

1 Pain Catastrophizing and Fear of Pain predict the Experience of Pain in Body Parts not targeted by a
2 Delayed-Onset Muscle Soreness procedure

3 Nils Georg Niederstrasser^{1,2,3}, Ann Meulders^{1,2}, Michel Meulders^{4,5}, P. Maxwell Slepian⁶, Johan W.S.
4 Vlaeyen^{1,2,7}, & Michael J.L. Sullivan³

5 ¹Research Group on Health Psychology, KU Leuven, Leuven, Belgium

6 ²Center for Excellence on Generalization Research in Health and Psychopathology, University of Leuven,
7 Leuven, Belgium

8 ³Department of Psychology, McGill University, Canada

9 ⁴ Department of Informatics, Simulation and Modeling, University of Leuven, Leuven, Belgium.

10 ⁵Research group on Quantitative Psychology and Individual Differences, University of Leuven, Leuven,
11 Belgium

12 ⁶Department of Psychology, University of Ohio, Athens, Ohio

13 ⁷Department of Clinical Psychological Science, Maastricht University, The Netherlands

14 Number of figures: 3

15 Number of tables: 3 (+1 supplementary table)

16 Key words: generalization; multisite pain; delayed-onset muscle soreness; pain catastrophizing; fear of
17 pain

18 Disclosures: The authors report no conflict of interest.

19 The contribution of Nils Georg Niederstrasser was supported by the Center of Excellence on
20 Generalization Research (GRIP*TT; University of Leuven grant PF/10/005). This research was supported
21 by funds from the Canadian Institute of Health Research (CIHR) and the Institut de recherche Robert-
22 Sauvé en Santé et en Sécurité du Travail (IRSST). Ann Meulders is a postdoctoral researcher of the
23 Research Foundation, Flanders, Belgium (FWO Vlaanderen) (grant ID: 12E3714N). This study was also
24 supported by the Odysseus Grant ‘‘The Psychology of Pain and Disability Research Program’’ funded by
25 the Research Foundation, Flanders, Belgium (FWO Vlaanderen) (grant ID: G090208N) to Johan W.S.
26 Vlaeyen and an EFIC-Grünenthal Research Grant (E-G-G ID: 169518451) to Ann Meulders.

27 These data were presented as a poster at the 15th World Congress of Pain, Buenos Aires, Argentina,
28 Poster number: PW300.

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30 Correspondence concerning this article should be addressed to Nils Georg Niederstrasser, M.Sc.
31 Department of Psychology, University of Leuven, Tiensestraat 102, box 3726, 3000 Leuven, Belgium. E-
32 mail: NilsGeorg.Niederstrasser@ppw.kuleuven.be, T: +32 (0)16 37 32 75, F: +32 (0)16 37 61 44.

1 Abstract: The present study examined whether pain catastrophizing and pain-related fear predict
2 the experience of pain in body regions that are not targeted by an experimental muscle injury protocol. A
3 delayed-onset muscle soreness (DOMS)-protocol was used to induce pain unilaterally in the pectoralis,
4 serratus, trapezius, latissimus dorsi, and deltoid muscles. The day following the DOMS-protocol,
5 participants were asked to rate their pain as they lifted weighted canisters with their targeted arm (i.e.
6 injured) and their not-targeted arm. The lifting task is a non-noxious stimulus unless participants are
7 already experiencing musculoskeletal pain. Therefore, reports of pain on the not-targeted arm were
8 operationalized as pain in response to a non-noxious stimulus. Eighty-two (54 females, 28 males) healthy
9 university students completed pain catastrophizing and fear of pain questionnaires and went through the
10 DOMS-protocol. The analyses revealed that catastrophizing and pain-related fear prospectively predicted
11 pain experience in response to a non-noxious stimulus. The possible mechanisms underlying this effect
12 and clinical implications are discussed.

13

14 Perspective: Pain catastrophizing, and fear of pain prospectively predict the pain experience in response
15 to a non-noxious stimulus. The pattern of findings is consistent with the predictions of current models of
16 generalization of pain-related fear.

17

1 **1. Introduction**

2 Multisite pain (MSP) is more common than single-site pain ^{7, 26, 42}, affecting 5-10% of the
3 population ¹¹. Compared to single-site pain, MSP is associated with higher pain intensity, functional
4 disability, and duration of pain complaints ^{9, 46}. The debilitating and treatment-resistant nature of MSP has
5 led to increasing calls for the identification of risk factors ^{2, 25}.

6 Numerous investigations suggest that pain catastrophizing ^{18, 22, 28} and pain-related fear ^{16, 38, 39}
7 might be implicated in the development of MSP following musculoskeletal injury. Pain catastrophizing
8 refers to a negative cognitive-affective response to actual or anticipated pain ^{12, 44, 50}. Fear of pain refers to
9 a distressing emotional affective experience aroused by impending pain ⁶³.

10 Bortsov and colleagues ³ reported that MSP following a motor vehicle accident was more strongly
11 related to pain catastrophizing than to crash characteristics or associated injury. Similarly, Sullivan and
12 colleagues ⁴⁹ found correlations between pain-related fear and the number of pain-sites following rear-
13 collision motor vehicle accidents. Although findings from these clinical studies suggest that pain-related
14 psychological variables augment the risk of developing MSP, their correlational nature precludes strong
15 statements regarding causality.

16 In a previous study, we examined the ‘antecedent’ status of pain catastrophizing and pain-related
17 fear for the ‘spreading’ of pain following experimental muscle injury ⁴¹. A delayed-onset muscle soreness
18 (DOMS)-protocol was used to induce pain in the muscles of the upper arms and shoulders. We assessed
19 pain catastrophizing and pain-related fear prior to the DOMS-induction, and pain distribution 24 hours
20 later. The results showed that pain catastrophizing and pain-related fear independently predicted the
21 number of pain sites reported following DOMS, including pain in regions distal from the muscles targeted
22 by the DOMS-protocol.

23 Assuming that the prospective relations between pain catastrophizing, pain-related fear and MSP
24 are replicable; questions arise concerning the pathways by which these psychological variables impact on
25 the spreading of pain. There are at least two possibilities. Catastrophizing or fear might be associated with
26 physical injury-characteristics, contributing to the spreading of pain ^{6, 20, 40}. Alternatively, generalization

1 of pain-related fear may increase pain in injury-free body sites, through activation of brain areas
2 responsible for pain hyperalgesia^{30, 31, 45}.

3 Our first objective was to replicate previous findings⁴¹ showing a prospective relationship
4 between pain-related psychological variables and MSP. The second objective was to bring greater
5 precision to the specification of processes underlying psychological influences on MSP. An important
6 innovative design feature of the current study is increased anatomical precision of the DOMS-induction
7 procedure. We used a DOMS-protocol that targeted muscles of the upper arm and shoulders unilaterally,
8 allowing us to assess the pain experienced in anatomically distinct regions that remained injury-free
9 following DOMS. The following day, participants were asked to lift a 3.6kg weight with their
10 experimentally ‘injured’ arm, and their ‘non-injured’ arm. An important characteristic of the protocol is
11 that, in the absence of muscle injury on the not-targeted arm, lifting a 3.6kg weight is not typically
12 experienced as a noxious stimulus. Of interest was whether psychological variables would be associated
13 with the experience of pain in muscle regions contralateral to the side of the experimentally ‘injured’
14 muscles. In the complete absence of tissue damage, there would be little basis to argue injury-related
15 responses would be responsible for the experience of pain in muscle regions contralateral to those
16 targeted by the DOMS-protocol. However, models of generalization of pain-related fear would predict
17 that, through activation of brain networks associated with hyperalgesia, pain experience could spread
18 from an injury site to an adjacent but injury-free area, even if the stimulus is not one that would be
19 expected to generate a pain response^{23, 24, 33}.

20 **2. Methods**

21 **Participants**

22 We used a convenience sample of 82 (54 women, 28 men) healthy participants with a mean age
23 of 23.2 years ($SD = 5.15$; range: 18 - 44 years). Participants were recruited through advertisements placed
24 in the classifieds section of the McGill University website. A standardized telephone interview was used
25 to screen participants for the exclusion criteria. Individuals were not considered for participation if they
26 (1) had a medical condition that could be aggravated by participation in this study, (2) suffered from a

1 chronic pain condition, (3) were currently experiencing joint or muscle problems, (4) had engaged in
2 resistance training of upper body or trunk muscles more than once per week in the 6 months prior to
3 participation, or (5) had consumed pain relief medication in the five days prior to the testing session. The
4 Physical Activity Readiness Questionnaire (PAR-Q) was used as a screening measure for potential
5 contraindications to participation in the DOMS-induction procedure. The PAR-Q screens for the presence
6 of factors that are linked to increased health risk when engaging in strenuous activity (e.g. shortness of
7 breath, muscle or joint problems, fainting, circulatory problems). Participants endorsing any item on the
8 PAR-Q were excluded from participation in the study ⁵⁶.

9 **Procedure**

10 Ethical approval was obtained from the Institutional Review Board at McGill University.
11 Participants were invited to the laboratory for two testing sessions scheduled 24 hours apart. Upon arrival
12 to the laboratory, each participant provided informed consent. Participants were told that the study was
13 aimed at investigating psychological and physical factors associated with pain after repeated physical
14 activity. Anthropometric measures were obtained and participants were asked to complete the Pain
15 Catastrophizing Scale (PCS) ⁵², and the Fear of Pain Questionnaire-III (FPQ) ³⁷. See Figure 1 for an
16 overview of activities during sessions 1 and 2.

17 *Standardized lifting task.* For the lifting task participants stood in front of a stool to lift an
18 unmarked 4-litre size paint canister partially filled with sand, weighing 3.6 kg. The height of the stool on
19 which the canister was placed was adjusted such that the top of the canister was 5 cm below participants'
20 standing wrist height. Participants were instructed to lift the canister three times per arm in a pre-
21 determined sequence. Participants lifted the canister in a forward lateral movement until they reached an
22 extension of 135 degrees of their arm and replaced the canister on the stool. Immediately after the lift,
23 participants verbally rated how much pain they experienced during the lift on a numerical rating scale
24 (NRS) from 0 to 10, with 0 'no pain' and 10 'excruciating pain'. For reference purposes the NRS was
25 placed on the wall facing the participants. Whether participants began the lifting sequence with their
26 dominant or non-dominant arm was randomized. Hand dominance was determined by verbal report. The

1 experimenter modeled the lift of the canister in order to minimize inter-individual variations in the
2 approach to the lifting task. The experimenter modeled the canister lift without actually lifting the canister
3 itself. A standardized power-point presentation guided participants through the procedure to ensure
4 standardized intervals. Pauses between lifts were set at 8 seconds.

5 *DOMS-protocol.* The procedure used to induce DOMS consisted of four different strength
6 exercises (i.e. chest fly, seated cable row, shoulder flexion, and shoulder abduction) involving repeated
7 eccentric muscle actions. The DOMS-protocol was modeled after procedures described by Udermann and
8 colleagues⁶⁰ and Sullivan and colleagues⁴⁸, and was adapted for execution with one arm. The exercise
9 protocol was performed using the K1 Strength Training System (Body Craft, Sunbury, OH, USA). All
10 exercises were performed in sets of five repetitions. To ensure appropriate resistance, participants
11 completed each eccentric contraction in time to a countdown, set to 10 seconds. Consistent with Sullivan
12 and colleagues⁴⁸, participants were asked to complete the first set of repetitions without any additional
13 weight to become familiarized with the testing apparatus. Then, after the completion of each set,
14 additional weight was added. The weight was increased in steps of ten pounds until participants reached
15 the point of volitional fatigue or completed ten sets⁶¹. Volitional fatigue was defined as the point at which
16 the participant could no longer control the descent of the weight²¹. For each participant the relative
17 intensity of the final set of repetitions was 80% of the estimated repetition max, which is defined as the
18 amount of weight a person could only lift one time⁴⁸.

19 Participants were asked to perform the eccentric contractions with maximal effort and were given
20 verbal encouragement during the contraction (e.g. ‘Good job’ or ‘Keep going’). A one-minute recovery
21 period was provided between each set. Breaks of two minutes between exercises were implemented to
22 avoid muscle fatigue. To ensure performance of resisted eccentric contractions only, the experimenter
23 moved the load for the participants on the return from full flexion. The emphasis on the eccentric portion
24 of the strength exercise is known to induce DOMS⁸. During an eccentric contraction (lengthening
25 contraction), the muscle elongates while under tension due to an opposing force, which causes

1 microtrauma to the muscle fibers. Peak exercise-induced DOMS has been noted to occur 24-48 hours
2 after DOMS-induction ⁵.

3 To induce DOMS in the pectoralis major and serratus anterior muscles a chest fly was used. This
4 exercise involves lying face-up on a horizontal bench, with buttocks on the bench, back flat on the bench,
5 and feet flat on the ground. Participants grasped a cable attachment at shoulder width while keeping their
6 elbow in a slight bend and lowered their arm out the side in a wide arc until their upper arm was parallel
7 to the ground. The seated cable row works the middle trapezius and latissimus dorsi muscles. Participants
8 sat facing the machine, gripping a pulley with their elbow at 90 degrees. While puffing out the chest
9 participants released the pulley forward until their arms were fully extended. To target the anterior deltoid
10 muscles participants performed a shoulder flexion. Participants stood with a straight back, legs slightly
11 apart holding a cable attachment in their hand. Starting with the arm raised slightly above horizontal out
12 to their side, participants lowered the cable attachment until it rested against their thighs. Lastly, to target
13 the upper trapezius and middle deltoid muscles, participants performed a shoulder abduction. Participants
14 were instructed to stand with their feet slightly apart, holding a cable attachment raised to eye level. The
15 cable attachment was lowered until it rested against the front of participants' thighs. Mean number of
16 performed sets for the chest fly was 5.90 (1.14), the seated cable row was 6.67 (1.3), the shoulder
17 abduction was 4.05 (0.74), and the shoulder flexion was 3.8 (0.79). At the conclusion of the protocol,
18 participants were asked to abstain from physical exercise and use of pain or anti-inflammatory medication
19 prior to the next session, unless experiencing significant discomfort. None of the participants reported use
20 of pain or anti-inflammatory medication.

21 *Second testing session.* The second testing session occurred 24 hours (± 3 hours) after the first
22 testing session, as done in previous studies ⁵⁸. During this session, the height of the stool was adjusted as
23 in session 1, and participants were asked to repeat the lifting task. Finally, participants were debriefed.

24 *Self-report measures.* All questionnaires were completed at the end of the first testing session.
25 The PCS was used to measure catastrophic pain-related cognitions. Participants indicated the frequency
26 with which they experienced each of 13 different thoughts and feelings when in pain. Ratings were made

1 on a five-point scale with the endpoints ‘0’ (not at all) and ‘4’ (all the time). The PCS comprises three
2 inter-related dimensions: magnification, rumination, and helplessness^{4, 48, 50, 57}. Magnification refers to an
3 exaggeration of the threat value of the pain stimulus (e.g. ‘I become afraid that the pain will get worse’),
4 rumination describes the inability to shift attention away from pain-related thoughts (e.g. ‘I anxiously
5 want the pain to go away’), and helplessness refers individuals’ negative evaluation of their ability to
6 cope effectively with painful stimuli (e.g. ‘I feel I can’t go on’)⁵³. Total scores range from 0 to 52, with
7 higher scores indicating higher levels of pain catastrophizing. The total score reliably covers all facets of
8 catastrophizing in the context of pain⁵³. Research has supported the reliability and validity of the PCS²⁷,
9⁵². The Fear of Pain Questionnaire-III was used to assess pain-related fears. The FPQ is a 30-item self-
10 report instrument describing different painful situations. Respondents are asked to rate how fearful they
11 are of experiencing the pain associated with each situation described in the item content (e.g. ‘Having one
12 of your teeth drilled’). Fear ratings are made on a 5-point scale with the endpoints ‘1’ (not at all) and ‘5’
13 (extreme). Total scores (range 30–150) were calculated, whereby higher scores represent more fear. The
14 FPQ comprises three subscales, minor pain (e.g. ‘Getting a paper-cut on your finger’), severe pain (e.g.
15 ‘Breaking your leg’), and medical pain (e.g. ‘Receiving an injection in your hip/buttocks’). Research has
16 supported the reliability and validity of the FPQ³⁷.

17 **Data analysis overview**

18 Descriptive statistics were computed on sample characteristics and questionnaire scores. T-tests
19 for independent samples were used to examine sex differences on demographic and dependent measures.
20 We predicted that scores on the measures of pain catastrophizing and pain-related fear would
21 prospectively predict the experience of pain on the not-targeted arm at session 2. Multilevel modeling was
22 used to test whether there was a linear change in pain ratings on the not-targeted arm for each session,
23 depending on individuals’ levels of pain catastrophizing and pain-related fear, after controlling for pain
24 on the targeted arm. To test these research questions, we defined two multilevel regression models. The
25 first model tests the effects of pain catastrophizing, and the second model examines the influence of pain-
26 related fear. A detailed description of both multilevel regression models can be found in the online

1 supplementary material. The effects included in each model were estimated simultaneously using the SAS
2 procedure MIXED⁶². Explained variance was computed as the squared correlation between observed and
3 predicted values for the dependent variable. Predicted values for the outcome measure are based on the
4 estimated regression model and on the estimated values for the random effects. For the estimation of
5 variance parameters we used the restricted maximum likelihood (REML) method of the SAS procedure
6 MIXED³⁴. Follow-up contrasts were calculated to test our a priori hypotheses.

7 **3. Results**

8 *3.1 Sample characteristics.* Table 1 presents the means and standard deviations for participants'
9 demographics and pain-related psychosocial measures. There were no significant sex differences for age,
10 ($t(78) = -1.2, p = .23$), and scores on measures of pain catastrophizing, ($t(80) = 1.02, p = .31$), while
11 women reported significantly higher scores on indices of pain-related fear than men, ($t(76) = 2.8, p <$
12 $.01$). Furthermore, scores on an index of pain catastrophizing were significantly correlated with scores on
13 pain-related fear. Scores on the PCS and the FPQ were comparable to those that have been reported in
14 previous studies using pain-free non-clinical samples^{37, 51, 54, 55}. Supplementary Table 1 presents an
15 overview of the number of participants scoring within a certain range of pain catastrophizing and pain-
16 related fear. It is apparent from this table that 4 participants scored more than 2 SD above the mean on the
17 PCS, while 0 participants scored less than 2 SD below the mean. For the FPQ, 1 participant scored more
18 than 2 SD above the mean and 4 participants scored less than 2 SD below the mean.

19 *3.2. Manipulation check: DOMS-induction.* Tests of simple effects revealed that while DOMS
20 was effective in increasing mean reported pain on the targeted arm (pre: $M = 2.25, SD = 2.13$; post: $M =$
21 $3.73, SD = 2.34, t(81) = -8.50, p < .01$), the DOMS-induction did not influence mean reported pain for the
22 not-targeted arm (pre: $M = 2.44, SD = 2.19$; post: $M = 2.38, SD = 2.03, t(81) = 0.37, p = .72$). The
23 majority of participants would be considered to be experiencing mild to moderate pain at session 2 on
24 their targeted arm. Participants' reported pain intensity ratings at session 2 were comparable to those that
25 have been reported in previous research using DOMS protocols in non-clinical samples^{14, 15, 41}.

1 Furthermore, following the DOMS-induction participants reported, on average, more pain on their
2 targeted arm than on their not-targeted arm (not-targeted: $M = 2.38$, $SD = 2.03$; targeted: $M = 3.73$, $SD =$
3 2.34 , $t(81) = -9.73$, $p < .01$)

4 *3.3.1 The role of pain catastrophizing in the prediction of pain on the not-targeted arm. A*

5 multilevel regression analysis was conducted to examine the contribution of pain catastrophizing to the
6 prediction of reported pain on the not-targeted arm at session 1 and 2 (Model A). This model assumes that
7 pain ratings for the not-targeted arm change linearly over subsequent lifts (1,2,3) within sessions 1 and 2
8 and that the linear change depends on the participant's pain catastrophizing level. Furthermore, pain
9 reported on the targeted arm in the corresponding session was entered as a covariate to the model and
10 made a significant contribution to the prediction of pain ratings on the not-targeted arm in both sessions.
11 The estimated coefficient for the covariate indicated that participants who experienced higher levels of
12 pain on the targeted arm also reported more intense pain on the not-targeted arm both in session 1
13 ($\beta_A^{(1)} = .79$, $p < .001$) and session 2 ($\beta_A^{(2)} = 0.99$, $p < .001$).

14 As shown in Table 2, there was no interaction between the linear trend of the change in pain
15 ratings at session 1 over subsequent lifts and levels of pain catastrophizing. There was, however, a main
16 effect of catastrophizing, which indicated that higher levels of pain catastrophizing predicted greater
17 levels of pain on the first lift at session 1 ($\beta_{PCS}^{(1)} = 0.46$, $p < .01$) – when actually the lifting task was not
18 expected to be painful. Given that pain was reported on a scale of 0 to 10, with a SD of 2.16, the results
19 can be interpreted as follows: a change of 1 SD in PCS score was associated with an increase of 0.21 SD
20 in reported pain on the first lift at session 1. Follow-up analyses revealed that high pain catastrophizers
21 (i.e., 2 SD above average) reported greater pain than low pain catastrophizers (i.e., 2 SD below average)
22 on the first lift ($M = 1.8$, $SD = 0.68$, $t(322) = 2.66$, $p < .01$), second lift ($M = 1.78$, $SD = 0.66$, $t(322) = 2.69$,
23 $p < .01$) and third lift ($M = 1.75$, $SD = 0.68$, $t(322) = 2.57$, $p < .05$) of the first session.

24 At session 2, the linear change in reported pain intensity changed as a function of participants'
25 pain catastrophizing scores (see Figure 2). Further analyses revealed that the slope of the linear trend of

1 the pain-ratings for the not-targeted arm at session 2 increased significantly for individuals with high
2 levels of pain catastrophizing (mean PCS +2SD, $\beta_T + 2\beta_{TxPCS} = 0.53, p < .001$; increase of 0.25 SD per
3 trial), while for participants with relatively low levels of pain catastrophizing the reported pain on the not-
4 targeted arm at session 2 did not significantly increase across trials (mean PCS - 2SD, $\beta_T - 2\beta_{TxPCS} = -$
5 $0.17, p = .07$; decrease of 0.08 SD per trial). Follow-up analyses revealed that there were no differences
6 between high and low catastrophizers (i.e., 2 SD above average versus 2 SD below average) on the first
7 ($M = 0.31, SD = 0.61, t(322) = 0.51, p = .61$) and second lift ($M = 1.00, SD = 0.59, t(322) = 1.7, p = .09$) at
8 session 2, while high catastrophizers reported significantly greater pain during lift three at session 2 ($M =$
9 $1.7, SD = 0.61, t(322) = 2.78, p < .01$).

10 Comparison of the slopes for the linear change in reported pain intensity between session 1 and
11 session 2 for the not-targeted arm revealed that high catastrophizers showed a significantly steeper slope
12 at session 2, while low catastrophizers changed from a positive slope at session 1 to a zero slope at
13 session 2. The findings imply that high pain catastrophizers' pain increased over repeated lifts with their
14 not-targeted arm at session 2, while low catastrophizers reported similar levels of pain for all lifts.

15 *3.4.2 The role of pain-related fear in the prediction of pain on the not-targeted arm.* Model B
16 followed the same assumptions as model A and examined the contribution of pain-related fear to the
17 prediction of reported pain on the not-targeted arm at session 1 and 2. In Model B, pain reported on the
18 targeted arm in the corresponding session was also entered as a covariate and made a significant
19 contribution to the prediction of pain ratings on the not-targeted arm in both sessions. The estimated
20 coefficient of the covariate in session 1 ($\beta_A^{(1)} = .77, p < .001$) and session 2 ($\beta_A^{(2)} = .98, p < .001$)
21 indicated that participants who experienced higher levels of pain on the targeted arm also tended to report
22 more intense pain on the not-targeted arm.

23 As shown in Table 3, there was no interaction between the linear trend of the change in pain
24 ratings at session 1 over subsequent lifts and levels of pain-related fear. In other words, the slopes of the
25 linear trends for varying levels of pain-related fear were parallel. There was, however, a main effect of

1 pain-related fear, which indicated that higher levels of pain-related fear predicted greater levels of pain on
2 the first lift at session 1 ($\beta_{FOP}^{(1)} = 0.74, p < .0001$; a change of 1 SD in pain-related fear score was
3 associated with an increase of 0.34 SD in reported pain on the first lift at session 1). Follow-up analyses
4 revealed that individuals with high pain-related fear (i.e., 2 SD above average) reported greater pain on
5 the first lift ($M = 2.96, SD = 0.69, t(306) = 4.3, p < .0001$), second lift ($M = 2.82, SD = 0.68, t(306) = 4.17,$
6 $p < .0001$), and third lift ($M = 2.68, SD = 0.7, t(306) = 3.81, p < .0005$) than individuals with low pain-
7 related fear (i.e., 2 SD below average).

8 For session 2, the multilevel regression analysis revealed a significant interaction effect between
9 the linear trend of pain ratings during the lifting task and fear of pain scores (see Figure 3). Similar to the
10 findings of Model A, the interaction effect indicated that the slope of the linear trend for the pain ratings
11 on the not-targeted arm at session 2 varied as a function of levels of pain-related fear. Further analyses
12 revealed that the slope of the linear trend of pain ratings for the not-targeted arm at session 2 increased
13 significantly for individuals with high levels of pain-related fear (mean pain-related fear +2 SD, $\beta_T +$
14 $2\beta_{TxFPQ} = 0.47, p < .001$; increase of 0.22 SD per trial), while the reported pain on the not-targeted arm at
15 session 2 did not change significantly across trials for participants with relatively low levels of pain-
16 related fear (mean pain-related fear -2 SD, $\beta_T - 2\beta_{TxFPQ} = -0.06, p = .48$; decrease of 0.03 SD per trial).
17 Follow-up analyses revealed that there was no difference between subjects with high and low pain-related
18 fear on the first lift at session 2 ($M = 1.01, SD = 0.66, t(306) = 1.55, p = .12$), while subjects with high
19 pain-related fear reported significantly greater pain during lifts two ($M = 1.55, SD = 0.64, t(306) = 2.41, p$
20 $< .05$) and three ($M = 2.08, SD = 0.67, t(306) = 3.12, p < .01$) at session 2 than subjects with low pain-
21 related fear.

22 Comparison of the slopes for the linear change in reported pain intensity between session 1 and
23 session 2 revealed that subject with high pain-related fear showed a significantly steeper slope at session
24 2, while subjects with low pain-related fear change from a positive slope at session 1 to a zero slope at
25 session 2.

1 Note that both model A and B performed rather well in predicting participants' pain ratings for
2 the not-targeted arm after DOMS induction, as they explained respectively 96.4% and 96.3% of the
3 variance in this variable. Furthermore, including random intercepts in the model to account for different
4 average pain ratings across subjects for the not-targeted arm at the first trials of sessions 1 and 2 was
5 clearly important, given that for each model, a considerable part of the variability on this variable was due
6 to differences between subjects. Indeed, for model A, 89% and 87% of the variability in the pain ratings
7 at the first trial of session 1 and session 2 respectively, were due to differences between subjects.
8 Likewise, 88% and 86% of the variability in the pain ratings at the first trial of session 1 and session 2
9 respectively, were due to individual differences for model B.

10 **4. Discussion**

11 The aim of the current study was to elucidate whether pain catastrophizing and pain-related fear
12 predict the verbal report of pain in body parts contralateral to those targeted by the DOMS-protocol. The
13 findings of the present study join a growing body of literature supporting the view that pain
14 catastrophizing and pain-related fear increase the risk of experiencing adverse pain outcomes. To date,
15 experimental research has focused on bringing greater precision to the specification of processes
16 underlying psychological influences on the experience of pain within the experimentally injured body
17 parts^{41, 48, 59}. The results of the present study extend previous findings in showing that pain
18 catastrophizing and pain-related fear, measured in a pain-free state, also augment the experience of pain
19 in response to non-noxious stimuli, as the canister lifting task is not painful unless the individual is
20 already experiencing musculoskeletal pain.

21 As expected, the DOMS-protocol caused an increase in pain on the targeted arm. The muscles on
22 the not-targeted arm engaged during the canister lifting were unaffected by the DOMS-protocol and the
23 strain of the lifting task at session 2 was identical to the previous session. Despite this, reported pain in
24 response to the canister lift with the not-targeted arm in session 2 increased across lifts, at a significantly
25 greater rate than session 1. Due to the within subject nature of the experiment, there is little basis for
26 suggesting that processes other than the pain increase on the targeted arm were responsible for the

1 increasing pain reports during the lifting task on the not-targeted arm at session 2. Furthermore, the extent
2 to which pain on the not-targeted arm changed over time was influenced by pain catastrophizing and pain-
3 related fear. These interactions were present only at session 2, whereby high levels of pain catastrophizing
4 and pain-related fear predicted increasing pain over successive lifts, and low levels predicted flat or
5 decreasing trends. Interestingly, high and low fear and pain catastrophizing individuals reported similar
6 pain intensities during the first lift on their not-targeted arm at session 2; however, while pain dissipated
7 over time among low fear and low pain catastrophizers, leaving them with pain only in targeted body
8 sites, those scoring high continued to report increasing mild to moderate levels of pain in not-targeted, as
9 well as targeted areas.

10 Recently, researchers have speculated about the peripheral and central mechanisms that could
11 lead to the experience of pain in the absence of noxious stimulation. Recent research has suggested the
12 ‘generalization’ of pain-related fear as a mechanism by which fear might contribute to the experience of
13 pain in body parts distal to the site of injury^{16, 38, 39, 41}. Generalization of pain-related fear occurs when the
14 expectation of a painful sensation and pain-related fear is associated with a stimulus that resembles (lifts
15 on not-targeted arm), but is not identical to, the original pain-provoking stimulus (lifts on targeted arm)¹⁶.
16 Research has shown that the fear-induced expectation of a painful sensation contributes to an increased
17 sensitivity to pain¹⁰, through the activation of brain areas responsible for pain hyperalgesia^{30, 31, 45}.
18 Through generalization of pain-related fear, the expectation of pain may have generalized to lifts with the
19 not-targeted arm, activated corresponding brain areas, and thereby led to reports of increasing pain.
20 Lastly, highly fearful individuals generally have more negative expectations towards pain, such as
21 predicting higher levels of pain^{13, 36, 59}, suggesting greater activation of brain areas associated with
22 hyperalgesia.

23 Two issues deserve further inquiry. First, the assessment of pain-related fear generalization,
24 which is not measured in the current study. To prove the involvement of the generalization of pain-related
25 fear in the experience of increasing pain in not-targeted body sites, changes in pain on the not-targeted
26 arm need to be mediated by pain-related fear. The second issue that deserves further inquiry is the

1 possible effect of pain expectancy. It is possible that high catastrophizers and high pain-related fear
2 individuals did not correct their pain expectancies based on their preceding pain experiences, leading to
3 increasing pain over the course of the lifts. Future studies should include measures of pain expectancy to
4 further explore this issue.

5 Previous studies have suggested that pain-related fear might lead to muscle activation alterations
6 aimed at protecting injured muscle tissue, which in turn lead to increased pain in surrounding muscle
7 tissue^{20, 35, 40}. In the absence of injury, there is little basis to suggest the presence of muscle activation
8 alterations, which can therefore be ruled out as a mechanism by which pain may increase in not-targeted
9 body parts.

10 The results of the current study suggest that pain catastrophizing predicts the experience of pain
11 in response to a non-noxious stimulus in body sites not-targeted by experimentally induced injury. Pain
12 catastrophizing has been hypothesized to affect the experience of pain in injury-free sites through its
13 relationship with altered endogenous modulation of pain^{18, 22, 28}. Pain catastrophizing has also been
14 associated with indices of central sensitization^{18, 43}, whereby neuroplastic changes in the dorsal horn,
15 enacted by repeated nociceptive stimulation, lead to an expansion of the receptive fields of nociceptors³².
16 Expanded receptor fields may lead to the spreading of secondary hyperalgesia beyond the site of injury,
17 thereby spreading nociceptive input into injury-free body parts⁴⁷. Furthermore, high levels of pain
18 catastrophizing are potentially associated with lower values on an index of descending inhibition²².
19 Normally, in the presence of ongoing noxious stimulation, spinal cord responses to additional noxious
20 stimuli are down-regulated in the brain, a modulatory response termed conditioned pain modulation^{1, 65}. It
21 should therefore have been expected that, with pain in one arm, stimulation of the canister lift on the not-
22 targeted arm would be inhibited; however, due to potentially lower values on an index of descending
23 inhibition, high catastrophizers reported increasing pain intensities over the canister lifts with their not-
24 targeted arm.

25 Recent studies have suggested that the association between pain catastrophizing and an increased
26 release of pro-inflammatory cytokines¹⁷ might contribute to the spreading of pain beyond the site of

1 injury^{6,17}. While DOMS is a valid model for musculoskeletal injury, there have been reports suggesting
2 that pro-inflammatory cytokines are not elevated following DOMS²⁹. The absence of an increased release
3 of pro-inflammatory cytokines following DOMS-induction therefore precludes effects of pain-
4 catastrophizing through inflammatory mediators.

5 Caution must be used when interpreting the study findings. To maximize homogeneity of the
6 study sample, several exclusion criteria were used, limiting its generalizability. Furthermore, while
7 exercise-induced DOMS is a useful technique to mimic musculoskeletal pain conditions, it lacks the
8 affective and traumatic components of musculoskeletal injuries. In addition, healthy undergraduates differ
9 from individuals suffering from chronic pain on a number of demographic and health status variables. As
10 such, the current sample includes few participants whose questionnaire scores divert more than 2 SD from
11 the mean. Nevertheless, the fact that in very few participants robust and significant effects are found, can
12 also be seen as a strength. It is reasonable to assume that these effects would only be stronger when
13 individuals with more extreme questionnaire scores would have been included. Future research should
14 focus on systematically selecting ‘extreme’ samples.

15 Lastly, the results of the current study might not be generalizable to MSP conditions that arise in
16 the absence of injury such as arthritis or chronic widespread pain. Alongside the presence of pain in
17 multiple sites, these conditions are also associated with developmental processes onset conditions,
18 symptom profiles, and pathophysiology different from those produced by DOMS-protocols^{19,64}.

19 The emerging body of findings raises the possibility that pain catastrophizing and pain-related
20 fear might be risk factors for the experience of pain in response to non-noxious stimuli. These findings
21 call for the inclusion of measures of pain catastrophizing and pain-related fear as screening measures for
22 identifying individuals at risk for problematic outcomes following musculoskeletal injury. Currently,
23 psychological interventions for individuals with MSP are typically offered only once the condition has
24 become chronic. Targeting these variables in the early stages of treatment might decrease the probability
25 of transitioning from acute pain to more serious chronic pain syndromes.

26

- 1 5. Conflict of interest statement
- 2 The authors report no conflict of interest.
- 3
- 4

1 6. Acknowledgements

2 The authors thank Alexandra Tighe for her assistance in the data collection.

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