure than the low fearful patients. (b) Fear of movement/(re)injury predicts a specific increase in lower left paraspinal muscle reactivity during the video confrontation. (c) Negative affectivity moderates the effect of fear of movement/(re)injury on muscular reactivity. (d) Increased paraspinal muscle reactivity covaries with increases in pain report during subsequent physical performance, and this effect is also moderated by NA.

2. Method

2.1. Subjects

Thirty-one chronic low back pain patients (17 from a waiting list of the Rehabilitation Center Hoensbroek and 14 from the Maastricht University Hospital) agreed to participate. All patients had minimal organic findings or displayed pain complaints that were disproportionate to the demonstrable organic basis of their pain. The sample consisted of 16 female and 15 male patients with a mean age of 41.61 years (range: 20–66, SD = 10.7). The mean duration of their pain complaints was 10.1 years (range: 0.7–35, SD = 8.9) and they had a mean score of 13.6 (range: 4–20, SD = 4.34) on the Dutch version of the Roland Disability Questionnaire (Roland and Morris, 1983) which suggests that these patients are moderately to highly disabled (Stratford et al., 1996; Gommans et al., 1997). Twenty-two patients also had radiating pain into one leg. All patients signed an informed consent form after being informed about the procedure, which was approved by the medical ethics committee. After completion of the experiment, all patients received a small remuneration for participating.

2.2. Measures

2.2.1. Muscular reactivity

For surface EMG recordings 1 cm² Ag–AgCl disposable electrodes were used in conjunction with SPA20/12 pre-amplifiers (K-Lab, Amsterdam) (CMRR > 110 dB; input impedance > 500 M Ω ; signal amplification 100; noise

referred to input $< 2 \mu V_{\text{rms}}$). Furthermore a K-Lab MF-118 amplifier with a 3rd order Butterworth high pass filter (cut-off frequency 20 Hz) was used. After removal of hair (if necessary) and cleaning the subject's skin with alcohol, the EMG electrode pairs were placed parallel to the muscle fiber direction of the different muscles. Position of the electrodes over the left and right erector spinae was 2.5 cm lateral to the spinous processus at level L3. The tibialis anterior muscle was chosen as non-specific control muscle. Electromyography electrodes for the left and right tibialis anterior muscle were placed on the bulkiest part of the muscle belly, approximately 5 cm caudoventral to the caput fibulae. The inter-electrode distance was 2.3 cm. A grounding electrode was attached to the anterior part of the left ankle. All analogue signals were A/D-converted using a sample frequency of 1000 Hz. Sample time was 180 s starting synchronously to the beginning of the video presentation. Electromyography data were analyzed off-line (MATLAB software, MA). After fullwave rectification of the signals mean EMG values were calculated for the last 20 s of baseline and last 20 s of video-exposure.

2.3. Questionnaires

Pain-related fear: The Dutch version of the Tampa Scale for Kinesiophobia (TSK; Kori et al., 1990; Vlaeyen et al., 1995a) was used for the measurement of trait fear of movement/(re)injury. The TSK is a 17 item questionnaire that measures the extent to which the individual fears that exercise may lead to (re)injury (e.g. 'I wouldn't have this much pain if there weren't something potentially dangerous going on in my body'). The Dutch version has been shown to have good reliability and validity (Vlaeyen et al., 1995a,b). *Negative affectivity:* the negative emotionality scale (NEM) is a 14-item 2-point scale derived from tellegen's multidimensional personality questionnaire (MPQ; Tellegen, 1982). The NEM is considered a trait measure of negative affectivity. High NEM-scorers describe themselves as nervous, apprehensive, irritable, overly sensitive, and emotionally labile. *Perceived tension:* Subjective tension level was measured with a visual analog scale (VAS) consisting of a 100 mm. horizontal line, anchored at the left and right by the words 'I am not at all tense' and 'I have never been so tense'. This measure was taken before and after the video-exposure. *Pain intensity:* a VAS consisting of a 100 mm. horizontal line was used anchored at the left and right by the words 'no pain' and 'worst imaginable pain'. This measure was taken three times: at the very start of the experiment, immediately before, and after the actual physical performance. *Socio-demographic and pain characteristics:* data on age, gender, working status, pain duration, use of medication, use of supportive equipment for ambulation and radiating leg pain were obtained during a semi-structured interview that was held after termination of the experiment.

2.4. Procedure

2.4.1. Preparation

All participants were called at home, and received a short introduction to the experiment. Written information was sent together with an informed consent form. After receiving written consent from the patients, two questionnaires (NEM, RDQ) were sent home with a clear instruction to complete them with no support from others, except from the research assistant who could be contacted by phone. When a patient arrived for the appointment, the procedures and EMG measures were explained, and the remaining questionnaires were completed. Each patient was seated upright in an armchair while EMG electrodes were attached. For the next 5 min the participant was required to sit quietly, and the signals for the EMG were checked. During this period eye blink responses to short loud noises were measured as part of another study. Immediately after this, the video was started simultaneously with EMG recordings.

2.4.2. Video-exposure

The video consisted of two parts, a baseline and an exposure part. The baseline part was introduced with a written and spoken message 'You will see two scenes, please take a comfortable position in which you can sit as quiet as possible'. After this introduction, a neutral, short fragment (66 s) taken from a nature documentary was shown. Thereafter, a second written and spoken instruction was given: 'You will now see two physical activities. After termination of this fragment, you will be asked to actually perform one of these activities yourself. Watch carefully!' Then, both activities were shown. The first activity consisted of a dummy patient riding vigorously on a stationary bicycle, while a trainer verbally encouraged the patient to perform even harder. The second activity consisted of a session in which isometric muscle strength was measured with the trunk-extension-flexion unit of the cybex system. Again, a trainer encouraged the patient to perform maximally. In both scenes, the dummy patient was visibly sweating and displayed overt pain behaviors such as sighing, grimacing, and groaning, suggesting that the activities caused a lot of pain and effort.

2.4.3. Actual performance

After the video, subjects completed three VASs: current level of subjective tension, current pain intensity and maximum pain during the video-exposure. Subsequently, the electrodes were removed, and the subject was brought to an adjacent room in which two activities in fixed order were to be performed: a lifting task and a bicycle riding task. For the lifting task, patients were asked to stand up and lift a 5.5 kg bag with the dominant arm and hold it until pain or physical discomfort urged them to place the bag back on the floor. A similar instruction was given for a stationary bicycle task. The patients did not receive any encouragement, and they were assured that they were free

Fig. 1. Mean values of experienced tension (VAS) before and after the video-exposure for the high and low fearful subgroups (based on TSK median split).

to decide when to stop the task (Results of the physical performance tasks are presented in Crombez et al., 1999).

2.4.4. Debriefing

The experiment ended with the completion of a number of questions regarding the video, their behavioral performance after the video-exposure, and the characteristics of the onset of their pain complaints. Patients received a small remuneration for their participation.

2.5. Statistical analyses

Subgroups of low and high fearful patients were formed based on the median-split of the TSK. The difference in perceived tension was tested using ANCOVA after the video-exposure between both subgroups with baseline tension as the covariate. Electromyography baseline differences between subgroups were tested with MANOVA. To examine the effects of the video-exposure on muscular reactivity for the high fearful and the low fearful patients, ANOVA's were carried out on the change in EMG readings between baseline and video-exposure (Cybex session) for each muscle separately. Subsequently, multiple regression analyses were applied with the change in EMG readings between baseline and video-exposure (Cybex session) as the dependent variable, and fear of movement(re)injury (TSK), gender, age, pain duration, radiation of pain into legs, and current pain intensity as the independent variables. A similar regression analysis was done to test the influence of muscular reactivity during video-exposure on pain report during actual physical performance, with maximum pain during the physical activity as the dependent variable. All regression analyses were carried out using a backward elimination method. Moderating effects of NA were examined according to Baron and Kenny (1986). To examine the moderating effect of NA on the effect of fear of movement(re)injury on muscular reactivity, the interaction

term TSK*NEM was included as well. For the analysis of the moderating effect of NA on the effect of muscular reactivity on pain report during physical performance, the interaction term $\Delta\text{EMG}*\text{NEM}$ was included. To avoid collinearity in the interaction terms, variables in the interaction terms were centered (mean subtracted from individual scores).

3. Results

3.1. Fear of movement/(re)injury and perceived tension

Based on the median-split of the TSK total score (range: 24–49; median = 40), two subgroups of high fearful and low fearful patients were identified. Fig. 1 shows the mean levels of subjective tension before and after the video-exposure for the fearful and non-fearful patients. Generally, fearful patients report higher tension levels as compared to the non-fearful. In addition, there was a significant difference in perceived tension during the video-exposure between fearful and non-fearful patients ($t = -2.599$, $P = 0.015$), even when baseline tension levels were controlled for (ANCOVA, $F = 6.352$, $P = 0.018$). Thus, video-exposure indeed increased subjective tension in chronic low back pain patients who reported substantial pain-related fear.

Table 1

Electromyography readings (in μV) at baseline and during video-exposure (Cybex session) across muscle groups for high and low fearful patients

	High fear (TSK ≥ 40)		Low fear (TSK < 40)	
	Mean	SD	Mean	SD
Er. Spinae left				
Baseline	238.7	224.9	191.9	144.0
Video-exposure	233.4	220.4	185.3	144.3
Video-exp. – baseline	–5.4	15.5	–6.6	11.2
Er. Spinae right				
Baseline	363.0	449.6	218.7	142.0
Video-exposure	365.0	442.7	218.0	149.7
Video-exp. – baseline	1.5	16.5	–0.9	13.4
Tibialis ant. left				
Baseline	568.2	503.8	356.6	408.1
Video-exposure	568.3	500.9	358.2	407.1
Video-exp. – baseline	0.1	7.6	1.6	5.1
Tibialis ant. right				
Baseline	172.9	252.5	192.7	386.5
Video-exposure	168.5	249.3	188.8	384.7
Video-exp. – baseline	–4.5	12.3	–3.9	22.4

Table 2

Results of multiple regression analyses for change in muscle reactivity during video-exposure (watching Cybex session) versus baseline (nature documentary), with gender, age, pain onset, pain duration, negative affectivity (NEM), pain intensity (VAS) and fear of movement/(re)injury (TSK) tested with backward elimination method. Results of the last step are displayed

Dependent variable	Independent variables	Adj. R ²	R ²	Beta	P
Er. spinae (left)	Pain duration	0.20	0.26	−0.44	0.018
	Fear of movement/(re)injury			0.31	0.089
Er. spinae (right)		0.00	0.00		
Tibialis ant. (left)		0.17	0.27		
	Fear of movement/(re)injury			0.07	0.735
	Negative affectivity			0.10	0.611
	Negative affectivity * fear of movement/(re)injury			−0.56	0.013
Tibialis ant. (right)		0.32	0.42		
	Radiation into legs			−0.32	0.065
	Negative affectivity			−0.48	0.007
	Fear of movement/(re)injury			0.13	0.428
	Negative affectivity * fear of movement/(re)injury			0.47	0.012

3.2. Muscular reactivity

Table 1 displays the baseline values of the EMG readings for the four muscle groups in the high fearful and low fearful patients. High fearful patients seemed to have higher baseline EMG readings than the low fearful patients for the paraspinal muscles, but MANOVA analysis did not indicate an overall fear effect across these muscle groups (Wilks Lambda = 0.95, $F(2, 25) = 0.717$, $P = 0.498$). Unexpectedly, after video exposure a minor decrease in muscular reactivity was observed for the erector spinae left and the tibialis anterior right. The ANOVA analysis showed that there were no significant differences in muscular reactivity between high-fearful and low-fearful patients for any of the muscles ($P > 0.561$). The results of the multiple regression are displayed in Table 2. The variable inflation factors (VIF) were low showing that here was no collinearity (range: 1.007–1.284). As hypothesized, fear of movement/(re)injury was only predictive of the reactivity of the left erector spinae, although significance was not reached. High fearful patients tended to show a smaller reduction in reactivity

than the low fearful patients. Pain duration was an additional predictor, patients with longer pain duration showing greater reduction in muscle reactivity than patients with more recent pain. For the right tibialis anterior muscle, NA was the most important predictor of muscle reactivity, with pain radiation as an additional and independent predictor. Patients with higher NA and patients with radiating pain into the legs show less reactivity than the patients without radiating pain.

3.3. Negative affectivity as a moderator

Following the procedure described by Baron and Kenny (1986) to examine the moderator effect of NA upon the influence of fear of movement/(re)injury on muscle reactivity, the interaction term NEM*TSK was included in the regression models, while controlling for main effects. As also displayed in Table 2, significant interactions were only found for the tibialis anterior bilaterally. For the patients scoring low on NA, fear of movement/(re)injury was not significantly associated with reactivity of the left

Table 3

Predictors of maximum pain experienced during behavioral performance. Results of multiple regression analysis for maximum pain during the lifting task (VAS), negative affectivity (NEM), paraspinal muscular reactivity (left) and the interaction term between the latter independents

Dependent variable	Independent variables	Adj. R ²	R ²	Beta	P
Maximum pain during lifting		0.19	0.28		
	Negative affectivity			0.21	0.231
	Change in paraspinal muscle reactivity (left)			0.43	0.043
	Negative affectivity * change in paraspinal muscle activity (left)			−0.56	0.010

Fig. 2. Role of fear of movement/(re)injury on muscle reactivity (Exposure minus baseline EMG readings in μV) in the high and low NA patients separately, for tibialis anterior left (L) and tibialis anterior right (R).

tibialis, while for the high scorers on NA, a significant but negative association is found ($\text{Beta} = -0.79$, $P = 0.015$). The more fear, the greater decrement in EMG readings. Also for the right tibialis anterior, fear of movement/(re)injury was associated with muscular reactivity for patients scoring high on NA only, but here the association was positive: the more fear, the greater the increment in EMG readings ($\text{Beta} = 0.71$, $P = 0.012$). Fig. 2 shows the different patterns of fear of movement/(re)injury and muscular reactivity for high and low NA patients separately.

3.4. Muscular reactivity and pain during physical performance

Is muscle reactivity during video-exposure associated with subsequent pain during the physical performance task, and is this effect moderated by negative affectivity? Table 3 shows the results of the regression analyses testing the predictive model for maximum pain experienced during the lifting task. Again, low VIFs suggest no collinearity (range: 1.01–1.35). As expected, reactivity of the left paraspinal muscle is shown to significantly predict experienced pain during the lifting task: the smaller the reduction in muscle tone, the more pain being reported. Although there is a significant interaction between NA and muscle reactivity, the moderator effect of NA is in the opposite direction: the covariation between muscle reactivity and pain only holds for patients scoring low on NA ($r = 0.64$, $P = 0.007$) and not for the patients scoring high on NA ($r = -0.06$, NS), (for the other muscles, the correlations were low and not significant: erector spinae right ($r = 0.21$), tibialis anterior left ($r = 0.07$) and tibialis anterior right ($r = 0.20$). For those muscles, no moderation effect of NA was found). Similar analyses failed to show any association between change in muscle reactivity and

maximum pain during the bicycling task, suggesting that the effects are of short duration.

4. Discussion

This experiment was set up to test the hypothesis that confrontation with vigorous movements in chronic low back pain patients who fear that certain movements might lead to (re)injury would lead to increased paraspinal muscular reactivity. The fear-eliciting stimulus consisted of a video presentation displaying a dummy patient rigorously performing physical activities. The patients were instructed to watch carefully as actual performance of the exercises by themselves was intended to be the next task. Electromyography of the lower paraspinal muscles and the tibialis anterior muscles (lower leg) was carried out during video-exposure while the patients were sitting quietly in an armchair. We hypothesized that fearful patients report higher subjective tension to the video-exposure and that they would show greater increase in symptom-specific paraspinal muscular reactivity during the video exposure. We also hypothesized that negative affectivity (NA) would moderate the effects of fear of movement/(re)injury on muscular reactivity. Finally, we examined the association between lower paraspinal muscle reactivity and subsequent pain report during physical performance, and hypothesized that this association is moderated by NA.

As predicted, the fearful patients showed an increase in subjective tension during the video-exposure, suggesting that our manipulation had worked. However, given the salience of this particular fear-eliciting stimulus, this increase was lower than we had expected. A possible reason is that the video-exposure might not have been credible enough. During the debriefing at the end of the experiment, many patients reported that quite soon they decided not to perform the activities as vigorously as the video suggested them to do. An alternative explanation might be that the fearful patients voluntarily avoided watching these threat-relevant stimuli, as is often found in patients with specific phobias (Tolin et al., 1999). It remains to be examined whether these forms of cognitive avoidance of the stressor have to be taken into account in similar studies in the future.

Although self-reported subjective tension increased from nature documentary to the activity exposure in the fearful patients, muscular reactivity generally decreased during the video-exposure. One explanation could be that initial reactivity to the experimental setting may have been already elevated (the measurement of eyeblink responses to short loud noises may have added to this reactivity), and in combination with the safe feeling that they could withdraw any time during the experiment, habituation might have taken place. In line with our hypothesis, the decrement in EMG appeared to be less in high fearful patients and this pattern was symptom-specific. The regression analyses

showed that only the reactivity of the left erector spinae was predicted by fear of movement/(re)injury. The fact that statistical significance for this association was not reached may be due to the limited strength of our manipulation. Another reason might be the selection of subjects. The power of the analyses might be increased by deliberately selecting two (or more) contrasting fear groups. Selection can be based on the upper and lower quartile of the TSK distribution, or the receiver operating characteristic method (ROC; Hanley and McNeil, 1982). The ROC method produces a graph of 'true positive' versus 'false positive' for several cut-off points and the optimal point can be determined. We felt that our sample size was too small for the use of either method, but in a replication with a larger population size the application of the ROC method may reveal the optimal TSK cut-off score for the representation of differences in muscle reactivity. A final reason why statistical significance was not reached may be the large intersubject variability of the EMG readings, which is a typical problem in EMG research. Although normalization has been suggested to overcome this problem (e.g. Knutson et al., 1994), Yang and Winter (1984), who tested various normalization procedures, found that these techniques generally did not reduce intersubject variability.

Of particular interest is the fact that also pain duration remained in the final predictive model for reactivity of the left paraspinal muscle. This suggests that muscular reactivity associated with pain-related fear occurs early on in the development of chronic pain. An alternative explanation is that patients with longer pain duration are likely to be more disabled, and therefore more easily might have decided to ignore the instructions during the video-exposure. For the reactivity of the right erector spinae, none of the independent variables included in the model were predictive. These findings are consistent with those of Flor et al., (1990, 1991) who also found that symptom-specific muscular reactivity was restricted to the left side. In general, our findings suggest that pain-related fear is an important factor in eliciting symptom-specific psychophysiological reactivity in chronic pain patients.

Extending the diathesis-stress model, we anticipated that NA moderates the effects of stress on muscular reactivity. In the presence of a specific fear-eliciting stimulus, muscular reactivity was expected to be more pronounced in patients vulnerable to stressful demands. The results of our study partly confirmed this hypothesis. In symptom-specific muscular reactivity, which was predicted by pain-related fear, there was no moderating effect of NA. However, in both tibialis anterior muscles, we found a moderating effect of NA. In addition, NA directly predicted muscular reactivity in the right tibialis anterior muscle. For these non symptom-specific muscles, pain-related fear predicted muscular reactivity only in the high NA patients. In other words, our results appeared to extend the validity of the model that personally relevant stressors elicit muscular reactivity in chronic pain patients by showing that in high NA patients,

pain-related fear also affects muscular reactivity in other than the left paraspinal muscles. We also found that pain-related fear produced opposite effects in EMG readings at both body sides. For the left tibialis muscle, pain-related fear decreased muscle reactivity, while in the right muscle an increase in EMG readings was found. A clear explanation for this asymmetry is hard to find. Although very speculative, the findings may be linked to the characteristics of our study sample. The majority of the patients reported radiating pain into the right leg, which therefore might be more vulnerable to muscular reactivity under stressful circumstances. However, further research is warranted to clarify this issue.

Consistent with the findings of Burns et al. (1997) we found that fear-induced increases in lower paraspinal muscle reactivity predicted greater pain during a subsequent physical performance test. For the other muscles, no such association was found. During the second physical performance, the influence of muscular reactivity on subsequent pain disappeared, suggesting that the effect has a relatively short duration. Contrary to the hypothesis, the moderating effect of NA was in the opposite direction. The association was only found in low NA patients. One possible explanation is that high NA patients are likely to report pain complaints, irrespective of their current health status (Watson and Pennebaker, 1989), and possibly also irrespective of muscular reactivity.

This study is the first to show that the symptom-specificity model of psychophysiological reactivity in chronic pain also applies to the domain of pain-related fear. Although of relatively short duration, reactivity of the left paraspinal muscles is also associated with subsequent pain during a physical activity. In addition, we were able to show that in patients who report high NA, pain-related fear also influences muscular reactivity in other muscles as well. However, the study merits replication with a stronger, and perhaps more credible fear-eliciting stimulus.

What are the clinical implications of this study? For individuals suffering from excessive fears and phobias, graded exposure to the feared stimulus has proven to be the most effective treatment (Davey, 1997). After identification of the essential stimuli, gradual exposure following a preset hierarchy of situations which elicit increasing fear is carried out. This approach appears to be applicable in current settings for the treatment of chronic pain, but it has not been implemented and studied systematically (Philips, 1997; Crombez et al., 1999; Vlaeyen and Linton, 1999). Such an exposure is similar to existing graded activity programs in that it gradually increases activity levels despite pain (e.g. Lindström et al., 1992), but is different in that it pays special attention to the personally-relevant and specific pain-related fear stimuli. The results of this study suggest that graded exposure may be enhanced with applied relaxation techniques (Öst, 1988). Such an approach would not only be empirically based, it would also provide the possibility for a more customized treatment approach for the subgroup of chronic

low back pain patients who report substantial pain-related fear.

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